Rhabdomyolysis and polydipsic hyponatraemia

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A 41 year old man was brought to the emergency department late in the afternoon with a reduced conscious level. He was found unresponsive in his bedroom, having last been seen well that morning. On presentation, his temperature was 36.1°C, pulse rate 90/min in sinus rhythm, blood pressure 140/70 mm Hg, respiratory rate 15/min and oxygen saturation 96% on room air. He had a blood sugar level of 7 mmol/l. The patient had a Glasgow Coma Scale score of 5 (E1 V1 M3) without external signs of a head injury. His pupils were sluggishly reactive, at 4 mm on the right and 2 mm on the left. He had a history of chronic schizophrenia treated with thioridazine, a previous head injury and was a heavy smoker.

His biochemistry on presentation showed serum sodium concentration of 113 mmol/l, potassium concentration 3.4 mmol/l, chloride concentration 76 (100–110 mmol/l), bicarbonate 25 (22–33 mmol/l) and a creatine kinase (CK) of 179 (<200 U/l). No measured serum osmolality was performed, but his calculated serum osmolality was 229 (270–290 mmol/kg). CK concentrations rose over the next 48 hours, peaking at 49 300 U/l. Serum toxicology screening for paracetamol and salicylates was negative. His ECG showed an old right bundle branch block with a corrected QT duration of 453 msec. Computed tomography of the head and lumbar puncture were normal. Urine biochemistry before treatment showed an osmolality of 158 mmol/kg, sodium 29 mmol/l, potassium 6.7 mmol/l, creatinine 3.1 mmol/l and urea 51 mmol/l. The provisional diagnoses were psychogenic polydipsia induced hyponatraemia, leading to cerebral dysfunction and rhabdomyolysis. Although CK subtraction analysis was not performed, the magnitude of the CK rise, normal serial ECGs and the presence of urinary myoglobin favoured rhabdomyolysis over myocardial injury.

The patient was endotracheally intubated for airway protection by a rapid sequence induction (RSI) method using midazolam, fentanyl and suxamethonium. Correction of hyponatraemia with intravenous 3% hypertonic saline to raise the serum sodium by 1 mmol/l without external signs of a head injury. His pupils were sluggishly reactive, at 4 mm on the right and 2 mm on the left. He had a history of chronic schizophrenia treated with thioridazine, a previous head injury and was a heavy smoker.

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The patient was endotracheally intubated for airway protection by a rapid sequence induction (RSI) method using midazolam, fentanyl and suxamethonium. Correction of hyponatraemia with intravenous 3% hypertonic saline to raise the serum sodium by 1 mmol/l per hour was accompanied by a large diuresis and a significant improvement in conscious level, permitting extubation after 48 hours. Despite having rhabdomyolysis, his urine output, serum creatinine and urea levels remained normal. Causes of skeletal muscle injury such as seizures, compartment syndrome, recent intramuscular injections, pressure areas, dystonic reaction and hyperthermic syndromes were absent. Although the use of suxamethonium during RSI has been associated with rhabdomyolysis, there was no evidence of sustained rigidity, masseteric spasm or a hyperthermic reaction at the time of intubation.

Psychogenic polydipsia is the behaviourally driven consumption of large volumes of fluids in the absence of an appropriate physiological stimulus, where underlying organic disease has been excluded. It is seen in up to 17% of schizophrenic patients. Severe hyponatraemia (serum sodium < 120 mmol/l) develops in one quarter of these. Proposed mechanisms include disturbed osmoregulation of fluid intake, excessive secretion of antidiuretic hormone (ADH) and renal hypersensitivity to ADH. An association exists between psychogenic polydipsia and the use of antipsychotics and antidepressants, traumatic head injury and heavy smoking, which were present in our patient. Rhabdomyolysis attributable to hyponatraemia may be due to decreased extracellular sodium disturbing the function of membrane sodium–calcium pumps, which normally exchange an extracellular sodium ion for an intracellular calcium cation. A reduced concentration gradient for sodium entry into the muscle cell results in lessened outward shift of intracellular calcium. Intracellular incarceration of calcium activates enzymatic processes that leads to cell death. An association between rhabdomyolysis in schizophrenia and severe polydipsic hyponatraemia has been described infrequently. No other contribution to muscle damage aside from hyponatraemia was identified in this patient, and there was no personal and family history of neuropathy or myopathy. Patients with hyponatraemia related to psychogenic polydipsia are potentially at risk of developing rhabdomyolysis and myoglobinuric renal failure. Reported cases have involved serum concentrations of less than 120 mmol/l. This case report provides further evidence that patients with severe hyponatraemia associated with psychogenic polydipsia should have CK concentrations monitored to detect rhabdomyolysis, even in the absence of other recognised causes of muscle injury. More importantly in the emergency department, hyponatraemia induced cerebral dysfunction should be considered in any patient with a previous psychiatric history presenting with an altered conscious level, and the serum sodium level should be measured urgently.

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