Letters to the Editor

Rhabdomyolysis associated with cranial diabetes insipidus

Sir,

I read with interest the recent article by Dr Kung and colleagues1 documenting rhabdomyolysis in association with a hypernatraemic hyperosmolar state secondary to cranial diabetes insipidus. We recently reported a patient with rhabdomyolysis and acute renal failure who had developed hypernatraemic hyperosmolarity whilst on lithium therapy, presumably as a result of lithium-induced nephrogenic diabetes insipidus.2 These cases serve notice that non-traumatic rhabdomyolysis may occur in hyperosmolar states other than those associated with hyperglycaemia, as previously reported,3,4 and consequently that the hyperosmolar state per se predisposes to rhabdomyolysis. However, the cause of rhabdomyolysis in these cases remains uncertain. Concomitant deficiency of potassium and phosphate ions, also recognized predisposing factors for rhabdomyolysis,5 may be relevant, but masked by their release from damaged muscle cells. Singhal et al.6 found that hyperosmolar diabetic patients developing rhabdomyolysis had lower serum potassium concentrations than those who did not, and we noted relatively low serum potassium and phosphate concentrations in our patient at presentation.2

Experimental hypokalaemia has been associated with both reduced transmembrane electrical potential and histological damage in muscle cells,6 and similarly intracellular phosphate deficiency can lead to a fall in transmembrane potential since reduced ATP levels will lead to inhibition of the energy-dependent sodium pump. Hence, hypokalaemia and hypophosphataemia may be the significant biochemical derangements leading to muscle cell injury.

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References

Hyponatraemic encephalopathy complicating thiazide reserpine preparation

Sir,

We would like to report a case of hyponatraemic encephalopathy due to excessive water drinking in association with a thiazide diuretic.

Our patient was a 54 year old man who was admitted in an acutely confused state. Physical examination revealed no focal neurological deficit. The blood pressure was 130/80 mmHg and the pulse rate was 100/min and regular. He was clinically normovolaemic with a copious urine output of about 3 litres in 12 hours. Blood biochemistry revealed Na 114 mmol/l, K 1.9 mmol/l, Cl 79 mmol/l, serum osmolality 250 mosmol/kg, urine osmolality 68 mosmol/kg, spot urine Na 34 mmol/l, and spot urine K 32.1 mmol/l.

A detailed history was obtained from the relatives about 12 hours later. The patient was an anxious individual, who had been taking Adelphane Esidrex on and off for years (one tablet of Adelphane Esidrex consists of 0.1 mg reserpine, 10 mg dihydrallazine sulphate and 10 mg hydrochlorothiazide). He had recently been under great stress from his employer and from his family and combatted his anxiety by drinking about 8 litres of water a day. He also became over-concerned with his health, and insisted on a low salt diet.

The diagnosis was hyponatraemic encephalopathy due to psychogenic polydipsia and thiazide diuretic. He was treated by withdrawing the diuretic and restricting the water intake to 500 ml/day. 100 mmol of 0.9% NaCl was given over 1 hour. Over 3 days, the serum sodium rose to 132 mmol/l, and the chloride to 106 mmol/l. He recovered completely.

To our knowledge, there are twelve published cases of hyponatraemic encephalopathy caused by psychogenic polydipsia combined with a thiazide diuretic.1–9 However, all these concerned patients who had a specific neurological or psychiatric illness – chronic schizophrenia in 9 patients, epilepsy complicating structural brain disease in 2 patients and psychotic depression in 1 patient. Our patient, however, had no identifiable psychiatric illness, apart from an anxiety-prone personality.

Most reports of thiazide and polydipsia induced hypotraemia have a favourable outcome, but one reported case involved a 56 year old patient who died due to cerebral oedema with herniation.4 Thus clinicians should be alert to hypotraemia in patients taking thiazides, especially in those at particular risk: e.g. elderly, cirrhotic, and psychiatric patients.

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