In March, 2002, a 44-year-old ostrich farmer from outback Australia experienced a sudden onset of muscle weakness after returning home from an evening of shooting kangaroos. He had difficulty getting out of his bath, and had to wait on the floor for assistance because he was unable to stand. Over the following hour his weakness progressed, and he was taken by ambulance to the local hospital. He had no other symptoms and denied any history of recent drug use, alcohol consumption, or exposure to toxins. He had been previously fit and well, with no significant, medical history.

On examination we found generalised muscular weakness (grade 2/5). All deep tendon reflexes were present, and sensation was normal. Over the next few hours, his respiratory function deteriorated, necessitating endotracheal intubation and artificial ventilation. Blood tests showed hypokalaemia (1·4 mmol/L), and increased serum creatine kinase (289 U/L). Simultaneous urinalysis showed inappropriate kaliuresis (17·4 mmol/L) and osmotic diuresis (1160 mOsm/24 h). Thyroid function tests, urine drug screens, ACTH stimulation testing, plasma renin activity, and plasma cholinesterase concentrations were all normal. An ECG showed T-wave flattening and U waves (figure). We treated him with intravenous fluids, and his serum potassium concentration normalised within 24 h. He was extubated the next day and rapidly regained normal muscle strength. On further questioning, the patient admitted consuming approximately 4 L of Coca-Cola most days, and up to 10 L when he went kangaroo shooting at night. Kangaroos are considered to be agricultural pests in this region, and deliberate culls are done on a regular basis. He had been drinking these quantities of cola for the previous 3 years, and admitted to having polyuria and nocturia over the same period. We advised him to curtail his cola drinking, and over the next few days, his serum potassium remained normal on an ad-libitum diet, without requiring supplementation. When last seen, in October, 2002, he had continued to avoid soft drinks and was healthy, with no further complaints.

Hypokalaemic myopathy has been reported to occur following excessive consumption of cola.1,2 In this case, the severe hypokalaemia was multifactorial. Firstly, cola contains a lot of sugar (11 g/dL, osmolality 430 mOsm/kg) and negligible potassium. Our patient’s excessive consumption probably caused the osmotic diuresis, and the accompanying potassium loss and his inadequate dietary intake resulted in a potassium deficit. Secondly, cola contains caffeine; in Coca-Cola the concentration is 97 mg/L.3 Caffeine’s pharmacological effects occur via antagonism of adenosine receptors in many organs, including the kidney.4 Adenosine receptor antagonism contributes to excessive loss of urinary potassium in favour of sodium retention, thus exacerbating hypokalaemia. Caffeinated and/or hyperosmolar beverages should be considered as a cause of unexplained hypokalaemia with osmotic diuresis and kaliuresis. Patients need to be warned that immoderate soft drink consumption may cause severe or recurrent symptoms.

References