MUSCULAR PARALYSIS AND VENTILATORY FAILURE CAUSED BY HYPERKALAEMIA

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SUMMARY
We report the history of a diabetic man presenting with hyperkalaemia and rapidly progressing muscular paralysis with severe respiratory embarrass-ment. His symptoms resolved rapidly with reduction of the serum potassium concentration. Similar cases have been reported previously but this is a problem that few will have encountered and is a diagnosis which should not be overlooked before artificial ventilation is commenced. (Br. J. Anaesth. 1993; 70: 226-227)

KEY WORDS

CASE REPORT
A 75-yr-old man was admitted with a short history of progressive ascending muscular weakness. He had a history of maturity onset diabetes mellitus, severe peripheral vascular disease with a right femoro-popliteal bypass and benign prostatic hypertrophy for which he had undergone TURP 2 months before admission, when his serum creatinine concentration was normal (98 umol litre$^{-1}$).

His usual drug treatment was glibenclamide 5 mg twice daily and metformin 1 g twice daily. One week before admission, he was prescribed amiloride—hydrochlorothiazide (Moduretic) tablets one daily for peripheral oedema.

Thirty hours before admission he had noticed difficulty getting up from a chair, which had progressed rapidly to global muscle weakness. He had not noticed any sensory symptoms or sphincter disturbance and his history gave no suggestion of a recent viral illness.

On examination, he had profound muscle weakness of arms and legs (proximal muscles MRC grade 1/5; distal muscles MRC grade 0/5), together with difficulty talking and breathing. His peak ventilatory flow rate was 70 litre min$^{-1}$. There was marked muscular fasciculation in the arms and legs, reflexes were depressed and plantar responses absent. He had a “stocking” loss of sensation to below the knee, mild background diabetic retinopathy, but no cranial nerve abnormalities.

A presumptive diagnosis of acute postinfective polynейritis was made and he was transferred to the ICU for tracheal intubation and mechanical ventilation of the lungs.

ECG showed broad QRS complexes with peaked T waves, biochemistry showed serum concentrations of sodium 127 mmol litre$^{-1}$, potassium 10.2 mmol litre$^{-1}$, chloride 101 mmol litre$^{-1}$, bicarbonate 17 mmol litre$^{-1}$, urea 18.6 mmol litre$^{-1}$, creatinine 207 μmol litre$^{-1}$, glucose 22.0 mmol litre$^{-1}$. Arterial blood-gas analysis revealed pH 7.29, $PCO_2$ 4.4 kPa, $PO_2$ 13.6 kPa, base excess $-9$ mmol litre$^{-1}$. CSF pressure was 15 cm H$_2$O, protein 166 mg litre$^{-1}$ (ref. 155-450), glucose 8.6 mmol litre$^{-1}$, WBC $<1 \times 10^9$ litre$^{-1}$.

Treatment and progress
There was a dramatic response to treatment with i.v. calcium gluconate, an insulin and glucose infusion and i.v. sodium bicarbonate. Within 20 min he was able to lift both hands above his head and his peak ventilatory flow rate had increased to 350 litre min$^{-1}$. Treatment was continued with an insulin–glucose infusion, oral calcium resonium and fludrocortisone which was withdrawn after 12 days. His serum potassium concentration fluctuated over the next 36 h mirroring changes in his peak expiratory flow rate, but then settled within the normal range. He was discharged and, at follow up 10 days later, was well, with normal serum electrolyte and creatinine concentrations. Ultrasound showed normal kidneys with no evidence of obstruction. He had normal liver function tests and serum calcium concentration and a normal response to a short synacthen test.

DISCUSSION
Both hypo- and hyperkalaemia can produce a clinical picture of flaccid muscular paralysis very similar to that of acute postinfective polyneuropathy (Guillain–Barré syndrome) and the importance of measuring the serum potassium concentration and recording an ECG early in the investigation of suspected acute postinfective polyneuropathy has been emphasized [1, 2]. In our patient, rapid recovery of muscle power after emergency treatment of

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the hyperkalaemia, combined with normal CSF confirmed that hyperkalaemia was the cause of his paralysis.

Although cardiac arrhythmias are the usual cause of death in severe hyperkalaemia, muscular weakness may be the predominant manifestation, with ventilatory failure a potentially lethal complication, as in our patient, and occasionally artificial ventilation may be needed [3]. The use of suxamethonium as a rapidly acting neuromuscular blocker is associated with an increase in serum potassium concentration [4]; this increase may precipitate arrhythmias.

The dangers of using potassium-sparing diuretics in elderly patients, who may have some degree of renal impairment despite normal serum creatinine concentrations, are well known. Hyperkalaemia after treatment with amiloride–hydrochlorothiazide combinations has been reported previously [5]. In our patient, the period of hyperkalaemia correlated well with the duration of action of amiloride, started 1 week before presentation, and emphasizes the importance of early biochemical monitoring after starting treatment with a potassium-sparing diuretic in the elderly.

REFERENCES