



Hyperkalaemic paralysis

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Hyperkalaemia is associated with cardiac manifestations with characteristic electrocardiogram (ECG) changes. It rarely presents with acute tetra-paralysis. We present a case of secondary hyperkalaemic paralysis.

Case report

A 46-year-old gentleman presented with lethargy, nausea, vomiting and acute ascending weakness. He had end-stage renal failure secondary to diabetes and was on home haemodialysis. He dialysed every second day. He had Type 2 diabetes (on insulin), hypertension (on quinapril and metoprolol), anxiety and depression (on citalopram). Other medications were alphacalcidol, calcium carbonate, aluminium hydroxide, erythropoietin, simvastatin, frusemide and ranitidine.

He was transported to the hospital via ambulance. He complained of weakness in his legs. He had not completed his most recent haemodialysis which was due 3 days previously. On route he became hypotensive (60 mmHg systolic) and bradycardic (35/min). Intravenous adrenalin was commenced and systolic blood pressure improved to 100 mmHg systolic.

On presentation to hospital the weakness ascended and involved his upper limbs. He

was afebrile, had a blood pressure of 136/70 mmHg, pulse rate of 30/min and O₂

saturation of 100% on air. JVP was at the angle of the jaw. There was bilateral, symmetrical weakness of the upper and lower limbs (power 1-2/5). Reflexes and plantar responses were reduced.

Venous blood gas analysis showed a pH of 7.24, bicarbonate of 15.0 and a base excess of -9.8. ECG showed a junctional bradycardia with peaked T-waves. Blood tests revealed blood glucose 5.9, serum sodium 130, serum potassium 7.9, haemoglobin 126, WBC 11.7, platelets 270, urea 43.6, creatinine 1390, calcium 2.29 and phosphate 2.38.

His hyperkalaemia was initially managed with calcium gluconate and intravenous insulin. He was transferred to the renal ward where haemodialysis was commenced. He was dialysed for 4 hours and throughout the procedure his tetraparesis gradually improved—this was correlated with a reduction in repeated serum potassium (5.0). A repeat ECG showed normal sinus rhythm. His power was 5/5 universally. He received haemodialysis the following day and was asymptomatic for the remainder of admission.

He had two further presentations of tetra-paresis. Both were associated with hyperkalaemia and improved during haemodialysis. The episodes were due to chronic renal failure and non-compliance with haemodialysis.

Discussion

There are primary and secondary forms of hyperkalaemic paralysis.

Primary hyperkalaemic paralysis involves a genetically determined defect in sodium ion channels of muscle fibre plasma membranes.^{1,2} Presentations of flaccid paralysis happen in the first decade. The attacks last from a few minutes to hours but rarely exceed 4 hours. The attacks are initially infrequent and tend to increase in frequency and severity over time.¹ Common triggers include rest after exercise and intake of potassium.^{1,2} Salbutamol, intravenous calcium gluconate and acetazolamide all successfully treat attacks.^{1,2}

The exact mechanism of secondary hyperkalaemic paralysis is not known. It may be due to potassium directly affecting the muscle cell membrane or the peripheral nerves.³ Patients present later in life. Evers et al described 18 cases.⁴ Presentation classically involved an ascending flaccid paralysis with mild or no sensory deficit and spared cranial nerves. It may be mistaken for Guillain-Barré syndrome.⁴

Attacks are usually precipitated by chronic renal failure⁴ but have also been reported secondary to potassium sparing diuretics,⁵ ACE inhibitors,⁶ nonsteroidal anti-inflammatories,⁷ postoperative renal impairment⁸ and traumatic rupture of the bladder.⁹

Management involves correcting physiological disturbance, repeated ECGs and monitoring serum potassium. Treatment is through correcting hyperkalaemia through calcium infusion, insulin, salbutamol or dialysis. Long-term management involves reducing dietary potassium, avoidance of predisposing medications and compliance with dialysis.

The prognosis for secondary hyperkalaemic paralysis is good if recognised and managed appropriately; reported deaths were due to arrhythmias.⁴ There have been reports where no ECG changes have been present.⁵ Therefore both an ECG and serum potassium levels should be obtained in patients presenting with acute paralysis.

There must be a universal awareness because if not recognised, secondary hyperkalaemic paralysis can be a fatal condition.

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