Poisoning with sustained release potassium

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INTRODUCTION

Poisoning with potassium chloride is uncommon (Illingworth & Proudfoot, 1980) leading to controversy over the best sequence of therapeutic intervention (Whitehouse, 1980). A case of life-threatening hyperkalaemia following the ingestion of sustained release potassium tablets is reported.

CASE REPORT

A 27-year-old woman with anorexia nervosa took 60 tablets of Slow-K (sustained release potassium chloride 600 mg; 8 mmol each of K^+ and Cl^-) and soon started to vomit. Her potassium supplement therapy had been discontinued one week prior to admission when the plasma potassium concentration was 3.9 mmol/litre. Twelve hours after the ingestion she was cyanosed with cold extremities, poor peripheral pulses, a regular pulse of 70/min and blood pressure 100/60 mmHg. The electrocardiogram showed first degree heart block, wide QRS complexes and tall T waves (Fig. 1). The plasma potassium concentration was 9.1 mmol/litre and urea concentration 3.4 mmol/litre. Blood gas analysis revealed a mixed metabolic and respiratory acidosis; H^+ 58 nmol/litre, PCO_2 4.6 kPa, HCO_3 14.8 mmol/litre and PO_2 6.8 kPa. She was given oxygen and 30 g calcium polystyrene sulphonate (calcium resonium) orally. Twenty ml 10% calcium gluconate and 50 ml 50% dextrose with 10 units soluble insulin were administered intravenously; 100 ml 8.4% sodium bicarbonate were given to correct the metabolic component of the acidosis. Within 1-2 minutes of giving calcium gluconate, the QRS complexes on the electrocardiogram reverted to normal and the PR interval shortened (Fig. 1). Repeat blood gas analysis 30 minutes following admission revealed H^+ 50 nmol/litre, PCO_2 5.0 kPa, HCO_3 18.5 mmol/litre and PO_2 12.7 kPa. Intravenous fluids and frusemide were given to force a diuresis during which the urine output was

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Fig. 1. Electrocardiogram. Upper strip from monitor 12 h after overdosage (plasma potassium concentration 9.1 mmol/litre; lower strip 1–2 min later, after the administration of calcium gluconate.

about 500 ml/hour with potassium concentrations of 9 mmol/litre to 90 mmol/litre. A chest radiograph disclosed no abnormalities but the plain abdominal film revealed the cores of approximately 16 tablets (Fig. 2). A further 30 g calcium polystyrene sulphonate were given orally along with 200 ml 20% mannitol which produced a cathartic response within 30 minutes. Figure 3 illustrates the fall in plasma potassium concentration following treatment and the urinary potassium excretion. The electrocardiogram returned to normal and the patient made a full recovery.

DISCUSSION

Acute overdosage with potassium chloride including sustained release formulations is uncommon, although deaths have been reported in children (Bacon, 1974) and adults (Illingworth & Proudfoot, 1980) after accidental and deliberate ingestion. The principal toxic effect of potassium is on the myocardium resulting in life-threatening cardiac arrhythmias. As the extracellular potassium concentration rises, characteristic changes

Fig. 2. Plain abdominal radiograph revealing tablet cores.

occur in the electrocardiogram. Initially these are tall peaked T waves (serum potassium concentration 6–8 mmol/litre) followed by atrial asystole and complete heart block before bizarre widened QRS complexes, ventricular fibrillation and standstill occur (serum potassium concentration over 8 mmol/litre) (Browning & Channer, 1981).

Evidence of cardiotoxicity requires immediate action and should take precedence over measures to prevent absorption or enhance elimination (Table 1). Stabilization of the myocardial cell is achieved by the administration of calcium gluconate (10–20 ml of a 10% solution) and is indicated if there is QRS widening. This should result in a prompt reversal of the electrocardiographic signs and the danger of dysrhythmias is

Fig. 3. The plasma potassium concentration and urinary potassium concentration following the commencement of treatment.

Table 1 Principles underlying the therapeutic approach to hyperkalaemia

1. Ameliorate toxicity:	
(a) protect myocardium	Calcium gluconate i.v.
(b) redistribute absorbed potassium	Dextrose and insulin i.v. Sodium bicarbonate i.v.
2. Prevent further absorption:	
(a) gastric lavage	
(b) adsorbents	Calcium polystyrene sulphonate orally, rectally or via the lavage tube
(c) whole gut clearance	Osmotic catharsis e.g. mannitol
3. Enhance elimination:	
(a) renal	Forced diuresis with intravenous fluids and frusemide
(b) extracorporeal	Peritoneal dialysis Haemodialysis

rapidly diminished (Whitehouse, 1970). Bradyarrhythmias may be managed by temporary cardiac pacing (Rosenberg *et al.*, 1970).

The potassium gradient across the cell membrane may be reduced by shifting potassium intracellularly by giving dextrose (25–50 g) and soluble insulin (10–20 units) intravenously which will reduce the plasma potassium concentration by approximately 1 mmol/litre within 30 minutes, the effect persisting for one to two hours (Ledingham, 1987). The shift of potassium from extracellular fluid into cells is facilitated by alkalaemia produced after the correction of any metabolic acidosis with intravenous

sodium bicarbonate. The respiratory component of the acidosis in this patient presumably resulted from muscular weakness secondary to the severe hyperkalaemia (Barker, 1980). Hypoxia with hypoventilation is ideally managed by intubation and intermittent positive pressure ventilation. If this is not undertaken, as in this case, the patient should be carefully observed clinically with frequent measurement of blood gases. In addition, extreme caution should be exercised when administering sodium bicarbonate to a hypoventilating patient.

Plain abdominal radiograph was found to be of use in detecting residual tablet fragments in the gastrointestinal tract (O'Brien *et al.*, 1986), which was still positive 12 hours after ingestion. These fragments may represent 'tablet ghosts' consisting of the tablet core but no potassium. However some sustained release preparations release potassium up to 12 hours following ingestion, notably K-Contin Continus (Association of British Pharmaceutical Industry, 1988).

Following stabilization of the cardiac state, gastric lavage should be carried out. This would not have been of value in this case because of the delay in presentation. Lavage is unlikely to remove intact slow release tablets which may be too large to pass up the stomach tube. Further absorption of potassium can be prevented by giving calcium polystyrene sulphonate (60 g) orally, via the lavage tube or rectally—1 g of Calcium Resonium can adsorb about 1.3 mmol of potassium (Martindale, 1982). For tablets beyond the reach of the stomach tube whole gut lavage (Woo *et al.*, 1976) or catharsis (Shannon *et al.*, 1986) may be helpful especially with sustained release preparations which liberate potassium over three hours or longer. Osmotic catharsis using mannitol has been shown to be more effective than magnesium salts in clearing the bowel (Palmer & Khan, 1979).

A forced diuresis may be employed to enhance the elimination of potassium from the extracellular fluid by giving intravenous fluids and frusemide. It is interesting to note that in the first 12 hours of treatment the total urinary excretion of potassium was the equivalent of approximately 15 tablets i.e. a quarter of the amount said to have been taken. If the patient's cardiac state does not permit forced diuresis or renal function is impaired, urgent peritoneal or haemodialysis should be considered.

Sustained release potassium is seldom taken in overdose; however, prompt recognition of hyperkalaemia and immediate treatment may be life-saving.

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