was treated with frusemide 80 mg daily and isosorbide dinitrate 10 mg four times daily. Later he developed a chest infection and episodes of supraventricular tachycardia, which were suppressed with verapamil 80 mg three times a day. He was symptom free 14 days after admission, the radiographic signs of left heart failure had resolved, and he was mobilised. Five days later gross bilateral ankle oedema was noted. Jugular venous pressure was normal and the chest *x*-ray film and electrocardiogram were unchanged. Frusemide and verapamil were continued in the same dosage and isosorbide withdrawn. The ankle oedema resolved within 48 hours without accompanying weight loss. Isosorbide was reintroduced when he was fully ambulant: the oedema did not recur.

Case 3—A 61-year-old man with ischaemic heart disease and congestive cardiac failure was admitted for reassessment. He had basal crepitations, raised jugular venous pressure, enlargement of the liver, and bilateral ankle oedema. An electrocardiogram showed atrial fibrillation and previous anterior myocardial infarction. There was radiographic evidence of cardiomegaly and pulmonary venous congestion. Liver function tests were abnormal. Serum albumin, urea, and creatinine concentrations were normal. Treatment with digoxin, bumetanide, spironolactone, and isosorbide dinitrate 20 mg three times a day was continued. He was not confined to bed. Weight and ankle circumference were measured serially. Isosorbide was withdrawn after seven days. Over the next week, while symptoms and weight were unchanged, the ankle oedema decreased. Isosorbide was reintroduced. The oedema reaccumulated over the following week and his weight remained unchanged.

Comment

There is strong evidence that isosorbide dinitrate caused the peripheral oedema in cases 1 and 2 and aggravated it in case 3. On the basis of these and similar cases I suggest that peripheral oedema is a fairly common side effect of isosorbide dinitrate and that failure to recognise this may lead to inappropriate diuretic treatment.

Isosorbide dinitrate causes venous dilatation with resulting venous pooling and diminished venous return.[3] Peripheral oedema is thus an easily explicable side effect, and it is surprising that it has not been reported previously.

My observations suggest that isosorbide-induced oedema is most likely to occur in semi-ambulant patients. Case 3 raises the possibility that hepatic dysfunction with impaired degradation of the drug may be an additional predisposing factor.

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Hyperkalaemic cardiac arrhythmia caused by potassium citrate mixture

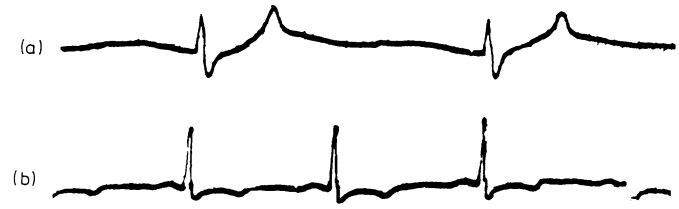
We report a case of severe hyperkalaemia after ingestion of potassium citrate mixture *BP*.

Case report

An 83-year-old woman presented to the casualty department after collapsing at home. She was unable to give a clear history and on examination looked pale and dyspnoeic. Peripheral pulses were poor and heart rate was regular at 40/min. Blood pressure was 100 mm Hg by palpation. Jugular venous pressure was normal, and there was no oedema. Auscultation disclosed a pansystolic murmur at the apex and crepitations at both lung bases. She had suprapubic abdominal tenderness. There were no abnormal neurological signs. Electrocardiography showed a junctional bradycardia with absent P waves (figure). A temporary transvenous endocardial pacing wire was inserted, after which her clinical state improved and she was able to give a history.

She had been well until one week before admission, when she had developed urinary frequency, haematuria, and dysuria. She had purchased and ingested indeterminate large volumes of potassium citrate mixture BP. Immediately before admission her urinary frequency had worsened and she had become faint and dizzy.

Initial investigations showed sodium 137 mmol(mEq)/l, potassium 8·7 mmol(mEq)/l, urea 6·0 mmol/l (36·0 mg/100 ml), chloride 102 mmol(mEq)/l, carbon dioxide 26 mmol(mEq)/l, and creatinine 0·11 mmol/l (1·2 mg/100 ml). A midstream urine specimen showed over 100 leucocytes/mm^3 and no bacterial growth. Chest radiography disclosed no abnormalities. Serum potassium concentration reverted to normal after the administration of glucose and insulin and thereafter remained within the normal range. Urinary symptoms responded to a course of ampicillin. Further investigations showed: creatinine



Rhythm strip (*a*) on admission and (*b*) 12 hours later, when serum potassium concentration normal.

clearance 37 ml/minute, urinary sodium 49 mmol(mEq)/l, urinary potassium 38 mmol(mEq)/l, urinary aldosterone 16 nmol (5·8 μg)/24 hours (normal 10-55 nmol (3·6-19·8 μg)/24 hours), and plasma aldosterone 200 pmol/l (7·2 ng/100 ml) (normal 100-500 pmol/l (3·6-18·0 ng/100 ml). Serial cardiac enzyme activities were normal, and a Synacthen test gave normal results.

Comment

Severe hyperkalaemia may cause life-threatening cardiac arrhythmias. As the extracellular potassium concentration rises characteristic changes occur in the electrocardiogram. Initially these are peaked T waves (serum potassium concentration 6-8 mmol(mEq)/l) and then atrial asystole and complete heart block before bizarre QRS complexes, ventricular fibrillation, and standstill occur (serum potassium concentration over 8 mmol(mEq)/l). Such bradyarrhythmias may be managed by temporary cardiac pacing[1] while the hyperkalaemia is corrected.

Hyperkalaemia is usually seen complicating acute or chronic renal failure, but in patients with normal renal function it is most commonly drug induced[2]—such drugs include potassium supplements and potassium-sparing diuretics—or due to hyporeninaemic hypoaldosteronism.[3] We believe that the transient hyperkalaemia in this patient was caused by the excessive ingestion of potassium citrate mixture *BP*. This is used to alkalinise the urine in urinary tract infections and contains 28 mmol(mEq) potassium/10 ml dose. The recommended dose is up to 40 ml daily. Hyperkalaemic ventricular fibrillation has been reported after ingestion of small amounts of potassium salts (40-60 mmol(mEq)/l) in patients with good renal function.[4] Potassium citrate mixture *BP* has a high concentration of potassium and may be hazardous to those who handle the ion poorly.

We are concerned that this potentially dangerous mixture may be purchased over the counter from any retail chemist.

We thank Professor J M Evanson for permission to report this case.

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