CASE REPORTS

Obsolete but dangerous antacid preparations

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Summary

One case of acute hypercalcaemia and two of recurrent nephrolithiasis are reported in patients who had regularly consumed large amounts of calcium carbonate-sodium bicarbonate powders for more than 20 years. The powders had been obtained from pharmacists unknown to the patients' medical practitioners. It is suggested that these preparations were responsible for the patients' problems, and that such powders should no longer be freely obtainable.

Introduction

Calcium carbonate is now rarely prescribed, but may still be obtained without prescription from pharmacists. It is an efficient antacid (Piper and Fenton, 1964) but it stimulates gastrin release (Levant, Walsh and Isenberg, 1973), rebound gastric hypersecretion (Fordtran, 1968), and may cause hypercalcaemia (McMillan and Freeman, 1965). Serious complications occurring in three patients who had regularly consumed large amounts of calcium carbonate-sodium bicarbonate powders over many years, unknown to their current medical practitioners, are now reported.

Case 1

A 67-year-old retired engineer presented with a 1-week history of confusion and dehydration. For the previous year he had suffered polydipsia and occasional vomiting. He had been admitted to hospital with a similar episode of confusion 5 months before, but had made a complete recovery. An intravenous pyelogram had been normal. For 20 years, he had been taking up to 15 g of calcium carbonate, 30 g of sodium bicarbonate and two pints of milk daily, for indigestion.

Investigations revealed a haemoglobin level of

12 g/dl; plasma urea, 30.5 mmol/l; sodium, 142 mmol/l; potassium, 2.8 mmol/l; total CO₂, 58 mmol/l; calcium, 3.45 mmol/l; phosphate, 0.88 mmol/l; magnesium, 0.97 mmol/l; normal alkaline phosphatase levels, and total serum protein 81 g/l with a normal albumin level. Parathyroid hormone was undetectable. His 24-hour urinary calcium and phosphate excretions were 2.3 mmol and 5.3 mmol respectively, and creatinine clearance was 14 ml/min. X-rays of his chest and hands were normal but barium meal demonstrated an active duodenal ulcer with some pylorospasm and delayed gastric emptying.

With rehydration, potassium supplements, and a non-absorbable antacid, he made a complete symptomatic recovery. His urea, electrolytes and calcium returned to normal and he remained well when reviewed 2 months later.

Case 2

A 45-year-old insurance clerk presented with renal colic, having previously passed calculi 15 and 4 years before. For 25 years he had regularly consumed about 7 g of calcium carbonate, 20 g of sodium bicarbonate and one half pint of milk per day for heartburn and indigestion.

Investigations revealed normal haemoglobin, blood urea, electrolytes, urate, phosphate, alkaline phosphatase, proteins and parathyroid hormone. The creatinine clearance was 74 ml/min and one of four serum calcium measurements was elevated (2.71 mmol/l). The 24-hour urinary excretion of calcium and phosphate on a ward diet without antacids ranged from 2.57 to 6.14 and 22.05 to 29.25 mmol respectively. An intravenous pyelogram demonstrated calculi in the right ureter and renal pelvis, which were removed by surgery. Subsequently, oesophagitis and active duodenal ulceration were demonstrated radiologically and endoscopically.

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Case 3

A 72-year-old retired business man presented with renal colic, having passed calculi 8 and 6 years previously. A duodenal ulcer had been demonstrated radiologically 30 years before, since when he had regularly taken 7 g of calcium carbonate, 20 g of sodium bicarbonate and less than one half pint of milk per day for heartburn and indigestion.

Investigations revealed normal haemoglobin and plasma sodium, potassium, bicarbonate, alkaline phosphatase, calcium, phosphate, urate, proteins and parathyroid hormone. The blood urea was 8.7 mmol/l and creatinine clearance 58 ml/min. Urinary calcium and phosphate excretion rates were normal on a ward diet without antacids.

An intravenous pyelogram showed calculi in both renal pelves and an obstructing mid-ureteric calculus, which subsequently passed spontaneously.

Comments

These patients had taken calcium carbonate and sodium bicarbonate regularly several times daily for 20 to 30 years. Excessive calcium ingestion produces hypercalciuria and renal calculi in susceptible individuals (Nordin, 1977). Case 1 closely resembled the acute form of the milk alkali syndrome (McMillan and Freeman, 1965).

Although these patients obtained their original prescriptions from their doctors, the antacid powders were subsequently supplied by pharmacists for 20–30 years, unknown to the patients' current general practitioners. These obsolete antacid mixtures are still causing clinical problems which could readily be prevented if such powders were no longer obtainable.

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Myocardial ischaemia in migraine sufferers taking ergotamine

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Summary

Two cases of acute myocardial ischaemia precipitated by oral ergotamine therapy for migraine are described from patients with no previous history of ischaemic heart disease. The relevant literature is briefly reviewed.

Case 1

An otherwise healthy 50-year-old man had suffered from migrainous neuralgia for 8 years. In recent months he had been taking up to 40 mg of ergotamine every week (as sublingual Lingraine)

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to control his attacks, and had occasionally noticed severe pain in his forearms some hours after taking the tablets. Early on the morning of admission he took his usual dose of 10 mg ergotamine to abort an impending attack, with success. At midday he developed forearm pain accompanied by severe central chest pain, and was brought to hospital. Physical examination was unremarkable, but an electrocardiogram (Fig. 1) showed gross elevation of the S-T segment in the anteroseptal leads, and he was admitted with a suspected myocardial infarct. Ninety min later his ECG had returned to almost normal, and he remained pain-free thereafter; however, on the fourth day after admission