Hypercalcaemia from non-prescription vitamin A

Sir,

Use of over-the-counter ‘natural’ supplements may have unnatural effects. We report this case as a cautionary example.

A 67-year-old white man was admitted to hospital in late January 2004, with malaise and hypercalcaemia. He had undergone kidney transplantation 6 years earlier for kidney failure of unknown cause. Chronic allograft nephropathy occurred and the plasma creatinine rose to 400 μmol/l. He began to feel unwell in December 2003, and had a 5 kg weight loss. There was constipation, melena and haematochexia. His medications included prednisone, mycophenolate mofetil, atorvastatin, labetalol, bumetanide and potassium chloride. Blood tests showed a total plasma calcium of 4.2 mmol/l and he was admitted to hospital. On examination, there was a tremor and unsteadiness of gait. The plasma creatinine was 450 μmol/l. The ionized calcium level was 1.7 mmol/l (normal 1.2–1.3). Intravenous normal saline was given. Chest X-ray was normal. Upper and lower gastrointestinal endoscopy showed antral gastritis and two benign colonic polyps. Serum and urine protein electrophoresis did not show a paraprotein. The parathormone (PTH) level was 17 pg/ml (normal 10–65) and that of PTH-related peptide was undetectable. The total plasma calcium reached 2.5 mmol/l by hospital day 4. On that day, further enquiry showed that he had been taking a dietary supplement containing vitamin A, on the advice of an eye doctor, for the possible diagnosis of macular degeneration. This over-the-counter supplement contained 7000 U of vitamin A, whereas the recommended daily intake is only 5000 U. The association of vitamin A toxicity and hypercalcaemia is rare but well recognized. We found only eight case reports of this association in the past 30 years, the most recent being in 1988 [1].

The hypercalcaemia of vitamin A toxicity may occur because of activation of bone resorption by vitamin A [2]. This man’s reduced baseline kidney function may have predisposed him to vitamin A toxicity. Given the modern prevalence of use of alternative medicines and supplements, more such cases may occur, which emphasizes the ongoing importance of vigilance and a careful medication history.

Conflict of interest statement. None declared.

Sir,

Aluminum (Al) toxicity in patients with end-stage renal disease is a well known adverse effect due to either dialysate Al contamination or oral intake of Al-containing phosphate binders [1]. At present, the clinical forms of Al toxicity have almost disappeared. Al-containing drugs are given mainly as antacid agents and are often used without special caution in patients with chronic renal failure (CRF) not yet on dialysis. Herein, we report a case of fatal Al-related encephalopathy in a patient with severe CRF, not on dialysis, due to the intake of large doses of antacids containing Al for at least 3 years.

Case. A 59-year-old white male patient with CRF due to diabetic nephropathy was followed as an out-patient in our chronic kidney disease clinic. When he was 47 years old, diabetes mellitus was diagnosed, and he was treated with oral antidiabetics for 2 years and thereafter with insulin. At 55, a severe polyneuropathy and distal occlusive arterial disease with foot gangrene occurred that required the amputation of the left foot. He suffered from gastric pain which he self-treated with Al hydroxide (Maalox® TC). A gastroendoscopy was performed that revealed antral gastritis positive for Helicobacter pylori. Despite the antibiotic treatment, the patient continued taking Al hydroxide. From the age of 57, he regularly attended our chronic kidney disease clinic. His serum creatinine was between 3 and 4 mg/dl