Vitamin D poisoning by table sugar

Reinhold Vieth, Tanya R Pinto, Bajinder S Reen, Min M Wong

In June 1999, a 29-year-old man came to the emergency department with right-sided flank pain, conjunctivitis, anorexia, fever, chills, increased thirst, and vomiting. He had lost 5 kg in weight, and was in acute renal failure. He was treated with steroids and discharged. In October, 1999, his 63-year-old father came to the emergency department with similar complaints. He was also in acute renal failure and denied a history of renal stones. His serum creatinine was 442 µmol/L (NR, 60–120 µmol/L); calcium, 3·82 mmol/L (2·0–2·65 mmol/L), PTH <1 pmol/L (1·3–7·6 pmol/L); 25-hydroxyvitamin D [25(OH)D], 1555 nmol/L (20–80 nmol/L) and serum 1,25(OH)2D, 151 pmol/L (30–140 pmol/L). More complete biochemical testing on the son in October, 1999, showed the same biochemical profile. Kidney biopsies of both patients showed severe nephrocalcinosis. The similarities initially suggested a genetic abnormality, or a granulomatous disease, but the 25(OH)D results showed severe vitamin D intoxication.

The patients continued to be treated with prednisone. Both denied taking any nutritional supplements. In December, 1999, the son was readmitted with extreme pain, nausea, and dehydration. Serum 1,25(OH)2D was 266 pmol/L; calcium, 4·39 mmol/L. The serum 25(OH)D was now 3700 nmol/L by radioimmunoassay, but chromatography revealed a huge excess of vitamin D3 (figure 1). The patient was given intravenous hydrocortisone, sodium phosphate, and pamidronate acetate.

We tested various foods from the household, including white table sugar sampled in December, 1999, after the son fell sick from drinking sweetened tea. One gram of sugar contained 21·4 mg vitamin D3, measured after extraction into ethanol. When the sugar was dissolved in water, the distinctive, long, white crystals of vitamin D3 floated up when centrifuged, and we did high performance liquid chromatography which confirmed their composition. A second sugar sample in January, 2000, contained 3·2 mg of vitamin D per gram of sugar. Assuming an average of 12·6 mg vitamin D3 per gram of sugar, and a conservative usage of 100 g sugar per month, the patient and his father had consumed more than 1·3 g of vitamin D3 per month, or 42 000 µg/day (1 700 000 IU/day), in vast excess of the minimal toxic level (95 µg, 3800 IU per day),1 for 7 months. This isolated incident was caused either by the intentional or accidental mixing of crystalline vitamin D3 into the table sugar of this family. When last seen, in April, 2001, the son’s serum 25(OH)D was 250 nmol/L; creatinine, 125 mmol/L. The father was last seen in June, 2001, his serum 25(OH)D was 181 nmol/L; creatinine, 179 µmol/L. Both had no symptoms, and continued to take prednisolone.

These patients initially posed a diagnostic challenge which highlights the need to consider poisoning as part of the differential diagnosis in metabolic disorders, especially if more than one family member is affected. Fortunately, the poison and its source were identified. All known poisonings of adults with vitamin D3 reflect misuse on an industrial scale. Huge excesses of vitamin D3 have been added in error to milk,2 or to a food supplement.3 There are two reports of households where industrial concentrates of vitamin D3 were mistaken for cooking oil.4,5 In contrast, all reports of iatrogenic vitamin D3 intoxication of adults have involved vitamin D2, a synthetic analogue of the physiological compound, vitamin D3.1 Our cases offer a perspective into the risks, management and prognosis of the worst possible form of vitamin D3 toxicity.

References

1 Vieth R, Chan PC, MacFarlane GD. Efficacy and safety of vitamin D(3) intake exceeding the lowest observed adverse effect level. Am J Clin Nutr 2001; 73: 288–94.