Calcitonin Therapy in Vitamin D Intoxication

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Hypervitaminosis-D is associated with elevated serum and urinary calcium levels, increased 25 OH vitamin D levels. Hypercalcaemia develops within 1–3 months of administration of vitamin D. However, it may go unrecognized because of non-specific nature of the symptoms, such as irritability, nausea, vomiting, and anorexia. We would like to draw attention to a patient with hypervitaminosis D in whom calcitonin therapy was administered because the classical therapy methods failed.

Case Report
An 8-month-old boy was admitted with a history of nausea and vomiting. These symptoms had been present for 2 months. A dose of 300,000 I.U. vitamin D was administered orally 2 months ago because of vitamin D deficiency. Then the parents repeated the same dose every 20 days; consequently, vitamin D had been administered four times.

Physical examination revealed a child with 7300 g weight (10 per centile), 67 cm length (25 per centile), 42.5 cm head circumference (25 per centile). Blood pressure was 70/40 mmHg. The rest of the physical examination was normal.

Laboratory examination
Blood calcium was 15.8 mg/dl, while phosphorus and alkaline phosphatase were 5.2 mg/dl and 861 U, respectively. Blood magnesium was 1.8 mg/dl. Creatinin, calcium, and phosphorus in the 24-h sample of urine were 0.07 g, 0.0215 g, and 0.13 g, respectively. Serum parathormone level was 21.4 pg/ml (normal range: 12–72 pg/ml). The level of serum 25 OH vitamin D was 90 mg/ml (normal: 10–40 mg/ml). Serum calcitonin was 24 pg/ml (normal: 0–50 pg/ml). Wrist X-ray was compatible with a bone age of 6 months. ECG was within normal limits.

Management
The patient was hydrated with intravenous fluids (3500 ml/m^2/day) and was given furosemide (1 mg/kg/dose) three times daily. Prednisolone was given at a dose of 2 mg/kg/day. Dietary calcium and vitamin D were reduced. Serum calcium was decreased with this therapy, but increased again within several days (Fig. 1). Salmon calcitonin 4 U/kg twice daily intramuscularly was started. Serum calcium gradually decreased to the normal levels. Nausea and vomiting were relieved. Calcitonin therapy was continued for 15 days and stopped. The calcium value on the 15th day of treatment was 9.8 mg/dl. Serum calcium did not increase again (Fig. 1). 25 OH vitamin D level was found to be 40 ng/ml on follow-up 2 months after treatment.

Discussion
Vitamin D intoxication is a rare and life threatening clinical situation. It causes marked and prolonged hypercalciuria by increasing intestinal calcium absorption and bone resorption. Hypercalcæmia may result in nephrocalcinosis, nephrolithiasis, renal failure, and other systemic complications.1

The patient was administered 1 200 000 U vitamin D


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Calcium intoxication

Orally dose 2 months, resulting in the symptoms of hypervitaminosis D like nausea, vomiting, lethargy, and growth retardation.

Classically, hypervitaminosis D was treated using hydration and diuretics. Other pharmacological agents such as glucocorticoids and bisphosphonates are associated with long-term side-effects. Oral bisphosphonates are generally unsuccessful in the initial treatment of hypercalcemia, but are effective in maintenance therapy. Salmon calcitonin has been known to initiate a rapid lowering of serum Ca, within 24 h, but requires injections every 12–48 h.\(^2^3\)

Initially, we used hydration and diuretics. Unfortunately, the calcium level was not improved. Loop diuretics may cause increase in the excretion of the calcium. The other classical agent for the treatment of hypercalcemia is corticosteroids, which may be about calcium homeostasis by influencing calcium level directly and by prolonging the effect of calcitonin.\(^4\)

Consequently, on the fourteenth day of the treatment, when we observed the failure of the conventional methods of treatment, we decided to use salmon calcitonin.

Previous trials in human beings with calcitonin showed good results in hypercalcemic states, like hyperparathyroidism, vitamin D sensitivity, hypophosphatasia, prolonged immobilization, and malignancies.\(^3^4\) Additionally, salmon calcitonin was used successfully in experimental intoxication in dogs.\(^5\)

To the best of our knowledge this is the first case of vitamin D intoxication treated with salmon calcitonin. We believe, it is a safe alternative to the treatment of vitamin D intoxication unresponsive to conventional treatments.

References