Vitamin D toxicity due to self-prescription: A case report

Javid Rasool Bhat, Sajad Ahmed Geelani, Afaq Ahmad Khan, Reshma Roshan, Santosh Govind Rathod

Department Clinical Hematology, SKIMS, Srinagar, Jammu and Kashmir, India

Abstract

Apart from maintaining healthy bones, vitamin D is also required for cell differentiation, cell growth inhibition, and immune modulation. Vitamin D deficiency is common in the Indian subcontinent. Vitamin D presenting toxicity, leading to hypercalcemia, acute kidney injury, and altered sensorium is very rare. Here we present a case of a 65-year-old man who presented to emergency with persistent vomiting, altered sensorium, and acute kidney injury. The cause of which was an unchecked intake of vitamin D for non-specific musculoskeletal pain. When treated with intravenous fluid, diuretics, calcitonin, and steroids, the patient improved clinically. Therefore, for any patient presenting with persistent vomiting, altered sensorium, and hypercalcemia, with normal to low parathyroid hormone levels, a diagnosis of an overdose of vitamin D should be considered. Early treatment of this condition not only improves the symptoms but also prevents further kidney damage.

Keywords: Acute kidney injury, altered sensorium, hypercalcemia, PTH, vitamin D toxicity

Introduction

Vitamin D is important for maintaining bone mineralization and serum calcium level. Vitamin D deficiency defined as serum [25-(OH) D] <20 ng/mL, and insufficiency concentration of 21‑29 ng/mL. Vitamin D toxicity when the level >150 ng/mL.[1] Recent research work shows that vitamin D deficiency is associated with an increased risk of autoimmune disorder, chronic obstructive pulmonary disorder, cancer, and metabolic disease. There is growing awareness about vitamin D deficiency, and related health problems among general population. People are taking vitamin D orally or intramuscularly in mega doses as a supplement without adequate medical monitoring and indication. This leads to vitamin D toxicity causing hypercalcemia, acute kidney injury, and altered sensorium. Vitamin D is fat-soluble and remains in the body for long time and is released slowly. People are not aware of this phenomenon. The present case illustrates Vitamin D toxicity due to self-prescription of the drug without medical indication and its level monitoring.

Case Report

A 65-year-old male, with no underlying co-morbid condition, was presented with vomiting for 2 days, and altered sensorium for 3 days. There was no history of fever, headache, seizure, and cough. On clinical examination, the patient was afebrile, drowsy, and disoriented with GCS‑E3V4M5, 12/15. No neck rigidity was observed. The laboratory investigation showed the following results: hemoglobin 11.2 g/dL, white blood cell counts 7.2 × 10^3/mm^3, platelet count 242 × 10^3/mL, blood urea nitrogen 95 mg/dL, creatinine 5.1 mg/dL, sodium 144 mEq/L, potassium 3.7 mEq/L, serum calcium 14.4 mg/dl, phosphorous 4.8 mEq/L, serum bilirubin 0.50 mg/dL, protein 6.2 g/dL, albumin 3.3 g/dL, globulin 2.7 g/dL, alanine transaminase.
22 IU/L, aspartate transaminase 20 IU/L, alkaline phosphatase 67 IU/L, angiotensin-converting enzyme was 16.20 mg/dL, and intact parathyroid hormone was 3.20 pg/mL. Magnetic resonance imaging (MRI) of the brain and cerebrospinal fluid study was normal with negative viral panel and cryptococcal antigen. Serum protein electrophoresis did not show an M band and whole-body MRI was normal. Bone marrow aspiration showed 2% plasma cells. Ultrasound of neck was unremarkable. Ultrasound scanning showed that both kidneys were normal as well as urine routine microscopy was normal too. However, vitamin D (25 hydroxy vitamin D) was 218 ng/mL. [Table 1]. The detailed history of the patient revealed that he was suffering from prolapsed intervertebral disc for which he had been prescribed vitamin D and calcium tablets. He was told to take injections of vitamin D 50000 IU per week for a total of 6 weeks. However, he lost to follow up and continued to take vitamin D injections every week for 1 year for non-specific musculoskeletal pain. This lead to the diagnosis of vitamin D toxicity. For hypercalcemia, he was treated with intravenous normal saline 3 L/day, diuretics, calcitonin, and dexamethasone. He showed clinical improvement. His serum calcium and creatinine levels were monitored regularly; they gradually decreased to normal levels in the next 10 days. The patient was discharged with a prescription of a calcium-restricted diet and advised not to take vitamin D supplementation, including the reduction of dietary intake, avoiding exposure to sunlight, and maintaining good hydration. After 1 month of follow up, the patient is doing well.

Discussion

Vitamin D is an important prohormone. Fat-soluble in nature and plays an important function in the human body like bone mineralization, calcium levels, cell growth, and immunomodulator. The recommended daily allowance of vitamin D is 800 IU/day and the upper limit of intake of vitamin D is 4000 IU/day as per guidelines by the Institute of Medicine and the Endocrine Practice Committee. Vitamin D toxicity when level >150 ng/mL. Being a fat-soluble in nature, vitamin D remains for a long time and is slowly released in the body. There is growing awareness in the general population about the importance of vitamin D for normal functioning of the body and health-related problems due to its deficiency. It has led to aggressive intake of vitamin D without medical indication and monitoring its level in the body.

In our case, the patient took vitamin D injection 50000 IU per week for a period of 1 year because of non-specific musculoskeletal pain. Clinically, vitamin D intoxication presents with the following symptoms and signs like hypercalcemia, nausea, vomiting, polyuria, dehydration, altered sensorium, acute kidney injury, pancreatitis, and short QT interval. In this present case patient presented with acute kidney injury, hypercalcemia, and altered sensorium.

The differential diagnosis of multiple myeloma, gastrointestinal, breast, lung malignancy, lymphoma, Sarcoidosis, vitamin D toxicity, and parathyroid adenoma considered.

In our case, there was no M band on serum electrophoresis. Bone marrow examination showed only 2% plasma cells only. Serum ACE level was normal. MRI of the whole body was normal. parathyroid hormone was reduced. Vitamin D (25 hydroxy vitamin D) was 218 ng/mL.

Hypercalcemia due to Vitamin D toxicity results from increased concentration of vitamin D metabolites reaching the nuclear Vitamin D receptor and causing exaggerated gene expressions in dose depending manner. The mechanism behind this is the direct action of 25-OH D and other vitamin D metabolites on the 1, 25-(OH)2 D3 receptor and the other is displacement of 1, 25-(OH)2 D3 from D-binding protein by the high 25-OH D levels. This leads to an increase in the concentration of active, free 1, 25-(OH)D3 levels. Vitamin D toxicity treated with vigorous hydration, diuretic, calcitonin, high dose steroid, and biphosonate. Dietary restriction calcium, avoiding sun exposure to light and discontinuation of vitamin D supplementation.

Primary care physicians are the first point of contact to patients in the health system. Increasing awareness among primary care physicians regarding the toxicity of mega doses of vitamin D and cautious use of vitamin D supplements will prevent this condition, and spread awareness among the general population.

Conclusion

Physicians should keep the differential diagnosis of vitamin D toxicity when the patient comes to an emergency with persistent vomiting and hypercalcemia and altered sensorium.

Table 1: Labrotary Parameters

| Investigation                        | Real time value | Interpretation |
|--------------------------------------|-----------------|----------------|
| Serum calcium                        | 14.4 mg/dl      | high           |
| Serum iPTH                           | 3.20 pg/ml      | low            |
| Bone marrow aspiration and biopsy    | No abnormal cell detected | normal        |
| MRI brain, thorax, abdomen           | Unremarkable study | normal          |
| Serum electrophoresis and immunofixation | No M band | normal        |
| CSF study                            | Normal cytology, negative for HSV, cryptococcus | normal |
| ACE                                  | 18 mg/dl        | normal         |
| Vitamin D                            | 218 ng/ml       | Toxic level    |

Table 1: Labrotary Parameters

PTH=parathyroid hormone, MRI=magnetic resonance imaging, CSF=cerebrospinal fluid
in the presence of normal to suppressed parathyroid hormone. Promptly treatment of this condition improves symptoms and avoids further damage to the kidney.

**Key points**

1. Vitamin D rarely causes hypercalcemia leading to acute kidney injury and encephalopathy.
2. Being fat-soluble in nature it is slowly released into the body so a close monitoring of patients is needed.
3. Primary care physicians should prescribe Vitamin D in medical indication, and monitor its level cautiously in order to avoid toxicity.

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**Conflicts of interest**

There are no conflicts of interest.

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