Case Report

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A Rare Case Report of Acute Vitamin D Toxicity due to Suicide Attempt

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Abstract

Acute vitamin D toxicity is an uncommon event that might happen especially owing to attempted of suicide. Vitamin D toxicity could induce hypercalcemia, Acute kidney injury, and cardiac problems in patients. A 49-year-old Iranian man who has a known case of cerebellar palsy and major depressive disorder refers to us with an issue of calcium – vitamin D tablets overdose due to suicide attempt. He admitted with impression of acute vitamin D toxicity. After treatment with hydration, dialysis, Lasix, calcitonin, Denosumab, and Zoledronic Acid patient clinics improved significantly. Physicians must treat vitamin D overdose patients immediately to prevent secondary adverse effects of hypercalcemia.

Keywords: Case report, hypercalcemia, suicide attempt, vitamin D toxicity

Introduction

Vitamin D is categorized as a fat-soluble vitamin that remains long time in humans' adipose tissue and is released in the body slowly (1, 2). Vitamin D improves humans' immune system and also prevents cell mutations, thus it could prevent many types of malignancies (3). In addition, vitamin D is mandatory for bone mineralization, skeletal firmness, and prevention of osteoporosis (1, 3). Not only in Iran but also in many countries a huge number of individuals suffer from insufficient levels of vitamin D as a global issue (4, 5). Therefore, it is common to purchase vitamin D supplements as on-the-counter medication or online (3, 4). By using the unmonitored and unprescribed amount of vitamin D, drug toxicities might be occurred (2, 6). Many studies mentioned that vitamin D levels for adults should be between 30 pg/ml and 60 pg/ml and the above level of 150 pg/ml is categorized as a toxic level that is harmful to the patient hemostasis, thus controlling the amount of vitamin D level is literally mandatory and helpful to prevent further medical conditions (1, 7, 8).

Individuals who suffered from toxic levels of vitamin D mainly presented with nausea, vomiting, headache, sensorium, loss of conciseness, drowsiness, polyuria, and in rare cases seizure (5, 6). Furthermore, toxic levels of vitamin D could affect different aspects of the human body. Hypercalcemia, short QT interval on electrocardiogram (ECG), pancreatitis, and acute kidney injury (AKI) are the main significant harmful consequences of hypervitaminosis D that must be treated immediately (3, 5, 7-9).

In many cases process of vitamin D toxicity happens chronically as a result of using uncontrolled amounts and self-prescribing amount of vitamin D supplements without any medical indication for a long period of time (4). Herein, in the current novel case report our aim is to present a patient who experienced vitamin D toxicity acutely as a consequence of suicide attempt.

Case Report

A 49 years old Iranian married man known case of cerebral palsy (CP) and major depressive disorder (MDD) was referred to Loghman Hakim Hospital, Tehran, Iran with refer reason of a high level of calcium and vitamin D owing to poisoning with 60 tablets of vitamin D - calcium, an unknown amount of ibuprofen 400mg and adult cold tablets. Moreover, he used Bupropion as a past drug history. As well, in his social history he did not use alcohol or cigarette and his family history was negative. At the time of admission, his blood pressure was 100/54, temperature 36.8 degrees

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Celsius, respiratory rate 17, heart rate 80, and O2 saturation 96%. In addition, although the condition of the patient was ill, he had a normal clinical examination, his neurological examinations such as deep tendon reflux, responses to light, and so on were literally normal. He is oriented to time, place, and person, however, he answered to our question with delay. Also, his Glasgow Coma State was 15 out of 15, and his muscle power was 4 out of 5. The patient did not have neck rigidity, headache, nausea, vomiting, and sensorium. The patient's laboratory investigation (table 1) depicted the following abnormal results: Calcium 17.7 mEq/L, Vitamin D level (25-OH vitamin D3) 2407 ng/ml, Blood Urea Nitrogen (BUN) 77 mg/dl, Creatinine (Cr.) 2.4 mg/dl, and LDH 781. His Vessels Blood Gas (VBG) showed ph 7.46, co2 51.1, hco3 36.9, and base excess 11. Also, his Urine Analysis showed a specific gravity of 1.020, urine protein +, and his stool exam was negative. He had a normal parathyroid hormone (PTH) level (6.4 pg/ml), normal electrocardiogram (ECG) without QT prolongation and ST changes, and normal abdomen-pelvic sonography. Finally, he was admitted to the intensive care unit (ICU) with the impression of hypercalcemia on the base of acute vitamin D overdose (toxicity) due to attempt suicide. During the course of hospitalization, as a consult with a nephrologist and an endocrinologist his hypercalcemia was treated with intravenous normal saline 4lit/ day, intravenous Lasix 20 mg, calcitonin, intravenous Denosumab 60 mg, and Zoledronic Acid 4 mg/ 100 ml solution for infusion. Furthermore, during patient hospitalization, he was faced with hypokalemia which was treated with a suitable amount of potassium in his intravenous serum therapy. Also, hemodialysis was performed for his AKI and his electrolyte imbalance. His laboratory data such as complete blood count (CBC), 25-OH vitamin D3, calcium, phosphor, sodium, potassium, BUN, Cr, VBG, and urine analysis monitored regularly. During 14 days, he illustrated clinical improvement and his laboratory data diminished gradually. Also, during the course of admission, several psychologists visited him owing to his attempted suicide. Finally, the patient was discharged after 3 weeks from the internal ward with a prescription for a calcium and vitamin D restricted diet. Moreover, the patient was referred to a psychiatrist for his suicidal ideas. We recommend him for 1 month follow up, however, due to living in other city he lost his follow up.

Table 1: Patient's laboratory test findings at the time of admission

Laboratory test	Result	Unit	Reference Range
CBC			
WBC	7.9	$10^3/\mu L$	4.5 - 11
RBC	5.07	$10^6/\mu L$	4.6 – 6.2
Hemoglobin	14.5	g/dL	13.5 – 17.5
Hematocrit	43	%	45 - 52
M.C.V.	84.81	fL	80 - 100
M.C.H.	28.6	pg	27 - 32
M.C.H.C.	33.72	g/dL	32-36
Platelets	213	$10^3/\mu L$	150 - 450
RDW-CV	14	%	11 – 14.5
Coagulation test			
PT (Patient)	13.4	Sec	10 - 14
INR	1.12		
PT (Control)	12.0	Sec	12.0
PTT	31	Sec	27 - 45
Biochemistry			
BUN	77	mg/dL	19 - 44
Creatinine	2.4	mg/dL	0.9 – 1.4
SGOT	17	IU/L	Up to 43
SGPT	24	IU/L	Up to 40
Alkaline Phosphatase	318	U/L	64 - 319
LDH	781		Up to 480
Calcium	17.7	mEq/L	8.5 – 10.2
Phosphor	3.4	mEq/L	2.6 – 4.5
Sodium	144	mEq/L	135 - 145
Potassium	3.5	mEq/L	3.5 – 5.1
Magnesium	2.0	mEq/L	1.9 – 2.5
25-Hydroxy Vitamin D3	2407	ng/ml	Toxicity: >150
Amylase	42	U/L	Up to 110
Lipase	21	U/L	Up to 60
Hormonal Test			
PTH	6.4	pg/ml	Up to 65
TSH	1.08	µlu/ml	0.3 – 4.04
T3	0.94	ng/ml	0.52 – 1.85
T4	64.1	Ng/ml	51 - 125

CBC: Complete Blood Count, WBC: Wight Blood Cell, RBC: Red Blood Cell, M.C.V.: Mea Corpuscular Volume, M.C.H.: Mean Corpuscular Hemoglobin, M.C.H.C: Mean Corpuscular Hemoglobin Concentration, RDW-CV: Red Cell Distribution Width–Coefficient of Variation, PT: Prothrombin Time, INR: International Normalized Ratio, PTT: Partial Thromboplastin Time, BUN: Blood Urea Nitrogen, SGOT: Serum Glutamic Oxaloacetic Transaminase, SGPT: Serum Glutamic Pyruvic Transaminase, PTH: Para-Thyroid Hormone, TSH: Thyroid Stimulating Hormone

Discussion

As we know, many previous studies depicted that vitamin D toxicity occurred chronically due to unmonitored use of vitamin D supplement both orally as tablet or pearl and/or intravenous as vitamin D ampoule (4, 6, 7). However, the current case report illustrated the patient had been poisoned with vitamin D acutely as a result of overdose of 60 tablets of calcium - vitamin D, so this case report is novel. In addition, as the same as our patient, John P. Lee et. al in their study mentioned that the serum level of vitamin D did not correlate with the patient's symptoms and in acute vitamin D toxicity many patients did not present any specific symptoms, for instance; nausea, vomiting, headache, and sensorium (5).

Vitamin D is a prohormone component that is mandatory for a healthy lifestyle and it affect calcium hemostasis (7, 10). Vitamin D prevents many types of mutation which

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occasionally happen in the pathophysiology of several malignancies (11). As well, it plays a significant role in cell growth. Also, it is quite mandatory for bone mineralization and stability of skeletal structure (12). In addition, vitamin D could improve the human immune system, especially during the COVID – 19 pandemic many studies illustrated the role of vitamin D immunization against the virus (13-15). Therefore, Vitamin D supplements are available as over-the-counter medication in drugstores and it could easily be accessible (4). As a result of that, many clients use vitamin D routinely without any vitamin D level monitoring or prescription (4, 7, 16). Thus vitamin D toxicitymight happen in a number of patients (6). As a consequence of hyper-vitaminosis D, life-threatening hypercalcemia might be happen and it could impact patients' kidneys and AKI as a main secondary disorder might presented (9, 17-19). Also, it could cause patients cardiac ventricular repolarization abnormalities thus cardiac arrhythmias might present in patients (20, 21), therefore immediate treatment of hypercalcemia and vitamin D overdoseis crucial (22).

In the current case report, the patient overdosed calcium - vitamin D tablets for suicidal issues. His hypercalcemia might be affected secondary to an overdose of vitamin D as a secondary metabolic pathway, and also as a main pathway induced by calcium tablets overdose. Although his ECG was normal, as a result of his laboratory data(BUN 77 mg/dl and Cr. 2.4 mg/dl), the patient initially suffered from AKI secondary to his drug overused.Thus, we performed hydration and hemodialysis for his kidney injury and electrolyte imbalance to prevent the worsening of his condition. As well, K. Feghali et. al illustrated that in vitamin D toxicity which induced AKI, a dialysis is a suitable option for patient treatment and it would reduce the side effects of hypervitaminosis D (4). In addition, as a consult with nephrologist and endocrinologist the patient (with a calcium serum level of 17.7 mEq/l)was treated by Calcitonin, Denosumab, and Zoledronic Acid were started to diminish his calcium serum level to prevent other adverse effects of hypercalcemia (23) as the same as M.M. Basso S. et al study (24). Furthermore, the patient was evaluated for his suicide attempt by a psychiatrist and his MDD treatments were prescribed. Also, at the time of discharge he did not suffer from any suicidal ideas.

Finally, we suggest that in acute toxicity with vitamin D, immediate patient management is mandatory and physicians must immediately proceed patient's treatment to prevent life-threatening adverse effect of acute hypercalcemia as a secondary pathway of vitamin D toxicity. Furthermore, when a patient admitted to the emergency ward owing to an attempted suicide, over-the-counter medication such as calcium–vitamin D tablet overdose must be considered.

Conclusion

This case report underscores the critical importance of recognizing that vitamin D toxicity can arise not only from chronic exposure but also following acute ingestion. Emergency physicians should remain alert to the potential for severe, and even fatal, complications associated with single high-dose administration. Early recognition and prompt management of acute vitamin D overdose are essential in mitigating the secondary effects of hypercalcemia and improving patient outcomes.

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