

Hypervitaminosis D Following Overdose in a Patient with SLE

Roy K¹, Basu S², Kundu R³, Das J⁴

¹Dr. Kakali Roy, MBBS, Post graduate trainee of Paediatric Medicine, ²Dr. Surupa Basu, MBBS, MD, Head of the Department of Biochemistry, ³Dr. Ritabrata Kundu, MBBS, MD, Professor of Paediatric Medicine, ⁴Dr. Joydeep Das, MBBS, MD, Assistant Professor of Paediatric Medicine All from the Institute of Child Health, Kolkata, India.

Address for correspondence:

Dr. Kalki Roy

E-mail:kakaliroy.nrs2007@gmail.com

How to cite

Roy K, Basu S, Kundu R, Das J. Hypervitaminosis D Following Overdose in a Patient with SLE. *J Nepal Paediatr Soc* 2016;36(2):196-197.

doi: <http://dx.doi.org/10.3126/jnps.v36i2.14847>

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Abstract

Vitamin D supplementation has come under lime light and is being prescribed randomly. Often physicians prescribe high doses of Vitamin D. Overdosing may be dangerous if not supervised or followed up. We are presenting one child with all features of hypercalcemia due to hypervitaminosis D following overdose. Doctors should be cautious during prescribing high dose of Vitamin D.

Introduction

Vitamin D (Vit-D) is a fat soluble vitamin and only vitamin which is produced endogenously at skin in response to ultraviolet B-light (290–320 nm)¹. It is present in few foods, mainly fish. It has many beneficial effects but excessive Vit-D following excessive overtreatment can cause toxicity which is injurious and even may result death. We are reporting a case of hypervitaminosis-D in a girl with SLE after excessive intake of vitamin D supplementation for bone pain.

The Case

Seven year old girl presented with joint pain for which a local doctor had prescribed Vit D 60,000 IU twice a month. But, due to clerical error by dispensing chemist or caregiver, she instead received the dose twice a day. On day 27 of medication, she presented to us with severe bone pain and all features of hypercalcemia like polydypsia, polyuria and constipation. She had already received a cumulative dose of 32.4 lakhs (=1,20000 *27) IU of cholecalciferol.

On investigation serum calcium was 12.2 mg/dl, SrVit-D was 400 ng/ml. Further evaluation showed ionised Ca 1.5 mmol/L, albumin 4.1 mg/dl, Phosphate 5.29 mg/dl, alkaline phosphatase 151 U/L and parathyroid (PTH) 10 pg/ml. Finally on investigation the cause of joint pain was diagnosed as SLE.

Vit D supplementation was stopped and hypercalcemia was treated by rehydration with normal saline, and intravenous frusemide. Repeat Ca level on third day of admission showed further increase to 13 mg/dl. Thereafter Inj. Zoledronate (Bisphosphonate) intravenous infusion was started. On day 7, symptoms subsided and Ca was 9.7 mg/dl while Vit-D was 315 ng/ml with normalisation of other biochemical parameters.

Discussion

Vitamin D is essential for calcium homeostasis and bone mineral metabolism. It is recommended for the prevention of rickets and bone loss, optimum bone mass formation. Additionally it has potential extra skeletal effects on the neuromuscular and immune system². Most common cause of hypervitaminosis D is exogenous intake either in the form of excessive supplementation of food and multivitamin or intake of over the counter pharmacological preparation of Vit D. The RDAs (recommended dietary allowances) is age specific and is between 400 I.U. to 800 I.U. for pediatric age group. Upper level of tolerance is between 1000-2000 IU³. Where our patient was getting 1,20,000 IU per day which is almost 60 times more than maximum tolerance level.

Cholecalciferol [25(OH)D] is the major form present in circulation and it is the best indicator of body Vit-D status. Serum 25(OH)D levels above 150 ng/ml is considered as toxic⁴. Vitamin D increases serum Ca and phosphate by increasing absorption from gut and reabsorption from kidney, and mobilising bone mineral. Signs and symptoms of hypervitaminosis D because of hypercalcemia are irritability, anorexia, constipation, polyurea, hypotonia and on long standing case it may result nephrocalcinosis and chronic renal failure.

The possible hypothesis of Vit-D toxicity involves increased Vit-D metabolites which reach the nucleus of target cell and cause exaggerated gene expression. Most available preparations contain calcidiol [25(OH)

D] and following overdose blood level increases and exceed Vit-D binding protein and binding capacity and 'free 25,(OH)D' enters the cell where directly affect gene expression⁵.

Treatment includes discontinuing intake of vitamin D and calcium and management of hypercalcemia. Hydration is maintained strictly as hypovolemia may be present which increase non enzymatic absorption of Ca from proximal convoluted tubule. Loop diuretic inhibits absorption of Ca from loop of Henley. Majority of patients improves with this treatment. In unresponsive cases, Bisphosphonate is considered which works by inhibiting osteoclastic activity and thus inhibits bone resorption (which is the dominant mechanism of hypercalcemia in hypervitaminosis D). Our patient did not show clinical or biochemical improvement with initial therapy but improved after zolidronate infusion due to persisting hypervitaminosis D being stored in the fatty tissues and liver⁶. Glucocorticoid that was given to our patient for treatment of her primary problem of SLE also has a known action of decreasing Ca absorption from gut by blocking calcitriol activity⁷.

Conclusion

Prescribing Vit-D is now rampant even for milder symptoms without much awareness and concern for the toxic effects. Administration of Vit-D should be supervised. Doctors should be cautious about dosing and parents should be well counselled about potent side effects of overdosing. Therapy should be followed up with repeat assessments.

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