Hypervitaminosis D presenting as Hypertensive Encephalopathy

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***Abstract*- Vitamin D** deficiency is quiet prevalent in India. This makes many Practitioners to prescribe Vitamin D supplements Emperically. Though there is a significant gap between Vitamin D therapeutic doses and toxic doses, in predisposed individuals excessive intake of Vitamin D can lead to some serious and life endangering side effects.

Here we present a case of 80 year old male patient who was a known case of **diabetes** and **hypertension**. The patients was taking oral supplements of Vitamin D daily which lead to patient presenting to us with Hypertensive Encephalopathy and ultimately Death. This emphasises the need to judiciously use Vitamin D supplements and to remain Vigilant for its side-effects in high risk individuals.

***Index Terms*-** Vitamin D, Hypercalcemia, Hypertension

I. INTRODUCTION

Vitamin D is an important pro-hormone and besides playing an important role in calcium homeostasis and bone mineral metabolism is also important for various fundamental biological functions like cell differentiation ,inhibition of cell growth as well as immune modulation. Daily requirement of Vitamin D is 200-600 Units and skin produces 10,000 units after total body exposure of UV rays. No toxicity occurs till 10,000 units daily and prolonged intake. Toxicity is noted after prolonged daily intake of about 50,000 units. Thus because of this wide therapeutic range Vitamin D is prescribed empirically and unjudiciously. However older individuals or those with renal impairment or PHPT could be predisposed to its toxicity and so care needs to be taken while prescribing Vitamin D in such individuals.

Here we present a case of 80 year old male diabetic and hypertensive who was taking more than normal requirement of Vitamin D daily for almost 6 weeks. Patient developed Hypercalcemic arteriosclerosis which ultimately led to Hypertensive Encephalopathy.

II. CASE REPORT

Mr.BabubhaiParmar, 70 yr old male patient presented with GTCS one episode in morning followed by altered sensorium. There was no history of fall, trauma, fever, focal neurologic deficit, weakness in limbs, loss of bowel bladder function. Patient was a known case of diabetes, hypertension and benign prostatic hypertension on T.velmisartan OD, T. veltam OD.

III. EXAMINATION

On examination patient was conscious, disoriented, irritable.

The other findings were as follows: Pulse: 90/ min, BP: 220/140 mm hg, Temperature: normal. Respiratory system: normal , Cardiorespiratory system: normal, Abdominal system : normal.

Central nervous system: conscious, disoriented.
- irritable
- pupils :B/l normal reacting to light
- plantar: bilateral flexor
- moving all 4 limbs

NCCT Brain: NAD
MRI Brain: NAD
MR Angiography+ venography: NAD
CXR (PA): NAD
XRAY SKULL: NAD

Laboratory investigations revealed following results: Hb:11.5 , WBC: 8230, polymorphs:85. Lymphocytes:12, monocytes: 2. Eosinophils: 1, basophils: 0. MCV: 85, platelet count: 2.02 lakh. PS-Normochromic, normocytic picture.

Urea : 40.38--following HD--->41.6,
Creat: 2.07---following HD--->2.51,
Sodium: 138--following HD---->141,
potassium: 3.25---following HD--->2.52.
Plasma Glucose-223,
S.Acetone-normal.

Bilirubin: Total-1.10,S.G.P.T-23.7, S.G.O.T-27.7, Protein:Total-7.2, Albumin-4.43.
PT: Test-10.7, INR-1.00
S.Amylase:1851
S.Lipase: 163
CPK Total-162------>4104
CPK-MB:11
Calcium: 14.5---following HD---->12.70
S.Uric Acid-3.95
S.Magnesium-1.57
phosphorous:2.15
Trop-I: negative

Vit D(25-OH) : >150
Parathyroid hormone: 14.2
hs CRP:55.7
TSH : 1.888
Urine for Bence jones proteins: negative
Protein Electrophoresis: normal
Urine for myoglobin: >1200
ECG: LVH, T inversion in V1-V6.
2D ECHO: EF:55%, RVSP: 28 mm hg, Grade I diastolic dysfunction.
USG ABDOMEN+ NECK: NAD.

Course in Hospital:

An 80 yr old Male was admitted in civil with history of convulsion. On admission the BP of patient was 220/110 Patient was given IV Labetalol to lower down the BP. Patient was irritable for which he was given IV Seranase. Rountine investigations revealed raised Creatinine suggesting renal failure. S.calcium was done in view of raised creatinine which turned out to be 14.1. Urine was negative for Bence Jones Protein, normal Protein Electrophoresis. This ruled out multiple myeloma.one cycle of HD was done due to raised creatinine.

IV. DISCUSSION

Clinical presentation of Hypervitaminosis D is mainly due to Hypercalcemia and Hyperphosphatemia Most of the patients usually present with nausea, vomiting, diarrhoea, polyuria, reanal failure. Hypercalcemia can also lead to Hypertension and coronary artery disease as in case of our patient. The initial presentation of the patient pointed towards a cerebrovascular accident which was ruled out by imaging studies. Second likely cause of raised calcium in old patients like him can be Multiple myeloma or any occult Metastasis which was ruled out by further investigations. Also PPTH was also ruled out by normal PTH . On proper history taking it was found that patient was taking daily oral supplements of Vitamin D in form of oral sachet.

This case highlights Pragmatic but safe approach while using empirical Vitamin D supplements to avoid any serious adverse effects. Serum Calcium and Renal Function Test should be regularly monitored in high risk patients.

V. KEY MESSAGE

- Vitamin D deficiency is widespread in India. Empirical use of modest doses in low risk individuals is safe.
- Hypervitaminosis D is uncommon but can occur in high risk and elderly patients. Thus regular monitoring of Serum Calcium and Serum Creatinine should be undertaken.
- Avoid high dose supplements in elderly, those with Renal Failure, or those patients with PPTH.

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