HYPERVITAMINOSIS D : AN UNCOMMON REALITY!

Presented By : ZH Mansuri[1], S Dumra[2], BC Kaji[1], HN Buch[1,2].

[1] B J Medical College and Civil Hospital Ahmedabad, [2] Sterling Hospital, Ahmedabad, Gujarat, INDIA

INTRODUCTION

Empirical Vitamin D therapy is widely used in India, in view of high prevalence of Vitamin D deficiency[1] and the high cost of Vitamin D assays. Vitamin D toxicity is uncommon,[2,3] although elderly patients and those with renal impairment or primary hyperparathyroidism (PHPT) are predisposed. We present a patient in whom empirical high-dose Vitamin D therapy led to serious toxicity, raising concerns about its unmonitored empirical use especially when used in high doses..

CLINICAL PRESENTATION

- An 89 year-old lady presented to an orthopaedic surgeon with bone aches and pains. She was suspected to be having Vitamin D deficiency and was prescribed 3 injections of 6mU Vitamin D3 IM monthly.
- 10 days after the 3rd dose she presented with nausea, dehydration, drowsiness and ataxia.
- Laboratory tests on admission as per table 1.
- Abdominal US: Altered cortico-medullary differentiation, suggestive of renal parenchymal disease.

MANAGEMENT

- She was managed with normal saline infusion and intranasal Calcitonin.
- In view of severe hypercalcaemia and worsening renal failure she required haemodialysis which was complicated by disequilibrium syndrome.
- She gradually recovered and was discharged after a week .
- Laboratory results during the hospital stay and on discharge are on table 1.
- 6 weeks following discharge she resumed her daily activities

TABLE1	REFERENCE RANGE	On admission	Post-treatment	Pre-discharge	6 weeks later
S. Calcium (mmol/l)	2.1 – 2.7	4.5	2.7	2.3	2.4
S. Phosphate (mmol/l)	0.8 – 1.4	1.6	1.4	1.3	1.3
S. Magnesium (mmol/dl)	0.6 - 1	0.8	0.8	0.9	0.8
S. Creatinine (mmol/dl)	44 – 130	390	210	200	120
eGFR (ml/min/1.73 m^2)	>90	10	21	21	41
PTH(pmol/ml)	15 – 65	NOT DONE	62.9	58.1	4.7
S. Vitamin D3 (nmol/dl)	75 – 260	>400	>400	>400	>400

LEARNING POINTS

- Empirical use of Vitamin D is a cost-effective option in the developing world, as toxicity is rare due to a significant gap between the usual replacement (1500-2000 IU/day[4]) and toxic doses (>40000 IU/day[5]). However toxicity may occur when high doses are used over a short period of time[4] in high-risk individuals i.e. the elderly and those with renal failure or primary hyperparathyroidism (PHPT)[3].
- Clinical presentation is due to hypercalcaemia and hyperphosphataemia as was seen in our patient[6].
- Prompt treatment with saline diuresis, bisphosphonates, calcitonin and glucocorticoids reverse the clinical and biochemical picture. In the absence of symptoms and significant hypercalcaemia, an observational policy can be maintained.
- This case highlights the need for a pragmatic but safe approach with following precautions;
  - To avoid empirical use of high-dose vitamin D therapy without laboratory confirmation of deficiency.
  - To assess serum calcium and serum creatinine prior to initiation of vitamin D therapy in high-risk individuals.
  - Periodic monitoring of serum calcium during the use of vitamin D replacement in such patients.
- In our patient, primary hyperparathyroidism was excluded retrospectively by normal calcium and normal PTH during the follow up period. However she was elderly with some degree of renal dysfunction and high dose of vitamin D was used all of which combined to lead to vitamin D intoxication.

REFERENCES

1. CV Harinarayan et al, JAPI 2009 Jan ; 57 : Vitamin D Status in India.
2. G Jones et al, Am J ClinNutr 2008;88:582S-586S.
3. JN Hathcock et al, Am J ClinNutr. 2007 Jan;85(1):6-18.
4. Ross AC et al, The National Academies Press, 2011.
5. R Vieth et al, J Bone Miner Res, 2007 ; 22: V64–V68.
6. R Joshi et al, J Trop Pediatr 2009 Dec;55(6):396-398.