International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: SJIF: 5.995
Available Online at www.journalijcar.org
Volume 7; Issue 1(D); January 2018; Page No. 8983-8985
DOI: http://dx.doi.org/10.24327/ijcar.2018.8985.1468

Research Article

AN INTERESTING CASE OF ACHES AND PAINS

Sunil Havannavar*

Sreenivasa Hospital #12, Park Road Balepet Bangalore

ARTICLE INFO

Article History:
Received 19th October, 2017
Received in revised form 10th November, 2017
Accepted 06th December, 2017
Published online 28th January, 2018

Key words:
Hypercalcemia, hypervitaminosis D, skeletal tuberculosis.

ABSTRACT

We report a case who presented with all symptoms of hypercalcemia. Initially hypercalcemia seemed to be iatrogenic secondary to hypervitaminosis D, but she did not respond to steroids. Symptoms and high levels vit D and calcium persisted and clinically her condition was worsening which made us to explore other causes of hypercalcemia. Biopsy of skeletal lesion found on PET scan reviled skeletal tuberculosis which was responsible for ectopic production of vit D leading to hypercalcemia. Rare but not uncommon cause of hypercalcemia.

INTRODUCTION

Aches and pains especially in elderly individuals are the common presenting symptoms in out patients department. Most of the times these are treated with empirically with calcium and vitamin D supplements. When symptoms persist or worsen despite of treatment they need detailed evaluation. Hypercalcemia is a relatively common clinical problem. It results when the entry of calcium into the circulation exceeds the excretion of calcium into the urine or deposition in bone. This occurs when there is accelerated bone resorption, excessive gastrointestinal absorption, or decreased renal excretion of calcium.[1] In some disorders, however, more than one mechanism may be involved. Hypercalcemia has been described in patients with most granulomatous disorders.[2] Hypercalcemia secondary to hypervitaminosis D due to ectopic production of 1, 25(OH) 2D3 in which the regulation of the synthesis is quite different from that in the normal kidney, is rare but not uncommon.[3-4]

Case

- 59 year old female with no co-morbid illnesses presented with history of aches and pains. She suffered severe back ache, muscle weakness, lethargy and fatigue. Occasionally associated with pain abdomen constipation and nausea all since two months. She had trivial fall and was treated for L1 fracture a month prior to this admission. Bone densitometry done at that time revealed severe osteoporosis (T score -3.3). She was treated with analgesics (NSAIDS), calcium and vit. D supplements for the same. Examination at admission showed blood pressure of 160/100, thorough systemic examination was normal no lymphadenopathy, organomegay or pallor was noted. Her complete blood counts were normal except for high ESR (67), creatinine was 3.3, blood urea was 68 but she was nonoliguric did not have signs of volume overload or uremia. Serum electrolytes, liver functions and chest X ray were normal and serologies were negative for HIV, HbsAg and HCV. Her serum calcium levels were high 13.6, phosphorous was normal. Other tests like thyroid functions, cortisol levels, blood sugar, urine proteins, cardiac evaluation and ultrasound abdomen were normal. At this point we were dealing with a "symptomatic hypercalcemia" in a patient with documented osteoporosis along with non oliguric renal failure. She had history of NSAID abuse for pains which could be possible reason for her renal impairment.
- She was treated initially with isotonic saline and furosemide. Fluid and electrolyte balance was maintained.
- We started evaluating her for hypercalcemia. More than 90 percent of hypercalcemia cases are either primary hyperparathyroidism or malignancy. The humoral hypercalcemia (mediated by PTHrP) of malignancy[5-6] implies a very limited life expectancy while, primary hyperparathyroidism has a benign course.[7] The other etiology for hypercalcemia is Vitamin D intoxication. Most common cause of hyper vitaminosis D is iatrogenic[8], others are granulomatous disorders (Sarcoidosis, Berylliosis, Tuberculosis) and Hodgkin's

Corresponding author:* **Sunil Havannavar
Sreenivasa Hospital #12, Park Road Balepet Bangalore

lymphoma. Rare causes include drugs and immobilization.

- Our patient's PTH and urine calcium levels were normal so hyperparathyroid state was ruled out. Bone marrow biopsy and Sr. protein electrophoresis were negative for multiple myeloma. Serum ACE levels were normal hence ruling out possibility of sarcoidosis.
- Serum vit d levels were high (108.6). Possibility of iatrogenic hypervitaminosis D with hypecalcemia was considered she was started on short course of steroids for hypervitaminosis D, analgesics (non- NSAID) and was advised to review after three weeks with repeat vit d and calcium levels.
- On follow up she continued to have symptoms and calcium level and vit D were high however her renal parameters were normal. In view of persistent hypercalcimia, PET scan was done to rule out occult malignancy.
- PET scan showed metabolically active lesions in bones and few lymph nodes however no abnormal FDG uptake or enhancing lesion was noted to suggest primary lesion. To find out exact pathology CT guided biopsy of the skeletal lesion was performed and sent for histopathology.
- Report revealed granulomatous inflammation suggestive of tuberculosis.
- She was started on ATT along with symptomatic treatment. During follow up after two months of ATT she is symptomatically better sr. calcium is 9. She was suffering from disseminated skeletal tuberculosis leading to hyper calcitrol causing hypercalcemia.

DISCUSSION

- Hypervitaminosis D is very uncommon with the doses that are usually prescribed in day to day practice. Iatrogenic vitamin D toxicity due to empirical administration of very high doses of intramuscular vitamin D injections at frequent intervals is not uncommon, especially in elderly. This kind of hypervitaminosis D responds well to steroids and stopping further supplementation of vitamin D. Hypercalcemia of vitamin D intoxication results from increased intestinal absorption of calcium and from the direct effect of 1.25[OH] 2 D3 to increase resorption of bone in severe cases. Hypercalcemia is responsible for producing most of the symptoms of vitamin D toxicity. Early symptoms of vitamin D toxicity include gastrointestinal disorders like anorexia, diarrhoea, constipation, nausea, vomiting and bone pains.[9] Vitamin D stores in fat may be substantial, and vitamin D intoxication may persist for weeks after Vitamin D ingestion is terminated. Such patients are responsive to glucocorticoids. Prednisone may help reduce plasma calcium levels by reducing intestinal calcium absorption.
- Hypercalcemia is associated with variety kinds of granulomatous diseases, including sarcoidosis[10] and tuberculosis.[11-15] The granulomatous tissue is believed to be the site of the ectopic production of 1, 25(OH) 2D3 in which the regulation of the synthesis is quite different from that in the normal kidney[8-9]. Hypercalcemia associated with elevated serum 1.25(OH) 2D3 levels is also found in patients with lymphomas and some other malignancies.
- Other granulomatous conditions causing hypercalcemia include Berylliosis, coccidioidomycosis, histoplasmosis, candidiasis, Crohn's disease], Langerhans-cell histiocytosis (also called histiocytosis X, and including eosinophilic granuloma), silicone-induced granulomas and Wegener's granulomatosis.
- Our patient presented with all symptoms of hypercalcemia secondary to hypervitaminosis D initially though looked like iatrogenic hypervitaminosis D due to supplements she did not respond to steroids. Symptoms and high levels vit D and calcium persisted and clinically her condition was worsening which made us to explore other causes of hypercalcemia. Biopsy of skeletal lesion found on PET scan reviled skeletal tuberculosis which was responsible for ectopic production of vit D leading to hypercalcemia. Rare but not uncommon cause of hypercalcemia.

CONCLUSION

Hypercalcemia is a common clinical problem, mostly asymptomatic but when symptomatic needs proper treatment evaluation of the etiology. Even though malignancy and hyperparathyroidism contribute to 90% of the cases of hypercalcemia, other cases like hypervitaminosis D is not uncommon. Iatrogenic hypervitaminosis leading to hypercalcemia is well known facts and many cases have been discussed but granulomatous condition like tuberculosis leading ectopic synthesis of vit d causing symptomatic hypercalcemia is rare. All hypercalcemia cases needs thorough evaluation as common treatable conditions like tuberculosis can have uncommon presentation.

Bibliography

1. Bushinsky DA, Monk RD. Electrolyte quintet: Calcium. *Lancet* 1998;352:306-11
2. Adams JS. Vitamin D metabolite-mediated hypercalcemia. *Endocrinol Metab Clin North Am* 1989; 18:765.
3. Jones G. Vitamin D in the 21st century: An Update-Pharmacokinetics ofvitamin D toxicity. *Am J Clin Nutr* 2008; 88:582S-586S.
4. Khazai N, Judd SE, Tangpricha V. Calcium and vitamin D: skeletal and extra skeletal health. *Curr Rheumatol Rep* 2008;10:110-117
5. Nielson PK, Rasmussen AK, Feldt-Rasmussen U, *et al*. Ectopic production of intact parathyroid hormone by squamous cell lung carcinoma in vivo and in vitro. *J Clin Endocrinol Metab* 1996; 81:3793-96.
6. Dorman EB, Yang H, Vaughan CW, *et al*. The incidence of hyeprcalcemia in squamous cell carcinoma of the head and neck. *Head Neck Surg* 1984;7:95-98.
7. Kumar R. Vitamin D and calcium transport. *Kidney Int* 1991;40:1177- 89
8. Hathcock JN, Shao A, Vieth R, Heaney R. "Risk assessment for vitamin D". *Am J Clin Nutr* 2007;85:6-18
9. Joshi R. Hypercalcemia due to Hypervitaminosis D: report of seven patients. *J Trop Pediatr* 2009; 55:396-398.

10. Winnacker JL, Becker KL, Katz S. Endocrine aspects of sarcoidosis. *N Engl J Med* 1968; 278:427.
11. Abbasi AA, Chemplavil JK, Farah S, *et al*. Hypercalcemia in active pulmonary tuberculosis. *Ann Intern Med* 1979; 90:324
12. Shai F, Baker RK, Addrizzo JR, Wallach S. Hypercalcemia in mycobacterial infection. *J Clin Endocrinol Metab* 1972; 34:251.
13. Gkonos PJ, London R, Hendler ED. Hypercalcemia and elevated 1, 25-dihydroxy vitamin D levels in a patient with end-stage renal disease and active tuberculosis. *N Engl J Med* 1984; 311:1683.
14. Cadranel J, Garabedian M, Milleron B, *et al*. 1,25(OH)2D2 production by T lymphocytes and alveolar macrophages recovered by lavage from normocalcemic patients with tuberculosis. *J Clin Invest* 1990; 85:1588.
15. Shek CC, Natkunam A, Tsang V, *et al*. Incidence, causes and mechanism of hypercalcaemia in a hospital population in Hong Kong. *Q J Med* 1990; 77:1277.

How to cite this article:

Sunil Havannavar (2018) 'An Interesting Case of Aches and Pains', *International Journal of Current Advanced Research,* 07(1), pp. 8983-8985. DOI: http://dx.doi.org/10.24327/ijcar.2018.8985.1468

****