



A Rare Case Report on Chronic Kidney Disease and Hypervitaminosis D

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ABSTRACT: INTRODUCTION: Vitamin D and sunlight have an important role in regulating homeostasis of calcium and bone strength within our body, but an excessive amount can lead to hypervitaminosis D, which is very rare to see in chronic kidney disease since kidneys fail to activate vitamin D. There has recently been an increase in the number of cases of hypervitaminosis because of an increase in the number of prescriptions of vitamin D for the treatment of hypovitaminosis D. The intake of large quantity of vitamin D₃ (or vitamin D₂) leads to hypercalcemia and hypercalciuria

CASE REPORT: A female adult of 80 years old was brought to the hospital and the patient was diagnosed with Hypervitaminosis D with comorbid conditions of Chronic kidney disease (CKD -III), hypothyroidism, hypertension and obstructive sleep apnea. The treatment was immediately started and the patient's serological reports were taken where vitamin D and calcium levels were found to be very high, and phosphorous and uric acid levels were also elevated. The blood urea and creatinine levels were elevated while serum sodium level was low. The patient was treated with hydrocortisone, levothyroxine, aspirin and atorvastatin, metoprolol, frusemide, lactulose, febuxostat, budesonide, ipratropium bromide and salbutamol, and injection heparin. The patient also underwent Hemodialysis. The patient was completely stabilized and was discharged.

CONCLUSION. The patient was admitted in the hospital for 8 days and was treated with corticosteroids, anti-hypertensives, anti-platelet, hypolipidemic, diuretic, xanthine oxidase inhibitor, bronchodilators and anticoagulant and underwent haemodialysis. After 8 days of treatment, the patient was stabilized and discharged.

KEY WORDS: Hypervitaminosis D, Hypercalcemia, Hypercalciurea, Chronic kidney disease, hypothyroidism, hypertension.

I. INTRODUCTION

Vitamin D and sunlight have an important role in regulating homeostasis of calcium and bone strength within our body, but an excessive amount can lead to hypervitaminosis D, which is a rare but potentially serious condition [1]. Vitamins such as Vitamin D that are fat soluble, because of their potential to aggregate within the body, have a higher potential to cause toxicity than vitamins that are water soluble [2]. The synthesis of Vitamin D occurs from Ergocalciferol caused by the sun light's ultraviolet rays. They engage within the metabolism of our body either as a co-factor of an enzyme or as a prosthetic group [3]. Over the previous couple of decades, vitamin D's interest has been increased significantly. Apart from playing roles that are important in calcium homeostasis and mineralization of bone, vitamin D has now been identified in playing a part in the immune system, cancer prevention and cardiovascular health. [4]. Concerns and recommendations over the deficiency of vitamin D has resulted in the extensive usage of vitamin D supplements, with doses upto 60,000IU/unit in practice from infantile age. Doses higher than 50,000 IU/day increase levels of 25(OH) vitamin D to greater than 150 ng/ml and have been linked with hypercalcemia and hyperphosphatemia [5]. The intake of large quantity of vitamin D₃ (or vitamin D₂) leads to hypercalcemia and hypercalciuria because of the production of excessive amounts of 25-hydroxyvitamin D [25(OH)D] that bind to the vitamin D receptor, although with lesser affinity than the active form of



the vitamin, 1,25(OH)2D, and the production of 5,6-trans25(OH)D, which strongly binds to the vitamin D receptor in comparison to 25(OH)D [6]. There has recently been an increase in the number of cases of hypervitaminosis because of an increase in the number of prescriptions of vitamin D for the treatment of hypovitaminosis D [7]. Majority of these cases are a result of prescribing inappropriately, and the usage of unlicensed preparations or high-dose over-the-counter preparations [8]. Most of the reports of acute toxicity of vitamin D have involved serum values of 25(OH)D greater 140 ng/mL,8 with the chief clinical indication being hypercalcemia and its related symptoms [9].

II. CASE REPORT

An 80 year old female patient was admitted in a tertiary care hospital with the chief complaints of drowsiness since the past 4 days. She also complained of anorexia and burning micturition. She has a past medical history of hypertension, hypothyroidism, chronic kidney

disease (stage III), obstructive sleep apnoea and left diaphragmatic hernia. Her past medication history suggested that she was on medications like Inj. Arachitol and Vitamin D supplementations from long time. She was also on medications like Tab. Met XL 50 mg (Metoprolol) and Tab. Thyronorm 100 mcg (Levothyroxine). As the patient was suffering from obstructive sleep apnoea, she has been using Auto CPap at home with oronasal mask. Her general physical examination revealed a pulse rate of 78 bpm, respiration rate 20/min, blood pressure with a systolic pressure of 130 over diastolic pressure of 80 mmHg. Her spO2 level was 92% on RA. Her first set of blood tests demonstrated Haemoglobin 11.1 g/dl, Red blood cells 3.87 million/cumm, White blood cells 6900 cells/ cumm and platelet count 2.34 lakhs/cumm. Her complete urine examination had revealed pus cells 10-15 /hpf and epithelial cells 6-10/hpf. She had undergone routine biochemical investigations which was repeated for 1 week and revealed the levels in Table 1. lab data day wise

LAB DATA	DAY-1	DAY-2	DAY-3	DAY-4	DAY-5	DAY-6
Blood urea	82 mg/dl	86 mg/dl	35 mg/dl	36 mg/dl	81 mg/dl	129 mg/dl
Serum creatinine	4.4 mg/dl	4.9 mg/dl	2.4 mg/dl	2.3 mg/dl	3.3 mg/dl	3.6 mg/dl
Serum sodium	133 meQ/ L	136 meQ/ L	135 meQ/ L	137 meQ/ L	138 meQ/ L	135 meQ/ L
Serum potassium	4.1 meQ/ L	3.8 meQ/ L	3.4 meQ/ L	3.6 meQ/ L	3.7 meQ/ L	3.0 meQ/ L
Serum Calcium	17.1 mg/dl	11.1 mg/dl	13.4 mg/dl	12.4 mg/dl	12.1 mg/dl	11.0 mg/dl

Table 1. day wise lab data

Further investigations revealed a normal Thyroid Stimulating Hormone (TSH)1.89 mU/L. Her vitamin D levels were 171.5 ng/ml which was high. Her PTH levels were 18.6 pg/ml which was normal. Her serum phosphorus levels were normal i.e., 4.5 mg/dl. Her uric acid levels were higher i.e. 8.7 mg/dl while the normal range is between 2-6 mg/dl. Her serum Lipase and serum Amylase levels were higher than the normal range i.e., 893 U/L and 132.8 U/L respectively. Her Ultra sound Abdomen and Pelvis had revealed Grade II renal parenchymal changes and renal cortical cysts in both kidneys. Her 2d echo had revealed Grade I LV diastolic dysfunction. Her ECG had shown prolonged PR interval and atrial premature complex. From the subjective and objective She was treated with the medications as follows:

findings the patient was diagnosed to have been suffering from Hypervitaminosis D (Hypercalcemia). So she was treated to achieve patient specific goals which include:

- To relieve breathlessness.
- To reduce drowsiness.
- To decrease serum calcium levels.

Disease specific goals were to:

- To improve the quality of life.
- To decrease the morbidity and mortality of the patient by least intrusive means possible.
- To prevent the development of further complications of the disease such as arrhythmia, kidney stones, kidney damage, calcification of arteries and soft tissues, excessive bone loss.



S L. N O	CURRENT MEDICATIONS	DOS E	R O U T E	FRE QUE N C Y	D A Y- 1	DAY- 2	DAY- 3	D A Y- 4	DAY- 5	DAY - 6	DAY -7	DA Y- 8
1.	TAB. ELTROXIN (LEVOTHYROXINE)	100mcg	PO	OD	√	√	√	√	√	√	√	√
2.	TAB. ECOSPRI NAV (ASPIRIN +ATORVASTATIN)	75/10mg	PO	OD	√	√	√	√	√	√	√	√
3.	TAB.MET XL (METOPROLOL)	50mg	PO	OD	√	√	√	√	√	√	√	√
4.	INJ. LASIX (FRUSEMIDE)	40mg	IV	BID		√		√	√	√	√	√
5.	SYP. DUPHALAC (LACTULOSE)	30 ml	PO	TID	√	√	√	√	√	√	√	√
6.	INJ. HYDROCOROTONE (HYDROCORTISONE)	100mg	IV	QID				√	√	Frequency changed.		
	INJ. HYDROCOROTONE (HYDROCORTISONE)	100mg	IV	BID						√	√	√
7.	TAB. FEBUGET (FEBUXOSTAT)	40mg	PO	BID	√	√	√	√	√	√	√	√
8.	DUOLIN RESPULE (IPRATROPIUM BROMIDE + SALBUTAMOL)	1 respule	INH	QID		√	√					
9.	BUDECORT	1 respule	INH	TID		√	√					



	(BUDESONIDE)	le						O P				
10	INJ. HEPARIN	5000 U	SC	TID		√	√	√	√	√	√	√

Table 2. Treatment chart

Day wise progression chart shows in table.3 below

DAY 1	Advised CBP, RFT, Serum phosphorous, Serum Uric acid, Serum Calcium, ABG, Intact PTH, 2 D ECHO, ECG, Vitamin D, Chest X ray. Results revealed severe hypercalcaemia. Advised 2 sessions of Hemodialysis.	BP: 130/70mmHg PR: 76bpm
DAY 2	Patient is stable. 2 D ECHO done. Repeat Serum Calcium for every 12 hours daily. Results revealed HIGH VITAMIN D LEVELS. Advised nebulisation	BP: 200/120mmHg PR: 102bpm
DAY 3	Advised Hemodialysis 2 sessions. Plan hydration 50 ml/ hr NS. Patient is stable. Advised to monitor vitals.	BP: 130/70mmHg PR: 90bpm
DAY 4	Up on examination altered sensorium. No fluid overload. Patient is stable.	BP: 150/100mmHg PR: 82bpm
DAY 5	Advised fluid restriction up to 1L/ day. Advised hydration 100 ml/ hr NS. Patient is symptomatically better.	BP: 120/80mmHg PR: 75bpm
DAY 6	C/o headache and weakness. Change Inj Hydrocort QID to BID. Patient is stable	BP: 140/90mmHg PR: 90bpm
DAY 7	Advised hydration 50 ml/ hr NS. Patient is stable.	BP: 130/80mmHg PR: 80bpm
DAY 8	Patient is symptomatically better. Patient is stable. DISCHARGED.	BP: 140/80mmHg PR: 83bpm

TABLE.3 PROGRESSION CHART

The condition of the patient was made stable and discharged with medications like

1. Tab. Wysolone 30 mg PO OD (PREDNISOLONE)
2. Tab. Met XL 50 mg PO OD (METOPROLOL)
3. Tab. Febuget 40 mg PO OD (FEBUXOSTAT)
4. Syp. Duphalac 30 ml PO TID (LACTULOSE)
5. Tab. Ecosprin AV 75/10 mg PO OD (ASPIRIN+ ATORVASTATIN)
6. Tab. Thyronorm 100 mcg before breakfast (LEVOTHYROXINE).

Suggestions were made that if her vitamin D levels decreased, it should not be treated with conventional vitamin d but to use 1-25 OH vitamin d instead due to shorter half life.

III. DISCUSSION

A female adult of 80 years old was brought to the hospital and the patient was diagnosed with Hypervitaminosis D with co-morbid conditions of Chronic kidney disease (CKD –III), hypothyroidism, hypertension and obstructive sleep apnea. The treatment was immediately started and the patient's serological reports were taken where vitamin D and calcium levels were

found to be very high, and phosphorous and uric acid levels were also elevated. The blood urea and creatinine levels were elevated while serum sodium level was low. Her serum Lipase and serum Amylase levels were higher than the normal range. The complete blood picture showed low levels of Red blood cells and Haemoglobin. The complete urine analysis showed elevated pus cells and epithelial cells. The ultrasound of the abdomen and pelvis was done and showed Grade II renal parenchymal change and renal cortical cysts in both kidneys, and 2D ECHO showed Grade 1 LV Diastolic dysfunction. The ECG showed prolonged PR interval and atrial premature complex. The patient was treated with hydrocortisone, levothyroxine, aspirin and atorvastatin, metoprolol, frusemide, lactulose, febuxostat, budesonide, ipratropium bromide and salbutamol, and injection heparin. The patient also underwent Hemodialysis. The patient was completely stabilized and was discharged.

IV. CONCLUSION

Hypervitaminosis D is a rare but potentially serious condition that is characterized



by excessive amounts of vitamin D in the body. The studied case had complaints of drowsiness, anorexia and burning micturition. The patient was diagnosed with Hypervitaminosis D with comorbid conditions of Chronic kidney disease (CKD –III), hypothyroidism, hypertension and obstructive sleep apnea as per clinical presentations. The patient was admitted in the hospital for 8 days and was treated with corticosteroids, anti-hypertensives, anti-platelet, hypolipidemic, diuretic, xanthine oxidase inhibitor, bronchodilators and anticoagulant and underwent haemodialysis. After 8 days of treatment, the patient was stabilized and discharged.

REFERENCES

- [1]. Uzma Tazeen, Tun Aung (2018) A case of hypervitaminosis D in an older patient
- [2]. Sutirtha Chakraborty, Ajoy Krishna Sarkar, Chandramouli Bhattacharya, Prasad Krishnan, Subhosmito Chakraborty (2015) A Nontoxic Case of Vitamin D Toxicity Laboratory Medicine, Volume 46, Issue 2, May 2015, Pages 146–149
- [3]. Elango et al / International Journal of Biomedical Research 2015; 6(03): 151-154 Hypervitaminosis
- [4]. Kornelia Galior, Stefan Grebe, and Ravinder Singh (2018) Development of Vitamin D Toxicity from Overcorrection of Vitamin D Deficiency: A Review of Case Reports Nutrients. 2018 Jul 24; 10(8):953.
- [5]. Aditi Das¹, Shaad Abqari², Kamran Afza (2018) Vitamin D Intoxication: Consequence of Misconception - A Case Report <http://dx.doi.org/10.21276/ijcmr.2018.5.8.16>
- [6]. Peter J. Tebben, Ravinder J. Singh, and Rajiv Kumar (2016) Vitamin D-Mediated Hypercalcemia: Mechanisms, Diagnosis, and Treatment Endocrine Reviews, Volume 37,5,521–547
- [7]. Tatiana Aporta Marins¹ Tatiana de Fátima Gonçalves Galvão¹ Fernando Korkes¹ Domingos Augusto Cherino Malerbi¹ Arnaldo José Ganc¹ Davi Korn¹ Jairo Wagner¹ João Carlos de Campos Guerra¹ Wladimir Mendes Borges, Filho¹ Fábio Teixeira Ferracini¹ and Hélio Korkes¹ (2014) Vitamin D intoxication: a case report
- [8]. Peter N. Taylor¹ and J. Stephen Davies².(2018) A review of the growing risk of vitamin D toxicity from inappropriate practice Br. J Clin Pharmacol 2018 Jun; 84(6): 1121-1127
- [9]. Daniel V. Dudenkov, MD, Barbara P. Yawn, MD, MS, FAAFP, Director of Research, [...], and Tom D. Thacher, MD Changing Incidence of Serum 25-Hydroxyvitamin D Values Above 50 ng/mL: A 10-Year Population-Based Study 2015 May; 90(5):577-86.