



## ENHANCING DYSELECTROLYTEA: A RARE PRESENTATION OF VITAMIN D DEFICIENCY

### General Medicine

**Bhushan D** Assistant Professor, Department of General Medicine, AIIMS, Patna.

**Kumawat P** Intern, Department of General Medicine, AIIMS, Patna)

**Verma P** Intern, Department of General Medicine, AIIMS, Patna)

### ABSTRACT

Vitamin D deficiency causes multisystem manifestations, directly or indirectly. This not only affects skeletal system, but cardiovascular, respiratory and neurocognitive system are also get affected. Here we present a case of elderly lady, who presented with ATT induced hepatitis and dyselectrolytemia. Workup for electrolyte imbalance ruled out other causes. She was found to have concomitant severe vitamin D deficiency and correction of which improve the condition of patient and her serum electrolytes markedly.

### KEYWORDS

vitamin D deficiency, hypomagnesimia, hypokalemia

### CASE

A 75 yr old lady admitted with swelling over neck since one month. She was shown to a private physician who started anti tubercular medicines. On seventh day of treatment she developed severe vomiting. She was admitted outside and investigated. Her liver enzymes were elevated. She was put on modified ATT and discharged in a week. After 2 days she again started vomiting. She came to our hospital and gets admitted. On examination her neck swelling had already subsided and all other findings were within normal limits. Investigations revealed deranged LFT with SGOT – 837 iu/l, SGPT – 232 iu/l, ALP – 555, Total Bilirubin – 3.29 mg% .She was provisionally diagnosed as ATT induced hepatitis and modified ATT continued. Upper GI endoscopy was done and found to be normal. Serum cortisol level was 10.6 normal. She was discharged after stabilization.

After fifteen days she again presented with complain of vomiting. She was not tolerating anything orally so her modified ATT was also stopped. Patient was found to have hypokalemia and hypomagnesaemia (serum potassium – 3.2 meq/dl and serum magnesium – 1.4meq/dl). Arterial blood gas analysis showed mild metabolic alkalosis (pH: 7.5, HCO<sub>3</sub>: 28, PCO<sub>2</sub>:38). Her urinary potassium and calcium were within normal limits. She was treated with intravenous magnesium sulfate and potassium chloride. Patient also found out to have low serum calcium – 6.6 meq/dl and serum phosphorus – 2.3 meq/dl. Replacement for calcium was done intravenously. Her serum vitamin D was - <4.2 ng/ml and serum intact PTH was (586.6 pg/ml), Aldosterone level was 9.6ng/dl and Plasma renin activity <0.10 ng/ml/hr (to rule out Gitlemans syndrome). In view of severe Vitamin deficiency as a cause of resistant hypokalemia, she was given 2ml IM injection (6 lac units) of Arachitol. At the time of discharge vomiting had subsided, patient was tolerating oral feeds and serum potassium levels had improved. She was discharged with diagnosis of severe vitamin D deficiency, hypocalcemia, hypomagnesemia, hypokalemia and advised for vitamin D3, calcium and syp potklor. She is still in follow up, and does not show any recurrence of lymphadenopathy and tuberculosis at other site.

### DISCUSSION:

Vitamin D deficiency is prevalent in India as well as United States. (1) Causes includes defective production, lack of dietary intake, accelerated loss, and resistance to its biologic effects. In elderly insufficiency of vitamin D production in skin and decreased absorption through intestine leads to marked decrease in its level.(2) It can further deteriorate the situation if person receive drugs causing hepatic enzyme induction like rifampicin, phenobarbitone and phenytoin as in our case. Hypocalcemia is a common manifestation of vitamin D deficiency. However severe deficiency can leads to hypomagnesemia.(3) This low magnesium leads to impaired parathyroid function and further decrease the calcium level. Hypomagnesemia also leads to hypokalemia. So while correcting serum potassium level we take care of serum magnesium also. Other important causes of hypomagnesemia are persistent vomiting, malabsorption syndrome, genetic magnesium wasting syndrome(

gitelmans syndrome, barter syndrome), hyperaldosteronism, SIADH, pancreatitis, and recovery from DKA. WE ruled out all these by measuring serum glucose, urinary potassium, urinary calcium and serum intact PTH. (4)

Treatment includes magnesium supplementation before potassium correction. Simultaneously vit D supplementation helps in early recovery.

**Conflict of interest:** none

**Funding:** not applicable

### REFERENCES

1. van Schoor NM, Lips P. Worldwide Vitamin D status. *Best Pract Res Clin Endocrinol Metab.* 2011;25:671–80.
2. Holick MF: Vitamin D deficiency. *N Engl J Med* 357(3):266,2007
3. Holick MF, Chen TC. Vitamin D deficiency: A worldwide problem with health consequences. *Am J Clin Nutr.* 2008;87:1080S–6S
4. Garg MK, Tandon N, Marwaha RK, Menon AS, Mahalle N. The relationship between serum 25-hydroxy Vitamin D, parathormone and bone mineral density in Indian population. *Clin Endocrinol (Oxf)* 2014;80:41–6.