

## **An Unusual Case of Constipation Unmasking Underlying Tuberculosis: Case Report**

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**Abstract:** In clinical practice, constipation is a common complaint and rarely secondary to endocrine or metabolic causes. We present here a case of an elderly male who presented with constipation was found to be having hypercalcemia as the cause of constipation.

**Keywords:** Constipation, Hypercalcemia, Tuberculosis

### **INTRODUCTION**

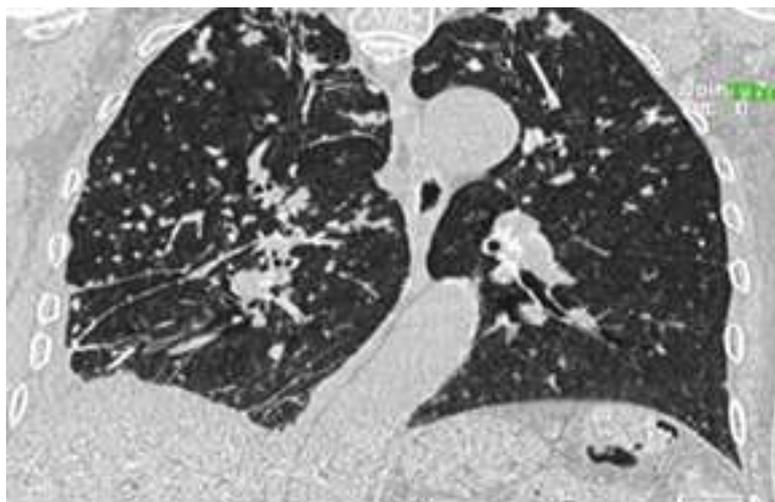
Constipation is a common complaint in clinical practice, although rarely an endocrine or metabolic problem is the cause of the same. We present here a case of severe constipation and mild renal dysfunction secondary to hypercalcemia and how we reached at the exact cause of hypercalcemia where the patient was apparently asymptomatic from that organ system point of view involved.

### **CASE REPORT**

A 75 year old monk was admitted with complaints of constipation for the past 3-4 months who was on Laxatives and intermittent enema with partial benefit. He had no other cardio-respiratory symptoms. He did not have fever. He had mild anorexia and no significant weight loss. On examination he was well built and moderately nourished elderly male. He had borderline hypertension. Systemic examination revealed mild right sided pleural effusion and other systems were unremarkable. His investigations revealed normal hemogram, normal thyroid function studies, mild renal dysfunction, normal urinalysis and moderate Hypercalcemia. He was treated with hydration for hypercalcemia. His serum protein electrophoresis was inconclusive for M band. Intact parathyroid hormone and Immunofixation electrophoresis were sent for

work-up of hypercalcemia. His Immunofixation electrophoresis was normal which ruled out monoclonal gammopathy as a cause of Hypercalcemia in our case.

The intact parathyroid hormone (PTH) was low suggestive of non-PTH related hypercalcemia. A 1, 25 (OH)<sub>2</sub> Vitamin D<sub>3</sub> level was sought. By this time his hypercalcemia was not getting optimized despite aggressive hydration and renal failure was worsening. His 1, 25 (OH)<sub>2</sub> Vitamin D<sub>3</sub> level was available after a week which was very much elevated. He was treated with intravenous fluids and other supportive medications. A differential Diagnosis of Sarcoidosis, Tuberculosis, Lymphoma was considered for our patient in view of high 1, 25 (OH)<sub>2</sub> Vitamin D<sub>3</sub>. His pleural fluid was aspirated for diagnosis which was transudate in nature. HRCT Chest was performed for our patient. It showed multiple nodular lesions noted along the broncho-vascular bundles noted in bilateral upper lobes, right middle lobe and anterior segment right lower lobe. Fibrotic streaks were also noted in bilateral apical region. Few calcified bilateral hilar lymph nodes noted (Fig. 1). He did not have any peripheral palpable lymph nodes. His CT Chest and abdomen did not reveal any clue of Lymphoma. His bone marrow aspiration and biopsy were normal. We were left either sarcoidosis or pulmonary tuberculosis.



**Fig. 1: CT Chest - Multiple nodular lesions along the broncho-vascular bundles in bilateral Upper Lobe / Rt Middle Lobe and anterior segment of Rt Lower lobe. B/L Calcified hilar lymph nodes(not enlarged). D/D Sarcoidosis / Tuberculosis.**

Our patient of discussion did not have fever, significant anorexia or weight loss. Absolutely there were no respiratory symptoms. Hence a provisional diagnosis of sarcoidosis was considered and our patient was started on prednisolone (0.5 mg / Kg / day). After 4 days of starting prednisolone he started having high grade fever with chills and cough with expectoration. There was worsening anorexia and drowsiness. He was initiated on nasogastric feeding. His sputum samples were sent for Zeil-Neilson's staining and gramstain. His sputum was positive for acid fast bacilli (two samples). He was started on Anti-Tuberculous Therapy (ATT). After about 15 days of starting steroids and 1 week of ATT his fever subsided, sensorium improved. Correspondingly his hypercalcemia and renal dysfunction normalized. His constipation got relieved. Steroids were withdrawn completely at the end of 4 weeks. He completed the full course of ATT for 6 months and at the completion of ATT he was free from constipation, renal functions remained normal and there was no hypercalcemia.

## DISCUSSION

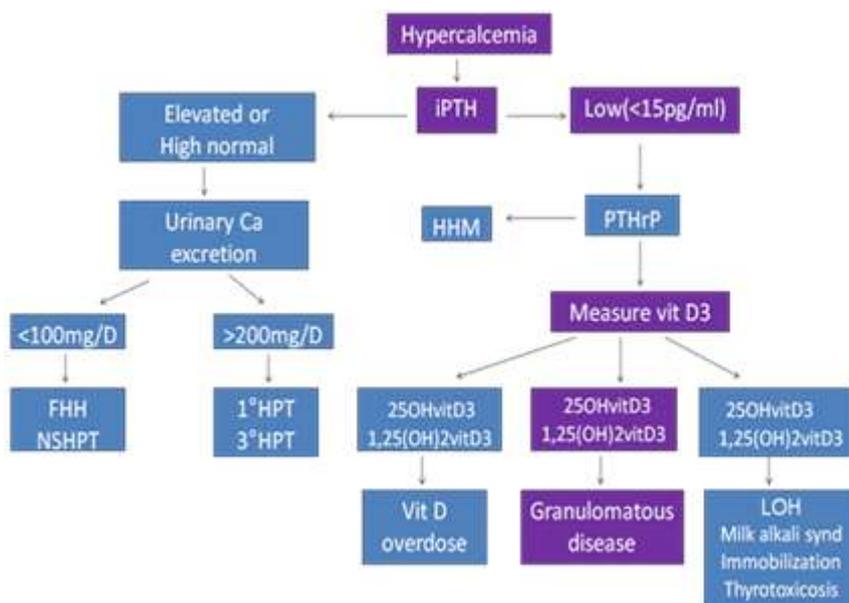
Constipation is a common complaint in clinical practice which sometimes only is secondary to endocrine or metabolic causes [1]. The elderly male who presented with constipation was found to be having hypercalcemia as the cause of constipation. On evaluation his intact-PTH was low, investigations for monoclonal gammopathy were negative and 1,25 (OH)<sub>2</sub> Vitamin D<sub>3</sub> was very much elevated. Elevated 1,25 (OH)<sub>2</sub> Vitamin D<sub>3</sub> narrowed down to few causes

of hypercalcemia, like Sarcoidosis, tuberculosis and lymphoma. Lymphoma was excluded with CT abdomen and pelvis and Bone marrow examination. There were no palpable peripheral lymph nodes. There were no features of active tuberculosis like fever, cough, anorexia or significant weight loss.

Probable diagnosis of sarcoidosis was considered and steroids were started as patient's renal functions were worsening secondary to hypercalcemia. The interesting and crucial turn noted in the case was unmasking of the symptoms with initiation of steroids which gave us the golden opportunity to prove AFB. The paradox of steroids unmasking fever comes as a curious clinical observation because in other situations they usually mask the symptoms. Waning immunity with age and immunological anergy in geriatrics population could be possible reasons and explanation for the co-synchronous occurrence of hypersensitivity and atypical disease state and steroids exhibiting such paradoxical action.

Hypercalcemia is seen in ~ 25 % cases of tuberculosis but ~ 7 to 12 % cases only are symptomatic.

Hypercalcaemia is not uncommon among patients with newly diagnosed TB, but rarely symptomatic [2]. Hypercalcemia in sarcoidosis is secondary to increased synthesis of 1, 25 (OH)<sub>2</sub> Vitamin D<sub>3</sub> by the activated macrophages [3].



**Fig. 2: Evaluation of Hypercalcemia. (FHH - Familial hypocalciuric hypercalcemia; HHM – Humoral hypercalcemia of malignancy; HPT – Hyperparathyroidism; LOH - Localized osteolytic hypercalcemia; NSHPT - Neonatal severe hyperparathyroidism )**

### CONCLUSION

The purpose of presenting this case is its rarity of presentation and the diagnosis of sarcoidosis is always by exclusion. The occurrence of sarcoid-tuberculosis is also talked of which may be difficult to be proven.

Limitations of our case are we did not have a histopathological evidence of granuloma and Kviems test was not performed.

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