ARIPET OF PRES	IGINAL RESEARCH PAPER	General Medicine
	ERCALCEMIA: AN UNUSUAL SENTATION OF TUBERCULOSIS	KEY WORDS: Hypercalcemia, Tuberculosis, 1- alpha hydroxylase, Renal insufficiency, Granulomatous disease.
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Tuberculosis (TB), a granulomatous disease, is known to cause hypercalcemia. Hypercalcemia and its manifestations can be the presenting complaints in patients with active TB1. However, the incidence of hypercalcemia in TB patients is rare, with incidence ranging between 2% and 25%, of which very few are symptomatic. A 62 year old presented with symptoms of epigastric and loin pain, intermittent fevers, progressive swelling of both loer limbs, tarry stools, dyspnea, decreased urineoutput and significant weightloss. A chronic smoker and pain killer abuse. Patient was found to have severe anemia due to iron deficiency with blood loss due to esophageal and duodenal ulcers, hypercalcemia, renal insufficiency. There was a suspicion of chronic kidney disease or malignancy. Further evaluation of hypercalcemia, lead us to a diagnosis of active pulmonary TB. Patient improved with aggressive hydration, hemodialysis with packed cell transfusion, parenteral iron, bisphosphonate and anti-tuberculosis therapy.

INTRODUCTION:

ABSTRACT

Tuberculosis (TB) is a major public health problem, especially in the developing countries like India. TB may cause hypercalcemial, which causes renal insufficiency and may lead to wrong diagnosis in the setting of anemia and renal calculi. Though Hypercalcemia is rare, it is a well recognised complication of active TB. The incidence of hypercalcemia in TB varies widely, because of variations in Vitamin D and calcum intake, the amount of sun exposure and differences in the population.

CASE REPORT :

A 62 year old male presented with ill-defined burning type epigastric pain, bilateral intermittent non-radiating loin pain, intermittent low grade fever, projectile bilious vomitings, progressive pedal edema, dyspnea, generalized weakness, tarry stools, nocturia and decreased urine output, decreased appetite and weight loss of about 5kg in a month. Regular smoker- 10 pack years and takes alcohol occasionally, known hypertensive and history of non-steroidal anti-inflammatory drug abuse.

On examination patient was pale with B/L Pitting pedal edema, weighing 41kg, height of 156cm, and a low BMI 16.84kg/m2, pulse 106/min, blood pressure 160/100mm Hg. Barrel shaped chest, diffuse rales and ronchi were present on both sides on chest auscultaion. No loin tenderness or mass per abdomen and no organomegaly.

Laboratory workup showed serum urea of 86mg/dl, creatinine of 5.9 mg/dl, haemoglobin 5.1 gm/dl, ESR 150mm/hr, peripheral smear read- Microcytic Hypochromic RBC. Low serum iron of <20mcg/dl, serum phosphorus of 4.9mg/dl, elevated serum uric acid of 11mg/dl, corrected serum calcium of 11.8mg/dl, serum albumin of 1.8mg/dl, urine albumin +. Ultrasonogram of Abdomen showed bilateral grade I renal parenchymal changes, right kidney measuring 8.1*3.5cm and left 9.4*4.3cm, with bilateral multiple renal calculi measuring 7-9mm, positive stool occult blood. Endoscopy showed linear ulcerations in distal esophagus and large D cap clean base ulcer and on histological examination, there is nonspecific duodenitis with mild villous atrophy and no evidence of malignancy. Sptum for Acid Fast Bacilli was positive (3+). Chest radiograph showed bilateral fibrosis of upper zones with cavitatory and bronchiectatic lesions. Contrast Computed Tomography scan of Chest showed Traction bronchiectasis, fibrotic bands with tree in bud opacities s/o active koch's, tree in bud nodules in both lower lobes, centrilobular emphysema in both lungs. Tests for other causes of hypercalcemia were negative, normal iPTH 35.5pg/ml, elevated Vitamin D3 and negative for paraproteinemias. Initially, Hemodialysis with blood transfusion was done along with aggressive hydration for hypercalcemia measures and Zolendronic acid was given., Parenteral Iron for iron deficiency. Started on antituberculosis therapy with Rifampicin 450mg, Isoniazid 300mg, Pyrazinamide 1000mg, Ethambutol 600mg (alternate day). Two weeks after the initiation of the treatment, corrected calcium was 9.6mg/dl and serum creatinine decreased to 1.3mg/dl.

DISCUSSION:

Hyperparathyroidism and malignancy account for 80-90% of cases of hypercalcemia2. As our patient has normal parathyroid hormone level, which would mean a parathyroid independent pathology as the cause of hypercalcemia. Parathyroid independent hypercalcemia should be evaluated for disseminated malignancy or multiple myeloma or granulomatous disorders. The tests for paraproteinemias were negative and no evidence of malignancy on contrast computed tomography of chest and abdomen. Of the granulomatous disorders, sarcoidosis is the common cause of hypercalcemia, followed by TB, silicosis1. The incidence of hypercalcemia in TB is 2% to 25%. There is increased conversion of 25-hydroxyvitamin D to 1,25-dihydroxyvitmin D due to increased 1 alpha-hydroxylase activity in the alveolar macrophages and granulomas. Hypercalcemia causes direct renal vasoconstriction and a decrease in glomerular filtration

PARIPEX - INDIAN JOURNAL OF RESEARCH | Volume-9 | Issue-2 | February - 2020 | PRINT ISSN No. 2250 - 1991 | DOI : 10.36106/paripex

rate3 and gastrointestinal symptoms (nausea, anorexia, constipation and gastric mucosal ulcers) and neuropsychiatric symptoms. Anemia in this case can be attributed to anorexia, blood loss due to esophageal and duodenal ulcers. In longstanding cases, nephrolithiasis is due to chronic hypercalciuria3. Treatment hypercalcemia is mainly hydration with normal saline and treating the cause4. The presence of hypercalemia in patients with pyrexia of unknown origin, TB should be a differential.

CONCLUSION:

Hypercalcemia and its complications can be presenting manifestations in tuberculosis even though its rare. Aggressive hydration4 is enough as most cases of TB have mild hypercalcemia and anti-tuberculosis therapy. Hypercalcemia can lead to acute deterioration of renal function in Chronic Kidney Disease. Reversible causes of renal insufficiency should be excluded before labeling as Chronic Kidney Disease.



Figure 1: Chest Radiograph





Figure 2: Sections of Computed Tomography Chest

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