Volume 06, Issue 04 (July-August 2023), PP 44-49 **www.ijmsdr.org**
ISSN: 2581-902X

International Journal of Medical Science and Dental Research

A Case Report- Sarcoidosis Presenting As Hypercalcemia

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ABSTRACT: Sarcoidosis is a chronic granulomatous disease that affects multiple organs systems in the body with unknown cause. Most of the patients are free of clinical symptom, sarcoidosis should be considered in differential diagnosis if non-caseous granuloma is noted in biopsies. Here we present a case of sarcoidosis related hypercalcemia due to increased level of 1,25-dihydroxyvitamin D level that increases the absorption of calcium from intestine.

I. INTRODUCTION:

Sarcoidosis is systemic disease affects multiple organs of an unknown cause. The lung disease is common and is responsible for most of the morbidity and mortality associated with sarcoidosis.

Hypercalcemia in sarcoidosis is due to the uncontrolled synthesis of 1,25-dihydroxyvitamin D3 by macrophages. Sarcoidosis is a systemic disease included in the wide group of interstitial lung diseases (ILDs) that commonly require a multidisciplinary approach for diagnosis and clinical management. Due to its systemic nature, sarcoidosis needs to be assessed by a holistic approach that should include all possible localizations and expressions of disease. Changes in calcium metabolism, including hypercalcemia and hypercalciuria, are quite common.[1-5]

1,25-dihydroxyvitamin D3 leads to an increase absorption of calcium in intestine and to an increased resorption of calcium in the bone, 1,25-dihydroxyvitamin is an inhibitor of interleukin-2 and interferon -gamma synthesis. This two cytokinins are take part in granuloma formation in sarcoidosis.[6]

Here we present a case of 57 year old male patient with sarcoidosis having hypercalcemia. HRCT chest revealed B/L hilar and paratracheal lymphadenopathy and on biopsy of axillary lymph node pathology report revealed non-necrotizing granuloma.

II. CASE REPORT:

57year old man came to emergency with complaint of chest pain and abdominal pain since 1 week and altered sensorium since 2-3 days. We shifted this patient in ICU, He had past medical history significant for hypertension, taken T. amlodipine as antihypertensive medication regularly. Patient had complaint of chest pain on left side and constipation intermittently since 1 month. The pain was insidious in onset and associated with poor oral intake, nausea, vomiting. He lost 10 kg weight in previous 2 month, now lost 2 kg more in a week, he denied history of recent travelling, sick contacts, diarrhea, fever, chills, dysuria or hematuria.

Family and social history were insignificant on general examination a blood pressure of 150/94 mmHg, a pulse rate 68 beats per minute, afebrile and epigastric tenderness on palpation. Patient having submandibular, cervical and axillary lymphadenopathy. ECG was normal, Cardiac markers are negative.

Initial laboratory workup revealed hemoglobin of 11.5mg/dl, TLC count 10,570 ul, platelets count 3,02 lac cell/ul, CRP Quantitative is 43.37mg/L (0-6 mg/L normal) and ESR is 70 mm (Normal 0-20mm), serum creatinine 2.62mg/dl(baseline 1.2mg/dl), serum calcium 12.3mg/dl (Ref 8.5-10.5mg/dl), serum phosphorus 5.12 mg/dl (Ref 4.8-5.6mg/dl). Initial evaluation for hypercalcemia revealed low parathyroid hormone (PTH) level of 8.4 pg/ml (Normal 15-65pg/ml), 25-hydroxyvitamin D level of 27.6ng/ml (Ref 30-100) and 1,25-dihydroxyvitamin D level elevated to 93.10pg/ml (Ref 19.90-79.90), 24 hourly urinary calcium levels 300mg/24 hours (20- 275 mg/24hr).

The complain of abdominal pain, acute kidney injury and hypercalcemia guided further evaluation for multiple myeloma. Serum protein electrophoresis and urine protein electrophoresis revealed no monoclonality and urine for Bence Jones Protein was negative. He was started on intravenous fluids for hypercalcemia. Patient having bilateral submandibular, sublingual, axillary lymphadenopathy on ultrasonography. HRCT chest revealed enlarged bilateral hilar and paratracheal lymph nodes. (Fig. I & II) Sputum for AFB was negative. Mantoux test done which was negative. FNAC taken from axillary lymph nodes revealed granulomatous non-necrotizing lymphadenitis. Serum angiotensin- converting enzyme level increases to 84 U/L (normal ref 7-65 U/L). FNAC and increase angiotensin converting enzyme (ACE) level with hypercalcemia suggestive of sarcoidosis. Patient was managed with iv fluids and iv steroids.

III. DISCUSSION:

Sarcoidosis is a multi-system, granulomatous disease without a known etiology. Most of the studies suggest that the pathogenesis is related to an exaggerated immune response to an environmental factor, microbe or antigen in a genetically susceptible individual.[7]

The reported incidence of hypercalcemia associated with sarcoidosis varies from 2% to 63%. Hypercalcemia tends to be transient in subacute sarcoidosis, but in chronic sarcoidosis, depending on the activity of the disease, the serum level of calcium may fluctuate.[8]

Calcium ion play an important role in cellular function, physiological processes, cardiac contractility and blood coagulation. Therefore, the extracellular concentration of calcium is tightly regulated through feedback mechanism that involve PTH and 1,25-dihydroxyvitamin D.[9]

Diagnosis of sarcoidosis can be challenging when hypercalcemia is the presenting symptom. Although hypercalcemia is well studied electrolyte abnormality in sarcoidosis, it is seen in lot of cases, with severe hypercalcemia of more than 14mg/dl rarely reported.

Diagnosis of sarcoidosis in hypercalcemic patient is difficult as the presentation may mimic other granulomatous disease and malignancies. The presentation of AKI, anemia, and hypercalcemia in the patient reported above initially raises clinical suspicion of multiple myeloma, Tuberculosis was also high on differential

on further evaluation showed lymphadenopathy bilateral hilar, paratracheal on HRCT cheat, Biopsy of axillary lymph node show non-necrotizing granuloma, increased ACE level confirm the diagnosis of sarcoidosis.[9]

Treatment of hypercalcemia in sarcoidosis consists mainly of iv fluids and corticosteroids. Furthermore, hypercalcemia is an indication to start steroid therapy even in the presence of only mild symptoms. However, other causes of granulomatous diseases including histoplasmosis and tuberculosis should be ruled out prior to steroids. steroids are the mainstay of treatment in sarcoidosis, they play an important role in managing hypercalcemia by inhibiting macrophage 1-hydroxylase activity.[9]

Treatment of hypercalcemia depends on the serum level of hypercalcemia and its persistence. Generally sarcoidotic patients should be advised to avoid sun exposition to reduce vitamin D3 synthesis in the skin, to reduce fish oils that are rich of vitamin D and to produce more than 2 liters urine a day by adapting fluid intake. Although severe hypercalcemia seems to be rare, Glucocorticosteroid treatment should be started if corrected total calcium level rises beyond 3 mmol/l. If hypercalcemia is symptomatic, treatment should be started even at lower levels. Glucocorticosteroids act by inhibition of the overly 1 alpha-hydroxylase activity of macrophages. Alternatively, treatment with chloroquine or ketoconazole can be established. If isolated hypercalciuria without hypercalcemia is present with evidence for recurrent nephrolithiasis, patients can be treated with a thiazide diuretic.[10]

Ketoconazole has also been reported to decrease the use of high-dose corticosteroids. Some case series report promising results with anti-tumor necrosis factor monoclonal antibody, infliximab, in treating refractory hypercalcemia.[9]

IV. CONCLUSION:

Sarcoidosis is a chronic granulomatous disease that can affect multiple organs but most common is pulmonary involvement. The goals of sarcoidosis management are to prevent or control organ damage, relive symptoms and improve patients quality of life. Emergency clinician are trained in diagnosing and treating the cause of hypercalcemia, but they also need to consider one differential diagnosis as sarcoidosis. It is low risk disease that carries a high degree of long term morbidity.

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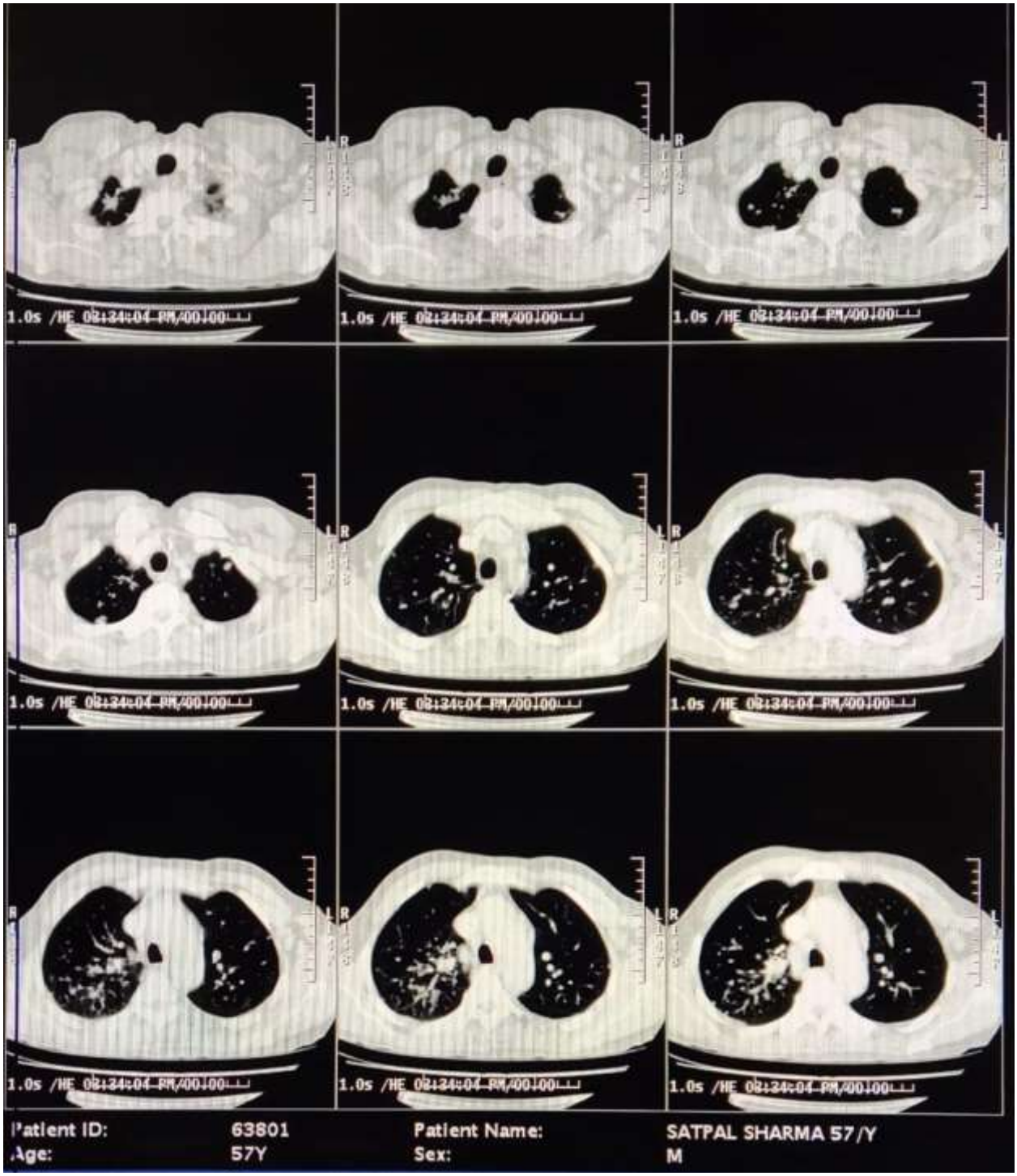

FIGURE I: HRCT THORAX (LUNG WINDOW)

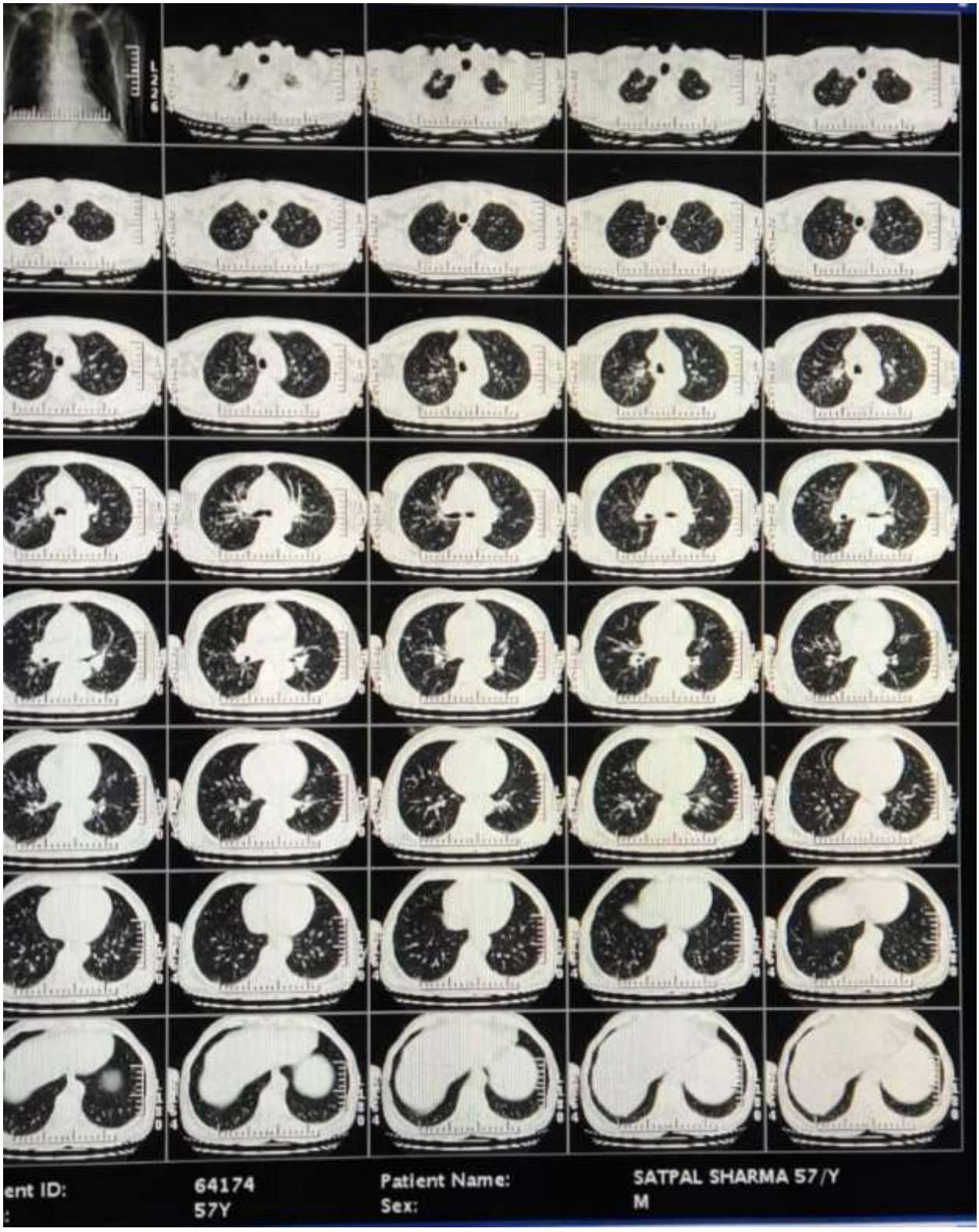

FIGURE II: HRCT THORAX

FIGURE III: AXILLARY LYMPHADENOPATHY