A Case of Tuberculosis Presented with Syncope and Hypercalcemia

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Introduction:

Tuberculosis may occur with atypical presentations in many patients without the classic clinical signs. Hypercalcemia is a common clinical problem for primary hyperparathyroidism (PHPT), but after excluded PHPT, the malignancies are responsible for the 90% of the cases presented with hypercalcemia (1). Another rare cause of hypercalcemia is granulomatous diseases such as tuberculosis (2). Herein, an elderly patient with tuberculosis presented with syncope and hypercalcemia will be discussed in lights of current literature.

Case report:

Seventy-year-old female patient was admitted to the geriatrics out-patient clinic with complaints of syncope, fainting, weight loss and weakness. She had hypertension diagnosis. Physical examination revealed cervical approximately 2x2 cm, submandibular 1x3 cm lymphadenopathies. Examinations which were made in another center to find out the causes of syncope, including echocardiography, electroencefalography, carotid doppler USG and brain magnetic resonance imaging showed no significant pathology. Laboratory investigations revealed that hemoglobin: 8.4 g / dl, creatinine: 1.11 mg / dL, calcium: 11.2 mg / dL, albumin: 3.8 g / dl, 25 OH

vitamin D: 9.5 g / l, serum parathyroid hormone: 6.45 pg / ml, respectively. 24 hour urinary calcium was 319.2 mg/d. In serum protein electrophoresis monoclonal gammopathy could not be detected. Endoscopy and colonoscopy revealed no evidence other than gastritis. Hypodense 17 mm nodule in thyroid gland, right hilar 22 mm and left hilar 20mm lymphadenopathies, parenchymal nodules smaller than 5 mm on both the pulmonary parenchyma were detected in thorax-abdomen-pelvic CTs (Figure 1). These nodules did not show significant uptake on positron emission tomography-CT imaging. Thyroid fine needle aspiration biopsy and bronchoscopic biopsy revealed no malignant lesions. Serum ACE level was found to be normal. Because of continued clinical suspicion, excisional biopsy of right submandibular lymph node was performed. Biopsy revealed caseified necrotizing granulomatous lymphadenitis (Figure 2). Although she was given intravenous hydration and diuretic therapy for hypercalcemia, it continued in the follow-up period and zoledronic acid 4 mg IV infusion was given. We started anti-tuberculosis therapy (isoniazid 300 1x1, 1x1 Rifampicin 300 1x1, pyrazinamide 500 1x2.5, ethambutol 500 1x2). In the 4 months of treatment, the patient's laboratory parameters were found to be stable and she was clinically asymptomatic.

Figure .1: Millimetric nodules showing random distribution in parenchyma (left) and apical lower lobe infiltrates (right)

Figure. 2: Necrotizing granuloma (asterisk), Langhans type giant cell around the granuloma (arrow)

Discussion:

While the most common cause of hypercalcemia in hospitalized patients is malignancy, the most common cause is PHPT in patients admitted to the outpatient clinic (1). In the presence of hypercalcemia neuromuscular symptoms such as changes in consciousness, muscle weakness and fatigue may occur as well as gastrointestinal and cardiac symptoms. Tuberculosis is one of the rare reasons of hypercalcemia (2). Incidence of the hypercalcemia due to tuberculosis varies depending on regional sunlight exposure, vitamin D and calcium intake (3). Calcium levels rarely increase in these group of patients and is generally asymptomatic. 1, 25 OH vitamin D production mediated by activated T lymphocytes and alveolar macrophages in patients with tuberculosis are held responsible for hypercalcemia (4).

Conclusion:

As the patient admitted to our clinic with syncope, this supports atypical presentations can be seen more frequently in the elderly patients. After the exclusion of common causes such as malignancy and PHPT, granulomatous diseases should be kept in mind in the differential diagnosis of hypercalcemia.

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