

Primary Tubercular Granulomatous Thyroiditis, Presenting as Thyroid Nodule with Hyperthyroidism, Pyrexia of Unknown Origin and Severe Anemia

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Abstract

Tuberculosis of Thyroid gland is a rare entity even in countries with high prevalence of tuberculosis. The patients present with a broad spectrum of manifestations ranging from an isolated painful nodule to frank hyperthyroidism. We report an interesting case of primary tubercular granulomatous thyroiditis, presenting as a thyroid nodule with pyrexia of unknown origin, hyperthyroidism and severe anaemia which responded to anti tubercular and anti-inflammatory treatment with complete recovery, as evidenced by blood reports and CT scan reports. Thus, proper diagnosis may avoid unnecessary surgical interventions, that were a trend in the past.

Introduction

Primary tuberculosis of thyroid gland is a very rare entity, exact prevalence being about 0.1% to 1.15% of the tubercular patients.^{1,2} The diagnosis was previously made from examination of specimens after surgical resection⁴ but now fine needle aspiration cytology (FNAC)³ and serology gives an excellent result. Pre-operative diagnosis of thyroid tuberculosis is important because of availability of medical treatment and limited role of surgery. This condition should be kept in mind while evaluating patients with thyroid nodule, especially in communities where prevalence of tuberculosis is high. Mis - diagnosis is common. Analysis of mis-diagnosis of four cases

of thyroid tuberculosis after surgery for “adenoma” in two cases, “thyroid carcinoma” in one case and “missed diagnosis” in fourth case suggests that it is important to re-inforce the knowledge of this disease.⁴

Although tuberculosis has been reported in many parts of human body, thyroid involvement is rare, probably because of bactericidal action of colloid material of thyroid gland, iodine stores, high vascularity, increased phagocytic activity in hyperthyroidism and possibly anti-tubercular action of thyroid hormones.^{5,6}

Case Report

A 41 year old Kashmiri women presented with a history of evening rise of temperature and moderately severe pain in the neck for about 45 days. She had till been treated by various physicians as a case of Acute

Pharyngitis, Urinary tract infection and Enteric fever.

On examination, she was remarkably pale with weight = 55 kg, pulse rate 100/min and B.P 120/80 mmHg in right upper arm. A right sided thyroid nodule was noted on general physical examination. Systemic examination including respiratory, cardiovascular and abdominal examination were unremarkable.

Investigations done revealed Hb=7.5gm%, TLC= 6,400/cumm, platelet count = 1.54, ESR= 154mm in first hour. Blood sugar level, kidney function tests, chest X-ray were within normal limits. Ultrasonography of abdomen revealed incidental bilateral nephrolithiasis, but no lymphadenopathy or any organomegaly. Mantoux test was negative after 48 hours. Thyroid functions test revealed FT3=6.69 pg/ml (normal 2.8 – 6.0) , FT4 = 2.18 ng/ml (normal 0.8- 1.9) and TSH= 0.01μ IU/ml (normal 0.03 – 5.0). CT scan of neck (Figure 1) revealed a nodular, asymmetric enlargement of right lobe of thyroid gland (46×24×26 mm). There was no cervical or mediastinal lymphadenopathy. FNAC (fine needle aspiration cytology) smears showed scattered or small clusters of mild pleomorphic follicular cells, epithelioid cell clusters, occasional giant cells and lymphoid cells at various stages of development. Though acid fast bacilli was not seen, cytomorphology was consistent with chronic granulomatous thyroiditis possibly tubercular. Blood was sent for TB Feron detection and Gamma Interferon was detected.

Patient was put on full anti tubercular treatment consisting of Isoniazid 300mg, ethambutol 1000 mg, rifampin 600mg and pyranzinamide 1500 mg for initial 4 months along with analgesics/ anti-inflammatory

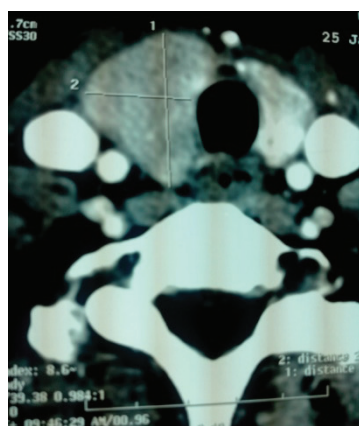


Fig. 1: Nodular, asymmetric enlargement of right lobe of thyroid gland

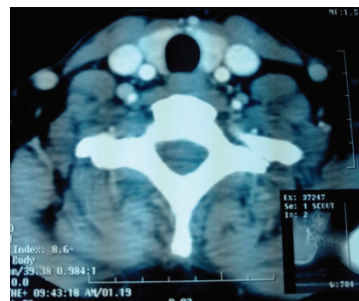


Fig. 2: CT scan done on same date showed normal thyroid structure on both sides

drugs as needed and iron, vitamins and other supplements. After 4 months anti tubercular treatment was reduced to isoniazid and rifampin for further 2 months. Patient became symptomless within 2 months of initial treatment and thyroid nodule started regressing in size with total clinical disappearance within 4 months. Thyroid hormone levels came down to normal limits after 3 months with T3= 1.94 (normal 1.3-3.1 nmol/litre), T4= 6.2 (normal 5.13-14.0 µg/dl), TSH= 2.57 (normal 0.35-5.5 µIU/ml). Haemogram also improved with Hb= 11.3gm% and ESR= 29 mm in first hour. After completion of six months, Hemogram revealed Hb=11.9gm% ESR=14mm in first hour and normal TLC and platelet count. CT Scan of neck done on same date revealed a normal study of thyroid structure on both sides (Figure 2).

Thus a primary nodular tubercular thyroiditis of right lobe of thyroid gland responded completely to a course of anti tubercular treatment, which could have been subjected to surgical intervention if tuberculosis of thyroid gland was not suspected.

Discussion

Pathologically tuberculosis of thyroid gland is divided into four types-

1. Cold abscess or cheese type (mis-diagnosed as adenoma).
2. Diffuse type (mis-diagnosed as diffuse goiter).
3. Granuloma type (mis-diagnosed as thyroid cancer).
4. Military type with multiple lesions (as in military tuberculosis)⁷.

Since demonstration of acid fast bacilli is not always possible, the diagnosis is made by histopathological examination showing caseous necrosis or epithelioid granulomas. FNAC (fine needle aspiration cytology) is equally diagnostic². When it is cheesy or pus like, aspirated material should be cultured for mycobacteria⁸.

Thyroid tuberculosis may present clinically with a broad spectrum of manifestations, ranging from an isolated painful nodule to frank hyperthyroidism. Patient may also present with thyroiditis, thyroid abscess or pyrexia of unknown origin or even mimic thyroid malignancy with dysphagia, dysphonia or laryngeal nerve palsy.⁹

Consistent with the literature, our patient had no extrathyroid involvement but was having hyperthyroidism, though thyroid dysfunction is rare in thyroid tuberculosis.⁸ Striking anaemia seen in this patient, is also

not a common feature in this disease. However there was complete recovery of nodule as well as anaemia.

Treatment of thyroid tuberculosis is effective anti-tubercular medicines involving HRZE (isoniazid+ rifampin+ethambutol+pyranzinamide) plus surgical drainage of pus, if needed. Anti tubercular treatment is effective because of high vascularity of the gland.¹⁰

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