Asymptomatic Thyroid Tuberculosis in a Multinodular Goitre Patient: a Case Report

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Abstract. Thyroid tuberculosis is a very rare disease of the thyroid gland. In recent years, the incidence of extra-pulmonary tuberculosis has been showing a progressive increase. We present the case of a 41-year old female patient, operated for an asymptomatic multinodular goiter, and who had a histopathological diagnosis of thyroid tuberculosis. Thyroid tuberculosis should be kept in mind in the differential diagnosis of thyroid nodules, even in patients with no history and no symptom of tuberculosis disease.

Introduction

Extra-pulmonary tuberculosis may have different clinical manifestations and may be difficult to diagnose compared to pulmonary tuberculosis. Caseous foci frequently occur in lymph nodes, bone and prostate gland, but remain undetected in the absence of clinical symptoms (1). Infectious diseases of the thyroid gland are uncommon because of the comparatively high resistance of the gland to infection (2). Isolated tuberculosis involving the thyroid gland alone is extremely rare (3); the incidence of thyroid tuberculosis is only 0.1-0.4 percent (4).

Pathological forms include multiple thyroidal granulomata, goiter with caseation, cold abscess, chronic fibrosing thyroiditis and less commonly acute abscess (5).

We present a case with thyroid tuberculosis in the light of literature.

Case report

A 41-year old woman presented with a midline neck swelling for a period of 2 months. There was no tuberculosis history in the patient’s anamnesis, family and relatives.

On physical examination, the pulse rate was 80/min, fever 36.8°C and blood pressure 120/80 mmHg. There was approximately a 2 × 2 cm non-tender nodule in the right lobe and 1 × 1 cm non-tender nodule in the left lobe of the thyroid gland. There was no palpable lymphadenopathy. There were no symptoms suggestive of hyper- or hypothyroidism. The cardiopulmonary and abdominal examination was normal. Indirect laryngoscopy showed both vocal cords to be freely mobile. On laboratory testing, the white blood cell count was 7300/mm, haemoglobin 11.5 g/dl, platelet count 488000/mm³ and erythrocyte sedimentation rate was 20 mm/h. Other laboratory investigations, including blood chemistry and urine analysis, were within normal ranges. The thyroid function tests were as follows: free T3 5.41 pmol/L; free T4 12.68 pmol/L; sensitive thyroid-stimulating hormone (TSH) 0.63 mIU/mL. The enzyme linked immunosorbent assay test for HIV was negative. The chest X-ray was normal. Thorax tomography, bone scintigraphy, kidney ultrasonography, 24 hours controlled urine culture, direct ARB in acid resistant bouillon culture and gastric lavage culture were made in order to rule out other tuberculous focus in the patient, and no other tuberculous focus was found. Thyroid ultrasonography revealed a 20 × 23 mm hypoechoic, heterogeneous and round cystic nodule within the right lobe and 13 × 10 mm isoechoic round cystic nodule within the left lobe of thyroid gland. Bilateral subtotal thyroidectomy was performed.

Sections from the left lobe showed nodular histology of hyperplasia, whereas sections from the thyroid right lobe showed not only areas of nodular hyperplasia but also granulomatosis necrosis including sporadic caseification necrosis in a focal point. These granulomas consisted of epitheloid histiocytes, lymphocytes, fibroblasts and Langhans cells. This image was consistent with tuberculosis though she had no history of tuberculosis and did not have any known contact with tuberculosis.

Isoniazid 300 mg/day, rifampicin 600 mg/day, pyrazinamide 1500 mg/day and ethambutol 1500 mg/day were administered for two months and isoniazid 300 mg/day, rifampicin 600 mg/day were subsequently administered for four months. After treatment, the patient was followed up for sixteen months without encountering any sign or symptom of recurrent disease.
Sectional image of granulomatosis inflammation including caseification necrosis besides thyroid tissue (H&E, ×100).

Discussion

Thyroid gland tuberculosis is a rare condition. Thyroid tuberculosis is reported at a rate of 0.1%-0.003% in postmortem studies (6). BRUNS reported the first case of tuberculosis thyroiditis in 1893 (5). Since then, there have been relatively few cases of tuberculous involvement of the thyroid gland reported, and almost all cases have been associated with tubercular foci elsewhere in the body. Isolated tuberculosis of the thyroid gland is a rare form of presentation of the disease (7).

Infectious diseases of the thyroid gland are uncommon due to the fact that the resistance of the thyroid gland to infections is attributed to many factors such as well developed capsule, high iodine content and rich lymphatics as well as the vascular supply (2).

Diabetes Mellitus, malnutrition, old age and AIDS can play a part in the occurrence of thyroid tuberculosis (8). Extra-pulmonary tuberculosis is increasing in frequency in patients with HIV – induced immunosuppression. Patients with tuberculosis and AIDS have high rates of extra-pulmonary disease, ranging from 45 to 75 percent (3).

Normal thyroid function is the most frequent laboratory finding. Thyroid function abnormalities may be said to be extremely rare in association with tuberculosis of the thyroid (9). In our patient the thyroid function was normal as well.

There are at least five pathological varieties of tuberculosis of the thyroid gland, and these are: (1) multiple lesions throughout the gland in association with military tuberculosis, (2) a goiter with caseation, (3) cold abscess formation sometimes presenting on the surface of the gland, (4) chronic fibrosing tuberculosis (which is difficult to distinguish from De Quervain’s thyroiditis) and acute abscess formation. Of these, a tuberculous abscess is the least common form of presentation (5).

The exact reason for the rarity of thyroid tuberculosis is unknown. Hypotheses in the literature include: (i) thyroidal colloidal material possessing bactericidal action, (ii) extremely high thyroidal blood flow and excess of iodine, (iii) enhanced destruction of tubercle bacilli by increased physiological activity of phagocytes in hyperthyroidism and (iii) oxygenation of the thyroid tissue (8). It has been considered that the thyroid gland must become invaded as a result of tuberculous bacteremia rather than retrograde flow of lymphatics into the thyroid gland. However, it is exactly unknown why the morbidity of the thyroid by the bacillus of tuberculosis is so low (10). In our patient, there was primary involvement of the thyroid gland by tuberculosis without an overt focus of tuberculosis seen elsewhere in the body.

Clinical diagnosis of thyroid tuberculosis is very difficult. Thyroid tuberculosis may present a broad spectrum of manifestations. As seen in our patient, thyroid tuberculosis may be asymptomatic. In our opinion, this may be due to the fact that our patient was young and that no diseases which may affect the immune system like particularly HIV or other systemic diseases did exist. Since we did not predict thyroid tuberculosis in our preoperative case, we put the diagnosis on histopathological basis.

In conclusion, the primary focus of tuberculosis presence of cervical lymphadenopathy, high erythrocyte-sedimentation-rate values and a past history of tuberculosis might help with diagnosis, but thyroid tuberculosis may occur in the absence of these features. Tuberculosis of the thyroid gland should be kept in mind in patients with non-tender cold nodules.

References


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