Brief Communication

A case of simultaneous occurrence of Graves' disease and Hashimoto's thyroiditis

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ABSTRACT

Introduction: Simultaneous occurrence of Hashimoto's thyroiditis (HT), and Graves' disease (GD) is rare. **Aims and Objectives:** We report a case of simultaneous occurrence of GD and HD, at presentation. **Case Report:** A 60-year-old lady presented with tremulousness of hands, palpitation, and excessive sweating. She had a history of weight loss and neck-swelling. Her weight was 46 kg, BMI 17, afebrile, regular pulse rate of 110/min with fine tremor in hands. Thyroid gland was symmetrically enlarged, firm, without any bruit, but mildly tender with lobular surface. There were no occular manifestations. Initial thyroid function tests (TFT) revealed: T3: 3.80 ng/ml (0.80-2.10), T4: 12.40 ug/dl (5.10-12), thyroid stimulating hormone (TSH): 0.20 µU/L (0.70–5). Her anti thyroperoxidase (TPO) antibody: 374 IU/ml (normal [nl.] <35) and TSH receptor antibody: 15 U/L (nl. <1) were both strongly positive. Ultrasonogram of thyroid revealed a hypoechoic enlarged gland. 99mTc pertechnetate scan showed an enlarged gland with increased uptake of radiocontrast: 17% (nl. 0.4-4%) with some patchy defects in both lower poles. Thyroid fine needle aspiration cytology (FNAC) showed sheets of Hurthle cells with abdunce of lymphocytes indicating HT. She was observed on beta blockers. Repeat TFT, 3 months later showed: T3: 4.20 ng/ml, T4: 14.40 ug/dl, TSH: 0.001 µU/L, with increased uptake on repeat scan. **Conclusion:** HT rarely occurs following GD. Our case of an elderly lady with no eye signs, lobular, firm tender goiter with patchy uptake in both lower poles on Tc99m scan were odd points in diagnosing isolated GD. FNAC confirmed simultaneous HD with GD.

Key Words: Graves' disease, Hashimoto's thyroiditis, simultaneous

Introduction

Hashimoto's thyroiditis (HT) and Graves' disease (GD) are the two main types of autoimmune thyroid disease. HT rarely occurs following GD. But combined occurrence of GD and HT are rare. We report a case of simultaneous occurrence of GD and HT, at presentation.

Case Report

A 60-year-old lady, presented with tremulousness of hands for 1 month and palpitation with excessive sweating for 3 months. She had a history of weight loss for 6 months, and gradually enlarging neck swelling for 3 years. She was nervous, irritable, and emotionally labile. Her weight was 46 kg, BMI 17, afebrile, pulse rate: 110/min/regular, blood pressure: 140/56 mm of Hg. Her thyroid gland was symmetrically enlarged, firm in consistency, nodular/lobular surface and mildly tender without any bruit. Palms were warm and sweaty with fine tremor in hands. All reflexes were exaggerated. There were no occular manifestations.

Initial thyroid function tests (TFT) revealed high T3: 3.80 ng/ml (0.80-2.10), T4: 12.40 ug/dl (5.10-12) with a low TSH: 0.20 mU/L (0.70-5). Her TSH receptor antibody (TSAb): 25 U/L (Normal [nl.] <1), anti TPO antibody: 374 IU/ml (nl. <35) and thyroglobulin antibody: 268 IU/ml (nl. <65) were all strongly positive. Complete hemogram and ESR-18 mm/h (nl.-1-25) were normal. Ultrasonogram of thyroid revealed an enlarged gland with hypoechoic parenchyma with fibrous septa. ^{99m}Tc pertechnetate scan (Tc99m scan) revealed enlarged thyroid

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gland with diffusely increased uptake of radiocontrast: 17% (nl. 0.4-4%) with some patchy defects in both lower poles. Thyroid FNAC showed sheets of Hurthle cells with abdunce of lymphocytes and sheets of regular follicular cells, indicating HT. She was observed on beta blockers. Repeat TFT, 3 months later showed: T3: 4.20 ng/ml, T4: 14.40 ug/dl, TSH: 0.001 μU. Repeat ^{99m}Tc pertechnetate scan was done, which again revealed enlarged thyroid gland with diffusely increased uptake of radiocontrast: 21% (nl. 0.4-4%) with same patchy defects in both lower poles. Antithyroid medication carbimazole was started.

Discussion

HT rarely occurs following GD. Our patient presented with clinical symptoms and signs of thyrotoxicosis along with slightly raised T4, T3 and low TSH with positive TSAb, and diffusely increased uptake on Tc99m scan, all in favor of a diagnosis of GD. However, there were some odd features such as elderly age, absence of eye signs, nodular, firm mildly tender goiter, hypoechoic parenchyma in ultrasonography (USG), and patchy uptake in both lower poles on Tc99m scan. FNAC of the thyroid gland was suggestive of ut FNAC of thyroid gland suggested HT. So we adopted a wait and watch policy, and put the patient on beta blockers. Repeat TFT, 3 months later showed persistent thyrotoxicosis and repeat Tc99m scans revealed an enlarged thyroid gland with diffusely increased uptake of radiocontrast. Diffusely increased uptake on Tc99m scan rules out Hashitoxicosis, which mostly resolves within 3-months time-frame. A diagnosis of combined occurrence of hyperthyroid GD and HD was established and anti-thyroid medication started. She responded to anti-thyroid drug carbimazole.

Sutradhar *et al.*[1] reported a case of combined occurrence of hyperthyroid GD and HD. Umar *et al.*[2] reported four cases of HT in previously diagnosed patients with GD hyperthyroidism. In three cases, HT occured 7-25 years after GD treatment; in one, it developed in a few months of GD treatment. The diagnosis of HT was based on clinical manifestation, positive TPO and thyroglobulin antibody, and FNAC. Ohye *et al.*[3] reported four cases of GD after painful HT. All were middle-aged women, who had high titers of anti-thyroid antibodies and thyrotoxicosis at the onset of painful HT. After 2-7 years, they developed GD with positive thyrotropin receptor antibody. They were successfully treated with antithyroid drug or radioactive iodine. Takasu *et al.*[4] reported seven patients with hypothyroidism due to Hashimoto's disease, who developed hyperthyroid GD. Some had transient, some persisistent hyperthyroidism due to GD following hypothyroidism due to HT.

HT and GD represent the main two types of autoimmune thyroid disease. GD is caused due to hyperplasia and hyperfunction of the thyroid gland by stimulating TSAb. On the contrary, HT is thought to be due to a TSH stimulation-blocking antibody (TSBAb) which blocks the action of TSH hormone and subsequently brings damage and atrophy to the thyroid gland.[2] Alterations in the thyroid state related to the balance between the activities of TSAb and TSBAb.[4] Moreover, approximately 15-20% of patients with GD have been reported to have spontaneous hypothyroidism resulting from the chronic HT.[2] Patients with GD who have achieved remission on anti-thyroid drugs may also experience HT or Hashitoxicosis or intermittent transient hyperthyrodism. They may have both HT and GD since antibodies associated with both diseases may be present. To distinguish from relapsed GD a wait and see approach is beneficial. A probable diagnosis can also be made by thyroid antibody tests. A definitive diagnosis can be made by FNAC biopsy, that is, fine-needle aspiration. Pathogenesis for chronic HT following anti-thyroid drug in GD remains unclear. Hashimoto's disease, which occurs following GD may be due to extended immune response to endogenous thyroid antigens, i.e. thyroid peroxidase and thyroglobulin, which may enhance lymphocyte infiltration and finally cause HT.[2]

In conclusion, HT rarely occurs following GD. In our case of an elderly lady, absence of eye signs, lobular-firm tender goiter with patchy uptake in both lower poles on Tc99m scan were odd points in diagnosing isolated GD. FNAC confirmed simultaneous occurrence of HT with GD at presentation.

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