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"Fine Needle Aspiration Cytology: A Key To Early Diagnosis Of Hashitoxicosis"

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Abstract:
The Graves 'disease and Hashimoto's thyroiditis both are organ-specific autoimmune diseases which are rarely seen in combination and reported. Graves 'disease shows features of hyperthyroidism while Hashimoto's thyroiditis causes hypothyroidism. When features of Hashimotos thyroiditis is seen along with thyrotoxicosis the term 'hashitoxicosis 'is used to describe this autoimmune thyroid disease where there is an overlap syndrome between these two clinical entities. Here a 25-year-old female patient turned up with this rare combination who presented with a diffuse thyroid swelling. It was diagnosed by cytological findings initially complemented by lab findings and clinical history.

Keywords: *Hashitoxicosis, Grave's disease, Hashimoto's thyroiditis, auto-immune*

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I. Introduction:

Graves 'disease and Hashimoto's thyroiditis are the two most common organ-specific autoimmune diseases with circulating auto antibodies [1] These antibodies abnormally stimulate the thyroid stimulating hormone (TSH) receptors in Graves 'disease leading to hyperfunctioning of the thyroid gland producing hyperthyroidism while in Hashimoto's thyroiditis the anti-thyroperoxidase (TPO) antibodies, the most specific antibody which causes progressive destruction of thyroid cells causing hypothyroidism [2] The combination of these two clinical entity is rarely seen and reported. The term 'hashitoxicosis' is sometimes used to describe an autoimmune thyroid disease where there is an overlap syndrome of Graves 'and Hashimoto's disease.

Here a 25-year-old female patient turned up with this rare combination who presented with a diffuse thyroid swelling. It was diagnosed by cytological findings initially complemented by lab findings and clinical history.

II. Observation:

A 25-year-old female complained of swelling in the midline of the neck for 2- 3 years (Fig-1).

The swelling had gradually increased in size and was occasionally painful. There was slight hoarseness in the voice with occasional dysphasia with a clinical suspicion of Graves' disease. A very faint degree of fine tremor was also seen, and mild thyrotoxicosis was observed. The patient was prescribed a tablet carbimazole and a beta blocker as well for her palpitations.

Total triiodothyronine(T3) (3.29 ng/dl) and Serum free triiodothyronine (FT3)-13.54pg/ml was increased, total thyroxine (T4) (18.67ug/dl) and Serum free thyroxine (FT4)-4.38ng/dl was increased while thyroid stimulating hormone was decreased (0.06uIU/ml) raising the suspicion of Graves' disease.

The patient was send for FNAC were we aspirated scant blood mixed colloid, smears were made and stained with LG and PAP. On microscopy we evaluated a cellular smears showing benign thyroid follicular cells arranged in sheets and macrofollicles against a background containing thin hemorrhagic colloid and pleomorphic lymphoid cells. Few thyroid follicles show lymphocytic impingement (Fig-2,3). Hurthle cell changes, few epithelioid cells, histiocytes and prominent fire flares were seen in few clusters (Fig-4). And hence it was reported as Hashitoxicosis - Chronic Lymphocytic Thyroiditis with features of Toxic Goiter (The Bethesda System of Reporting Thyroid Cytopathology - Category II). The sample was sent for anti-thyroid antibodies and the Anti thyroid peroxidase estimation (1297.24 U/ml) which was found highly increased, raising the suspicion of Hashimoto's thyroiditis even though free T4 was increased and TSH was decreased.

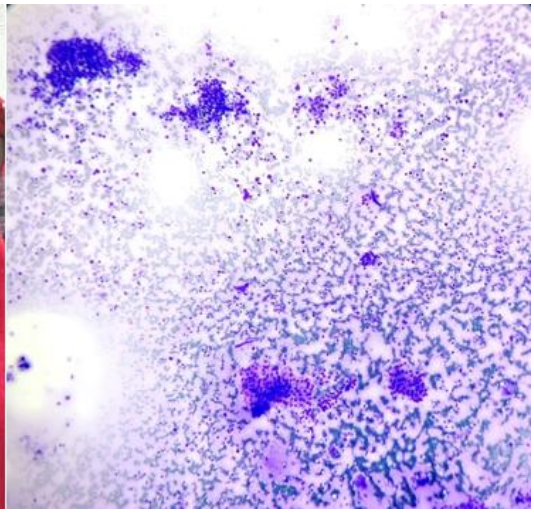
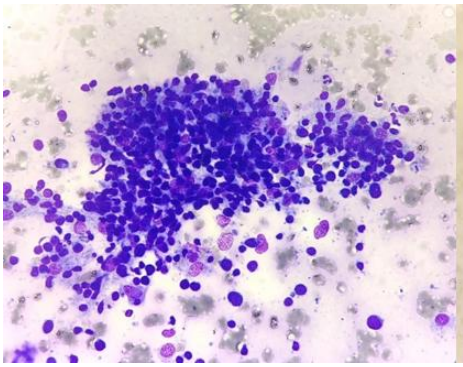


Fig 1: A 25-year-old female with midline neck swelling

Fig 2: Clusters of benign thyroid follicle cells showing lymphocytic impingement; MGG stain ; 4x



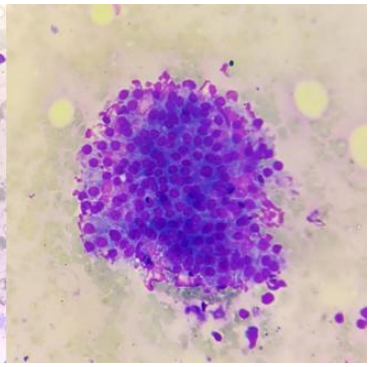


Fig 3: Cluster of benign thyroid follicle cells showing lymphocytic impingement and hurthle cell change; MGG stain; 40x

Fig 4: Cluster of benign thyroid follicle cells showing lymphocytic impingement and fire flares; MGG stain; 40x

III. Discussion:

Grave's disease, toxic multinodular goiter, and solitary toxic nodules are considered amongst the commonest etiologies in a patient with thyrotoxicosis while Hashimoto's thyroiditis is the most commonly causes hypothyroidism, but it can also have a variable clinical presentation [3]

Both Graves' disease and Hashimoto's thyroiditis are more frequently seen in females than in males, with a sex ratio varying from 1/5 to 1/10. As said earlier while Graves' disease generally shows features of thyrotoxicosis, Hashimoto's thyroiditis has a variable clinical presentation such that it may begin with a phase of hyperthyroidism called Hashitoxicosis caused due to destructive inflammatory conditions which damage the thyroid gland and cause the "leakage" of hormones into the blood resulting in transient thyrotoxicosis followed by a phase of euthyroidism before the onset of permanent hypothyroidism .[1,2] A few case reports have shown the sequential occurrence of either Hashimoto's thyroiditis followed by treated Graves 'disease or vice versa [4,5,6] This could suggest the possibility of co-existence of these two pathologies.

Multiple genetic and environmental factors both play a role in ultimately causing damage to thyroid cells d by the production of autoantigens responsible for an immune reaction. An imbalance in the TH1/TH2 balance leads to activation of cellular immunity which in turn causes destruction of the thyroid cells in Hashimoto's thyroiditis. While a predominance of TH2 activates humoral immunity which produce antibodies to the TSH receptor causing Graves 'disease. [1]

In 90% of the patients with Hashimoto's thyroiditis, there is presence of Anti-TPO antibodies, but there have been a few cases of Graves' disease which also shows TPO antibody positivity determining that the immune reaction in Graves 'disease that leads to the production of anti-THS receptor antibodies might have also lead to the production of anti-TPO and anti-thyroglobulin antibodies. [7]

In Hashitoxicosis multiple genetic and environmental factors as mentioned earlier are known to be involved. Genetic factors mainly include human leucocyte antigen (HLA), major histocompatibility

complex(MHC), and cytotoxic T lymphocyte association (CTLA). While Environmental factors can be infections, cytokine therapy, selenium, iodine uptake, and smoking [5].

Other autoimmune diseases such as type 1 diabetes mellitus, systemic lupus erythematosus, multiple sclerosis, rheumatoid arthritis, celiac disease, vitiligo, and chronic urticaria are also found to be associated [6]. But, our patient did not have any other autoimmune disease. Certain drugs like pegylated interferons α2b (PEG-IFNα) and ribavirin can produce hashitoxicosis followed by type 1 diabetes [8]

While performing fine-needle aspiration biopsy (FNAB), MacDonald and Yezdi emphasized that there is a need for adequate sampling of the thyroid by comparing it with their surgically excised specimen of the same and concluded that there's a significant chance of missing Hashimoto's thyroiditis in diagnosis. in smears that show cytological evidence of hyperplasia or abundant colloid. [9]

According to a study conducted by Nabhan et.al out of 69 patients with autoimmune thyroiditis only eight (11.69%) had hashitoxicosis It is observed in 4.47%. (10). The normal course of this condition is remission in due course of time or it may develop into hypothyroidism at a later stage. The investigation of choice for hashitoxicosis is the estimation of auto-antibodies, which includes antithyroglobulin and antimicrosomal antibodies. The most sensitive of these is the antimicrosomal antibody. [3,8].

Patients with hashitoxicosis are first managed with β blockers and thyroxine supplementation. However, in case of an unresponsive mass despite of medical management or in case of suspicion of malignancy on a cutting needle biopsy the patient might have to undergo surgical resection. [9,11]

IV. Conclusion:

Hashitoxicosis refers to a transient state of hyperthyroidism seen in HT, representing the aggravated destruction of the follicles. In our case, Hashitoxicosis was diagnosed cytologically and confirmed with biochemical investigations. These anti-thyroid antibodies were found at high levels and clinically presented with hypothyroidism. This association between Graves 'disease and Hashimoto's is not accidental. It can be explained by a common pathogenesis on certain points that are confirmed by various studies and literature. In patients presenting with toxic nodular goiter, possibility of Hashimoto's thyroiditis should be considered and excluded by obtaining representative samples and estimation of thyroid antibodies should form integral part of any investigative protocol for thyroid disorders.

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