



A RARE CASE OF PROLONGED HASHITOXICOSIS INDUCED CARDIOMYOPATHY

General Medicine

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ABSTRACT

Hashitoxicosis is the initial hyperthyroid phase of the patients with Hashimoto's thyroiditis and usually this phase lasts for one to two months. We had a rare case of Hashimoto's thyroiditis with prolonged phase of Hashitoxicosis and Hashitoxicosis induced cardiomyopathy. A 27 year old male presented with 6 months history of heat intolerance, sweating, tremors, weight loss and 3 week history of shortness of breath, palpitations and swelling of lower limbs. On examination, patient had exophthalmos, bilateral pitting pedal edema, goitre, tremors, tachycardia, tachypnea, raised JVP, S3, pansystolic murmurs in mitral and tricuspid areas. Thyroid profile showed elevated T3, T4 and suppressed TSH. Anti-TPO antibodies were elevated. USG neck and FNAC were suggestive of Hashimoto's thyroiditis. 2D Echo showed dilated cardiomyopathy. Patient was started on anti thyroid drugs, diuretics and advised for follow up.

KEYWORDS

Hashimoto's thyroiditis, cardiomyopathy

INTRODUCTION:

Hashimoto's thyroiditis is a common disorder characterised by autoimmune destruction of thyroid gland. It is more common in women with female to male ratio of about 7:1. Both genetic and environmental factors influence the development of this disease. Anti-Thyroid Peroxidase antibodies play a key role in inducing the immune response resulting in infiltration of lymphocytes in thyroid cells and destruction of the cells. Clinical spectrum of Hashimoto's thyroiditis can range from 1) Hashitoxicosis, when the preformed and stored thyroid hormones are release from the destroyed thyroid follicles into the circulation; 2) Euthyroidism, when the preserved thyroid tissue compensates for destruction. 3) Hypothyroidism, when there is insufficient production of thyroid hormones from the gland.¹

It is a well known fact that excess thyroid hormones have a significant effect on structure and function of heart. It can alter the cardiac haemodynamics and causes high output cardiac failure and dilated cardiomyopathy. Thyrotoxic cardiomyopathy occurs due to toxic effects of thyroid hormones on the heart. There is an alteration in myocyte energy production, intracellular metabolism, contractility of myofibril. There can be left ventricular hypertrophy, atrial fibrillation, dilatation of chambers of heart, diastolic dysfunction, heart failure and pulmonary hypertension.² It is essential to differentiate Hashitoxicosis from other causes of primary hyperthyroidism like Grave's disease as they differ in their management. Hashitoxicosis usually has a shorter course and lasts for only a few months.³

Case Report:

A 27 year old male patient presented with 6 months history of heat intolerance, sweating, tremors, loss of weight inspite of good appetite, hair loss and with 3 week history of breathlessness, palpitations and swelling of both lower limbs. He noticed swelling in the front of neck 6 months back which gradually increased in size. There was no previous history of diabetes mellitus, hypertension and other cardio respiratory diseases. He was not an alcoholic/smoker. There was no significant family history.

On examination, the patient had bilateral pitting pedal edema, was afebrile, pulse rate was 138 beats per minute, respiratory rate was 28 per minute, blood pressure was 120/80 mm Hg, JVP was elevated. Goitre was present. He had a staring look with exophthalmos. Hands were hot and moist. Tremors were present. Cardiovascular system examination -patient had S3, pansystolic murmurs in mitral and tricuspid areas.

Laboratory investigations-CBC, RFT, LFT, RBS, lipid profile were normal. Total T3-784 ng/dl, Total T4-27.3 µg/dl, TSH-0.02 µIU/L, anti-TPO-258.2 U/ml, negative for Thyroid stimulating antibodies. USG showed features suggestive of thyroiditis. FNAC showed Hurthle cell change suggestive of Hashimoto's thyroiditis. ECG showed sinus tachycardia. 2D ECHO showed four chamber dilatation

and depressed left ventricular function with an ejection fraction-39%. He was started on diuretics, carbimazole, propranolol and advised for follow up.



Figure 1 : patient with Hashitoxicosis showing exophthalmos

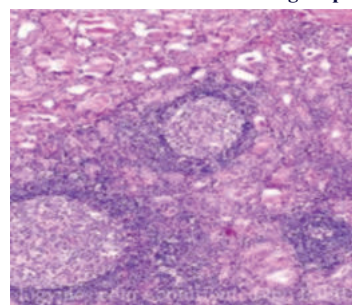


Figure 2 : Histological section of thyroid gland of the patient showing Hurthle cell change

DISCUSSION:

Hashimoto's thyroiditis is the most common cause of hypothyroidism. It is an autoimmune disorder. It is characterized by the presence of anti-TPO antibodies which are present in about 90% of the patients. Anti-thyroglobulin antibodies are seen in 60% of the patients. Hashimoto's thyroiditis usually presents as hypothyroid or euthyroid state. Hashitoxicosis is the initial hyperthyroid state which occurs due to the release of stored thyroid hormones from the destroyed gland. This phase usually lasts for only a few weeks to months.⁴

In our patient, the presentation is consistent with Hashitoxicosis. He had features of hyperthyroidism and elevated levels of anti-TPO antibodies. Due to elevated thyroid hormone levels for a long duration, he developed dilated cardiomyopathy. He had no other comorbidities and no other risk factors for the development of heart failure.

Clinical features of Hashitoxicosis are very similar to those observed in Grave's disease. Hence it poses a diagnostic challenge.

Hashitoxicosis is the second most common cause of thyrotoxicosis in childhood. Over a period of time, they tend to become euthyroid or hypothyroid.⁵ The effects of thyroid hormones on heart are mediated mainly by triiodothyronine(T3). It has positive inotropic and chronotropic effect on heart. It causes cardiac hypertrophy and diastolic dysfunction. Patients present with chest pain, exercise intolerance, systolic hypertension, systolic murmurs, atrial fibrillation and heart failure.⁶ Anti thyroid drugs and beta blockers are the main stay of treatment in these patients. Digoxin and anti coagulants are used if associated atrial fibrillation is present.⁷ Nabhan et al. Found that hashitoxicosis was the initial presentation in 11.5% of the patients with Hashimoto's thyroiditis enrolled in their study. The hyperthyroid phase lasted for 1-5 months.⁸ Wasniewska et al. observed that the resolution of hyperthyroidism occurred 8.3 ± 6.3 months after the diagnosis.⁹ This case represents an unusually prolonged case of Hashitoxicosis with features of dilated cardiomyopathy secondary to excess thyroid hormones.

CONCLUSION:

Prolonged phase of Hashitoxicosis can therefore present with hyperthyroid features including cardiomyopathy. They require regular long term follow up to avoid prolonged treatment with anti thyroid drugs/radioactive iodine/surgery to prevent iatrogenic overt hypothyroidism.

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