CLINICAL VIGNETTE

Atypical Presentation of Hyperthyroidism

Anita Srinivasa, MD

Introduction

Hyperthyroidism is a clinical condition caused by an overproduction of thyroid hormone.

The overall prevalence is about 1.3% in the general population but increases to about 4-5% in older women.it is also more common in smokers.[1]The etiology is usually Graves' disease in younger women, while toxic nodular goiter is more common in older women. Hyperthyroidism is usually evident in patients with unequivocal clinical and biochemical manifestations of the disease, however some patients have fewer but less obvious clinical signs and symptoms.[1]

Case

A 55-year-old generally healthy female with no significant past medical history presented to the primary care with chief complaint of fever. She reports fever about one week prior to her visit. Home temperature ranged from 100- 100.5°F. She was taking round the clock antipyretics, alternating between acetaminophen 325 mg and ibuprofen 400mg to control the fever.

Review of systems was positive for chronic anxiety which recently worsened due to stress.

She denied night sweats, weight loss, tremors. Rash or palpitations. Vital signs were HR 85, TEMP 99.0°F, BP 110/70. The etiology was presumed to be infectious with testing including chest x-ray, CBC. CMP, UA. All tested were normal and she was referred to Infectious Disease.

Her only symptom was the ongoing fevers and additional labs were obtained including ESR, ANA, MONO ANTIGEN, blood cultures, cocci EIA, MTB. RPR, and TSH.

All labs resulted normal except TSH <0.2. Subsequent thyroid testing included TSH < 0. 2(0.3-4.7 mcIU/ML), FT4 3.8 (0.8-1.7 ng/DL), FT3 1,120(222-383 pg. /DL), TSI < 0.10 (<0.54IU/L). She was referred to endocrinology and bedside thyroid ultrasound demonstrated autoimmune changes in the thyroid gland.

She was diagnosed with hyperthyroidism due to hashitoxicosis, described as an overlap syndrome of Graves 'and Hashimoto's Thyroiditis.

Her TSI was normal and she did not require specific antithyroid therapy, only medications for symptom control. She responded to low dose propranolol 10 mg TID with gradual improvement in symptoms. At 6 weeks her labs were as follows: TSH 9.4(0.3-4.7mcIU/ML), FT4 0.80(0.8-1.7ng/DL), FT3 220(222-383pg/DL), TPO <9.O. This was consistent with subclinical hypothyroidism. She continues to do well on propranolol and will return in 6 months.

Discussion

Hyperthyroidism is caused by overproduction of the thyroid hormone thyroxine.[1] The disease is more common in women and symptoms of hyperthyroidism are usually resent in patients with unequivocal clinical and biochemical markers of the disease.[1]

Many symptoms of hyperthyroidism are non-specific, and can often be missed.

Overt hyperthyroidism generally includes a dramatic constellation of symptoms including anxiety, weakness, tremors, palpitations, and weight loss despite a normal or increased appetite.[2]

Other symptoms include oligo menorrhea, hyper defecation (not diarrhea) and in males gynecomastia and erectile dysfunction.[3]

In patients with mild hyperthyroidism as well as in older patients the symptoms are often referable to one or a few organ systems, so symptoms of unexplained weight loss, new onset atrial fibrillation, and myopathy should lead to an evaluation of hyperthyroidism.[3]

Older patients may also have a phenomenon called "apathetic thyrotoxicosis" which manifests as weakness and asthenia with no other symptoms.[3]

The etiology of hyperthyroidism could either be Graves' disease, thyroiditis, or Plummer disease also known as toxic multinodular goiter with hyperthyroidism[3]

Graves' disease is an autoimmune disorder that may consist of hyperthyroidism, orbitopathy and the occasional dermopathy referred to as pretibial myxedema. The most common feature of Graves' disease is hyperthyroidism and is caused by TSH

receptor antibodies that activate the receptor, thereby stimulating thyroid hormone synthesis and secretion as well as thyroid growth (causing goiter).

The presence of TRAb in serum and orbitopathy on clinical exam distinguish the disorder from other causes of hyperthyroidism.

Hashitoxicosis is a term used to describe patients with autoimmune thyroiditis who initially present with hyperthyroidism. This is followed by the development of hypothyroidism due to infiltration of the gland with lymphocytes and resultant autoimmune destruction of the thyroid tissue.

Toxic multinodular goiter is a result of diffuse hyperplasia of the thyroid follicular cells whose functional capacity is independent of regulation by TSH.

Therapeutic Approach

The therapeutic approach to Graves' hyperthyroidism consists of both rapid amelioration of symptoms with a beta blocker and measures aimed at decreasing thyroid hormone synthesis[4]

This approach is consistent with the management guidelines from the American Thyroid Association:

1) Symptom control: A beta blocker should be started (assuming no contraindications) in most patients as soon as a diagnosis of hyperthyroidism is made, even before confirming the etiology. Beta blockers ameliorate the symptoms caused by increased beta-adrenergic activity like palpitations, tachycardia, tremulousness and anxiety.

2) Decrease thyroid hormone synthesis[4]

   There are three treatment options for Graves' disease and they are the following:

   Anti-thyroid drugs (thionamides), radioiodine therapy or surgery. All three options are effective, but all of them have significant side effects. Because there is no consensus as to the best treatment the ATA guidelines emphasize the importance of fully discussing options with patients and considering their values and preferences before deciding on a treatment plan.

   The three treatment options are not mutually exclusive. Anti-thyroid drugs may be used initially to control symptoms before radioiodine or surgery or they may be used long term. Radioiodine therapy may also be given as initial therapy or after pretreatment with anti-thyroid drugs. Surgery is usually preceded by anti-thyroid drugs to achieve a preoperative euthyroid state.

REFERENCES

1. American Thyroid Association. Hyperthyroidism. [Internet] Available at: https://www.thyroid.org/hyperthyroidism/.
2. Mayo Clinic. Hyperthyroidism (overactive thyroid) – Symptoms and causes. [Internet] Available at: https://www.mayoclinic.org/diseases-conditions/hyperthyroidism/symptoms-causes/syc-20373659.
3. **Ross DS**. Graves' hyperthyroidism in nonpregnant adults: Overview of treatment. In: *UpToDate*, Post, TW (Ed), UpToDate. Waltham, MA, 2022.
4. Mayo Clinic. Hyperthyroidism (overactive thyroid) – Diagnosis & treatment. [Internet] Available at: https://www.mayoclinic.org/diseases-conditions/hyperthyroidism/diagnosis-treatment/drc-20373665.