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Review Article

Thyroid Infections: Hyperthyroidism

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Abstract

Thyrotoxicosis is the clinical syndrome of excessive circulating thyroid hormones, regardless of their source, whereas hyperthyroidism is characterized by increased thyroid hormone synthesis and secretion from the thyroid gland. Graves' disease is the most common cause of hyperthyroidism, followed by toxic nodular goitre. Thyroiditis, iodine and drug-induced thyroid dysfunction, and factitious ingestion of excessive thyroid hormones are additional significant causes of thyrotoxicosis. Graves' disease can be treated with ant thyroid medications, radioactive iodine therapy, or surgery. whereas, due to the high rate of thyrotoxicosis relapse following discontinuation, ant thyroid medications are typically not utilized for an extended period of time in toxic nodular goitre. In symptomatic thyrotoxicosis, blockers may be the only treatment required for thyrotoxicosis that is not caused by excessive thyroid hormone production and release. Thyroid storm and hyperthyroidism during pregnancy and after birth are unique conditions that require careful evaluation and treatment.

Keywords: Disease, Treatment options, Radioactive iodine therapy, Surgery; Anti thyroid drugs

INTRODUCTION

The pathological condition of hyperthyroidism is one in which the thyroid gland produces and secretes too much thyroid hormone. Thyroid radioactive iodine uptake is either normal or high (thyrotoxicosis with hyperthyroidism or true hyperthyroidism). Hyperthyroidism can be overt or subclinical, and it can be caused by extra thyroidal sources of thyroid hormone or by the release of preformed thyroid hormones into the bloodstream with low thyroid radioactive iodine uptake. Low serum concentrations of Thyroid-Stimulating Hormone (TSH) and elevated serum concentrations of thyroid hormone are hallmarks of overt hyperthyroidism: Tri-Iodothyronine (T3), Thyroxin (T4), or both. Serum levels of T4 and T3 are normal but serum TSH is low in subclinical hyperthyroidism. Subclinical hyperthyroidism, which was recently discussed in another Lancet Seminar, is not the subject of our discussion here (de Jonge P et al., 2018).

Europe has a prevalence of 0-8 percent of hyperthyroidism, while the United States has a prevalence of 13%. Hyperthyroidism is more common in women and gets worse with age. Overt hyperthyroidism affects 0-5 percent of people in Europe and 0–5 percent in the United States. Although there are few data on ethnic differences, white people appear to have hyperthyroidism slightly more frequently than people of other races. It has also been reported that iodine-deficient regions have a higher rate of mild hyperthyroidism than iodine-sufficient regions, but that this rate has decreased since universal salt iodization programs were implemented (Park C, 2013).

Graves disease is the most common cause of hyperthyroidism in areas with enough iodine. Graves disease is becoming more common each year in Sweden, with one new case per 100,000 people in the 2000s. The disease is thought to be caused by multiple factors, including the development of autoantibodies that stimulate thyroid follicular cells by binding to the TSH receptor and the loss of immunotolerance. The concordance rate in monozygotic twins is only 17-35%, indicating low penetrance, despite

the fact that several studies have provided some evidence for a genetic predisposition to Graves' disease. Immune-regulatory genes (HLA region, CD40, CTLA4, PTPN22, and FCRL3) and thyroid auto antigens (thyroglobulin and TSH-receptor genes) are the genes that are involved in Graves' disease.8 Non-genetic risk factors for the development of Graves' disease include female sex, psychological stress, and smoking. Sex hormones and chromosomal factors, such as the skewed inactivation of the X chromosome, are thought to be triggers given the higher prevalence of Graves' disease in women. Due to a mechanism of molecular mimicry with the TSH receptor, other potential causes of Graves' disease include infection, particularly with Yersinia enterocolitica, vitamin D and selenium deficiency, thyroid damage, and immunomodulation drugs.8 More research is needed to determine the precise role of these potential causes (Sarris J et al., 2014).

DISCUSSION

Toxic multinodular goitre and single toxic adenoma are two additional common causes of hyperthyroidism. Toxic multinodular goitre and toxic adenoma account for 50% of all cases of hyperthyroidism in iodine-deficient areas, and they are more common in elderly people. In iodine-sufficient areas, about 80% of patients with hyperthyroidism have Graves' disease. Thyroid nodules develop autonomy and independently produce thyroid hormones in response to TSH or TSH-receptor antibody signals (Liem A et al., 2017).

Risk and Complications

The patient's age and gender, comorbidities, duration of the disease, and cause all influence clinical manifestation. While older patients are more likely to experience cardiovascular complications, they present with fewer and less prominent symptoms than younger patients. Hyperthyroid individuals are three times more likely to develop atrial fibrillation than those with healthy thyroids who are over 60. The incidence of embolic stroke caused by atrial fibrillation caused by hyperthyroidism is significantly higher than the incidence of embolic stroke caused by atrial fibrillation caused by non-thyroidal causes. However, the use of anticoagulants in hyperthyroid patients with atrial fibrillation is still up for debate. In patients with hyperthyroidism, it is also thought that atrial fibrillation is an independent predictor of congestive heart failure. Patients with hyperthyroidism were found to have a higher risk of all-cause mortality, with heart failure accounting for the majority of cardiovascular events (Vohra S et al., 2005)

Phytotoxic periodic paralysis is another serious side effect of hyperthyroidism. Asian patients are more likely to experience it: In Japan, incidence is 2% while it is 0-2% in North America. It is brought on by a transfer of potassium into the muscle cells and is characterized by the triad of muscle paralysis, acute hypokalaemia, and thyrotoxicosis. The disease may be caused by mutations in potassium channels, which are regulated transcriptionally by thyroid hormones. To prevent arrhythmias and restore muscle function, treatment with low potassium doses and non-selective blockers should be initiated as soon as possible if the condition is suspected. Osteoporosis and abnormalities in the reproductive system, such as gynaecomastia in men40 and decreased fertility and menstrual irregularities in women, are additional long-term thyrotoxicosis complications (Grace S et al., 2010).

Diagnosis

Because it has the highest sensitivity and specificity for diagnosing thyroid disorders, serum TSH should be measured first. To distinguish between subclinical hyperthyroidism (with normal circulating hormones) and overt hyperthyroidism (with increased thyroid hormones), serum free T4 or free T4 index, and free or total T3 concentrations should be measured if they are low. As in patients with TSH-secreting pituitary adenomas or peripheral thyroid hormone resistance, it also identifies disorders with normal or slightly elevated TSH concentrations and elevated thyroid hormone concentrations. Thyrotoxicosis is often diagnosed using a variety of different methods. These variations are in part due to differences in population characteristics, cultural backgrounds, and socioeconomic factors. Unless the clinical diagnosis of Graves' disease is established, the American Thyroid Association (ATA) and the American Association of Clinical Endocrinologists (AACE) recommend a thyroid radioactive iodine uptake test for hyperthyroidism and thyrotoxicosis. Europe, Japan, and Korea favor the utilization of thyroid ultrasound and the evaluation of TSH-receptor antibodies (also known as thyroid-stimulating immunoglobulin, or TSAs). When the radioactive iodine uptake test is unavailable or contraindicated, the US guidelines suggest measuring TRAb as an alternative method of diagnosis for Graves' disease. The Brazilian Thyroid Consensus concurs with this recommendation, stating that radioactive iodine uptake is preferred over TRAb testing for the initial assessment of thyrotoxicosis and that TRAb testing is only useful in a small number of cases. Utilizing ultrasound and TRAb measurements, we adhere to the practices of our European and Asian colleagues in our clinical practice. In Graves' disease patients, a thyroid radioactive iodine uptake test would reveal a diffusely increased uptake. Radioactive iodine uptake, on the other hand, would be normal or high, with an asymmetrical and irregular pattern in toxic multinodular goitre and a localized and focal pattern in toxic adenoma. The remaining thyroid tissue would also have less radioactive iodine uptake (Templeman K et al., 2011).

Symptoms

1. Fatigue

2. Weight gain

3. Cold intolerance

4. Slowed heart rate, movements, and speech

5. Joint and muscle pain, cramps, and weakness

6. Constipation

7. Dry skin

8. Thin, brittle hair or fingernails

9. Decreased sweating

10. Pins and needles

11. Heavy periods, or menorrhagia

12. Weakness

13. High cholesterol

Treatment

Antithyroid Drugs (ATDs), radioactive iodine ablation, and surgery are the three treatment options for hyperthyroidism. Graves' disease patients would benefit from any of the three treatment options, but patients with toxic adenoma or toxic multinodular goitre should have surgery or radioactive iodine therapy because these conditions rarely go into remission. ATDs are typically used to restore euthyroidism in patients with toxic nodular goitre prior to surgical or radioactive iodine treatment. They are rarely used as long-term treatment unless the other two therapies are contraindicated or the patient has a short life expectancy (Lake J et al., 2012).

Depending on where you live, Graves' disease can be treated in a variety of ways. In North America, radioactive iodine therapy is frequently used as a first treatment beyond the United States; Primary treatment with ATDs is preferred, while definitive therapy is only offered to patients with hyperthyroidism that persists or recurs. Additionally, thyrotoxicosis patients may take blockers to alleviate symptoms (Pengpid S et al., 2018).

CONCLUSION

Patients with difficult sub-intense or easy (most regularly happening during the early post pregnancy time frame) thyroiditis have a self-restricted course of thyrotoxicosis followed by hypothyroidism and as a rule reclamation of thyroid function. Effortless or post pregnancy lymphocytic thyroiditis habitually repeats during ensuing pregnancies and can bring about long-lasting hypothyroidism. In painless or postpartum lymphocytic thyroiditis, positive thyroid peroxidase antibodies are almost always present. As a result, these patients require ongoing monitoring for the possibility of developing hypothyroidism. Because thyroid radioactive iodine uptake is low and thyroid hormone synthesis is not increased, ATDs and radioactive iodine therapy are contraindicated in both disorders. During the thyrotoxic phase, blockers are frequently administered to patients. NSAIDs or salicylates may help alleviate the thyroid pain and other systemic symptoms in patients with painful sub-acute thyroiditis. In more severe cases, glucocorticoids like prednisone (15-40 mg daily) are preferred.139 In contrast to patients with painless lymphocytic thyroiditis after childbirth; those with painful sub-acute thyroiditis rarely develop permanent hypothyroidism (Stepleman LM et al., 2015)

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None

CONFLICT OF INTEREST

None

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