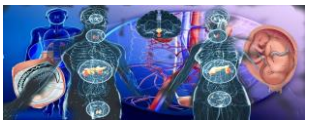
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Short Communication **International Journal of Endocrinology and Disorders** **Open Access**

Global Prevalence of Endocrine Disorder Hyperthyroidism Risks and Management

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Abstract

Hyperthyroidism is characterised by increased thyroid hormone synthesis and secretion from the thyroid gland, whereas thyrotoxicosis refers to the clinical syndrome of excess circulating thyroid hormones, irrespective of the source. The most common cause of hyperthyroidism is Graves' disease, followed by toxic nodular goitre. Other important causes of thyrotoxicosis include thyroiditis, iodine-induced and drug-induced thyroid dysfunction, and factitious ingestion of excess thyroid hormones. Treatment options for Graves' disease include antithyroid drugs, radioactive iodine therapy, and surgery, whereas antithyroid drugs are not generally used long term in toxic nodular goitre, because of the high relapse rate of thyrotoxicosis after discontinuation. β blockers are used in symptomatic thyrotoxicosis, and might be the only treatment needed for thyrotoxicosis not caused by excessive production and release of the thyroid hormones.

Keywords: Hyperthyroidism; thyrotoxicosis; thyroid hormones; thyroxine; tri-iodothyronine; salt iodisation; programmes

Introduction

Hyperthyroidism is a pathological disorder in which excess thyroid hormone is synthesised and secreted by the thyroid gland. It is characterised by normal or high thyroid radioactive iodine uptake (thyrotoxicosis with hyperthyroidism or true hyperthyroidism). Thyrotoxicosis without hyperthyroidism is caused by extrathyroidal sources of thyroid hormone or by a release of preformed thyroid hormones into the circulation with a low thyroid radioactive iodine uptake.

What causes hyperthyroidism?

Hyperthyroidism has several causes. They include:

- Graves' disease, an autoimmune disorder in which your immune system attacks your thyroid and causes it to make too much hormone. This is the most common cause.
- Thyroid nodules, which are growths on your thyroid. They are usually benign (not cancer). But they may become overactive and make too much thyroid hormone. Thyroid nodules are more common in older adults.
- Thyroiditis, inflammation of the thyroid. It causes stored thyroid hormone to leak out of your thyroid gland.
- Too much iodine. Iodine is found in some medicines, cough syrups, seaweed and seaweed-based supplements. Taking too much of them can cause your thyroid to make too much thyroid hormone.
- Too much thyroid medicine. This can happen if people who take thyroid hormone medicine for hypothyroidism (underactive thyroid) take too much of it.
- **What are the treatments for hyperthyroidism?**

The treatments for hyperthyroidism include medicines, radioiodine therapy, and thyroid surgery:

- **Medicines** for hyperthyroidism include
  - Antithyroid medicines, which cause your thyroid to make less thyroid hormone. You probably need to take the medicines for 1 to 2 years. In some cases, you might need to take the medicines for several years. This is the simplest treatment, but it is often not a permanent cure.
  - Beta blocker medicines, which can reduce symptoms such as tremors, rapid heartbeat, and nervousness. They work quickly and can help you feel better until other treatments take effect.
- **Radioiodine therapy** is a common and effective treatment for hyperthyroidism. It involves taking radioactive iodine by mouth as a capsule or liquid. This slowly destroys the cells of the thyroid gland that produce thyroid hormone. It does not affect other body tissues. Almost everyone who has radioactive iodine treatment later develops hypothyroidism. This is because the thyroid hormone-producing cells have been destroyed. But hypothyroidism is easier to treat and causes fewer long-term health problems than hyperthyroidism.
- **Surgery** to remove part or most of the thyroid gland is done in rare cases. It might be an option for people with large goiters or pregnant women who cannot take antithyroid medicines. If you have all of your thyroid removed, you will need to take thyroid medicines for the rest of your life. Some people who have part of their thyroid removed also need to take medicines.

Pathophysiology

Graves' hyperthyroidism is an autoimmune condition that arises as a result of the loss of immunological tolerance to the thyroid-stimulating hormone receptor (TSHR). Elevated circulating thyroid hormones in Graves' hyperthyroidism arise because of stimulating TSHR autoantibodies (TRAbs), which bind to leucine-rich repeats in the extracellular domain of the TSHR located on the surface of the thyrocytes This mimics the action of thyroid-stimulating hormone (TSH; thyrotropin) resulting in excessive, autonomous thyroid hormone production and hyperplasia of thyroid epithelial cells. The mature TSHR is a G protein–coupled receptor found primarily on thyroid follicular cells.

The TSHR is also found in orbital fibroblasts and the upper dermis where binding of TRAbs results in a proliferative response that contributes to the extrathyroidal signs seen in Graves' hyperthyroidism, GO, and pretibial myxedema. Clinical manifestations of GO affect about 25% of people diagnosed with Graves' hyperthyroidism, and it can be facially disfiguring, may cause functional visual disabilities, and carries the potential for occasional loss of sight.

Novel Therapeutic Strategies

B-lymphocyte depletion (CD20 depletion)

As a humorally driven condition, the essential role of B cells in Graves' hyperthyroidism provides a logical therapeutic target for immunomodulatory treatment. The B-cell depleting therapy rituximab (RTX) has been used for more than 20 years to treat lymphoproliferative malignancies, such as lymphoma, with increasing use over the past decade to treat autoimmune disease.

Blocking immunoglobulin recycling (FcRn therapeutics)

The long half-life associated with IgG antibodies, such as TRAbs, is attributed to the neonatal immunoglobulin Fc receptor (FcRn), which recycles endocytosed IgG antibody by binding it in the acidic conditions of the lysosome and recycling it to the cell membrane for release back into the circulation.

Inhibition of B-cell proliferation and differentiation (blockade of B-cell activating factor)

B-cell activating factor (BAFF) is a member of the TNF family of cytokines and has an essential role in B-lymphocyte activation, differentiation, and survival. Elevated circulating BAFF levels have been found in patients with several autoimmune conditions, including active Graves' hyperthyroidism, where both degree of elevation of thyroid hormones and TRAb concentrations have been demonstrated to correlate with serum BAFF levels.

TSHR-specific immunotherapy

Conventional approaches to combating autoimmune disease involve using drugs with broad immunosuppressive effects and hence the potential side effect of susceptibility to infection. An alternative approach that has been successfully used to treat allergic disease is to desensitize the immune response to the specific allergen (immune stimulant).

Conclusion

Treatment of hyperthyroidism has not changed greatly in the past several decades. Choices are between long-term therapy, with risk of relapse, or destruction of the thyroid gland with subsequent hypothyroidism. ATDs are a conservative option, but have about a 50% relapse rate; however, thyroidectomy and radioactive iodine treatment are definitive therapies, but with subsequent hypothyroidism needing lifelong therapy with thyroid hormone replacement.

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