CAUSES AND TREATMENT OF HYPERTHYROIDISM

Is It Graves' Disease or Toxic Multinodular Goiter or Thyroiditis?

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Hyperthyroidism has several causes besides Graves' disease, and treatment varies with each cause. This article describes the causes and treatment of hyperthyroidism.

Graves’ disease

Graves’ disease is responsible for most cases of hyperthyroidism, especially in younger women, although men and women of any age may be affected. Graves' disease is an autoimmune disorder that targets the thyroid gland. In Graves’ disease stimulating TSH receptor antibodies (also known as thyroid stimulating immunoglobulins or TSI) react with the TSH receptor (TSHr), which is positioned on the surface of thyroid, orbital, pituitary, dermal, and skeletal muscle cells.

The TSHr is the primary autoantigen targeted in Graves’ disease. The subsequent binding of TSI to the TSH receptor stimulates thyroid cells, ordering them to produce more of the thyroid hormones: thyroxine (T4) and triiodothyronine (T3), causing hyperthyroidism.

The gender profile for Graves' disease, similar to that of most autoimmune diseases, is that it is a predominately a female issue. Ranges vary from publication to publication, however the general consensus is that a ratio of 8:1 women to men are affected.

Toxic Multinodular Goiter

Thyroid nodules are clusters of thyroid tissue. When more than one nodule is seen, the condition is called multinodular goiter. When the nodules begin to produce excess thyroid hormone, the condition is called toxic multinodular goiter. In this condition toxic refers to thyrotoxicosis, a condition caused by hyperthyroidism.

Similar to patients with Graves’ disease, patients with toxic multinodular goiter may have levels of Free T3 (FT3) that are higher relative to levels of FT4. In both conditions, the RAI-uptake is elevated. Because patients with toxic multinodular goiter are initially hypothyroid, they may show elevated levels of thyroglobulin and TPO antibodies. These antibodies may also be elevated in patients with Graves’ disease.

Thyroiditis

Thyroiditis is a condition of thyroid gland inflammation. Thyroiditis may occur during the postpartum period (postpartum thyroiditis, which occurs within one year after childbirth, miscarriage, or abortion; after respiratory or sinus infection (infectious
thyroiditis) or for no apparent reason (silent thyroiditis). In these conditions, hyperthyroidism generally lasts about 8 weeks and is followed by a similar period of hypothyroidism, which may be permanent. Hyperthyroidism may also be preceded by a temporary condition of hypothyroidism.

In addition, patients with Hashimoto’s thyroiditis may have an early hyperthyroid phase or they may develop TSI antibodies, which cause transient symptoms of hyperthyroidism. Patients with Hashimoto’s thyroiditis may also spontaneously move into autoimmune hyperthyroidism or Graves’ disease after several months or many years of treatment with thyroid replacement hormone.

**Treatment**

Current treatment strategies are Radioiodine Ablation (RIA), Antithyroid drugs (ATDs) and surgical removal of the thyroid (thyroidectomy). While ATDs offer the possibility of remission, surgery and RAI permanently remove or destroy the gland. Many experts today recommend that aggressive therapies be reserved for patients who can not tolerate ATDs or alternative medicine. In most parts of the world, ATDs remain the treatment of choice for patients with hyperthyroidism.

In the United States, it has been reported that almost 70% of the members of the American Thyroid Association prefer the ablative approach as a first option for treatment of Graves' patients. Physicians claim that the correct amount of I131 (Radioactive Iodine, RAI) can be administered to create a euthyroid or "normal" condition. Physicians try to adjust the dose of radioactive iodine to destroy only enough of the thyroid gland to bring its hormone production back to normal, without reducing thyroid function too much; others use a larger dose to completely destroy the thyroid. Most of the time, people who undergo this treatment must take thyroid hormone replacement therapy for the rest of their lives. (Merck Rx).

Nonetheless, a long-term study based on data from 1965-2002 that included over 2000 patients, and was published in Clinical Endocrinology 2004, concluded that; RAI treatment of Hyperthyroid Graves' patients resulted in 82% developing Hypothyroidism between 1 and 25 years post treatment. The ablative therapy leaves the patient without any alternative treatment options and a lifetime of hormone replacement therapy.

Antithyroid drug (ATD) therapy involves the prescription of immunomodulatory drugs like Methimazole (MMI) or Propylthiouracil (PTU). These strategies allow the patient to maintain their thyroid gland and end up with normal thyroid function. The standard school of thought has been 12-18 months of treatment. However, remission can take considerably longer and treatment should continue until remission occurs and not be based on an arbitrary timeframe.

A paper published in Clinical Endocrinology in 2005 described a study of patients that remained on ATD for an average of 4 years (some as many as 10 years) who had a
remission rate of 88%. Additionally, another study found in Thyroid 2000 vol. 10 demonstrated that Graves’ disease patients that exhibited a "smooth" decline in their antibody levels, or population, after ATD therapy and was said to be a predictor of remission. 36 out of 44 patients, or 82%, exhibited a "smooth" decline of TSI levels and were antibody and symptom free 1 year after ATDs were discontinued.

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