

What's the Rush in Treating Graves Disease?

This article explains the reasons why Graves' patients should not be rushed into having aggressive permanent treatment.

Patients newly diagnosed with Graves' disease (GD) are frequently rushed into having aggressive treatment to destroy their thyroid glands. The reasons for this, they're told, are to prevent the condition from worsening and to ward off conditions of thyroid storm. The reasons I have been telling patients not to rush into treatment are similar.

Aggressive treatment tends to worsen the existing autoimmune disorder, increasing thyroid antibody production and causing a transient increase in hyperthyroidism and perpetuating the autoimmune process. In addition, thyroid storm is often triggered by radioiodine because of the increase in thyroid hormone levels and thyroid antibodies and occasionally it is triggered by surgery. Finally, unlike some other diseases, Graves' disease is not typically progressive. Its natural course is a waxing and waning of symptoms and periods of remission alternating with periods of variable symptoms. Only rarely does the disease progress although over time the effects of extended untreated hyperthyroidism can eventually damage other systems. Treatment, whether with medications or herbal remedies, is usually needed to lower thyroid hormone levels until the natural disease course winds down.

Autoimmune Nature of Graves' Disease

Another very important reason I object to rushing into aggressive therapy is that Graves' disease is an autoimmune disorder. The thyroid gland is the victim, not the cause in GD. The disease process in Graves' disease is fascinating and fairly straightforward. The immune system, sensing that our thyroid tissue is foreign, is triggered or stimulated to launch an immune response directed at thyroid tissue. Normally, the immune system protects us from foreign substances and infectious agents, and it prevents cancerous cells from developing. In autoimmune diseases, the immune system is hyper-vigilant and confuses our body's cells with foreign substances.

In the autoimmune response responsible for GD, the immune system produces various destructive immune system chemicals as well as autoantibodies that react with a specific protein known as the TSH receptor. This protein is like a lock sitting on the edge of thyroid and other cells. Normally, the pituitary hormone TSH, which is thyroid-stimulating—hormone or thyrotropin, is the only substance that can open or react with this lock. When it opens the lock, TSH enters and tells thyroid cells to produce thyroid hormone.

Normally, small pulses or bursts of TSH activate the lock all day long, picking up speed at night. By doing so, the pituitary ensures that thyroid cells are producing adequate hormone for the body's needs.

TSH Receptor Antibodies

In Graves' disease, the immune system produces stimulating TSH receptor antibodies, which are also known as thyroid stimulating immunoglobulin (antibodies) or TSI. As their name implies, they too, like TSH, stimulate or activate the TSH receptor. In doing so, they also cause increased production of thyroid hormone.

Even when the pituitary gland tries to fix the problem and stops secreting TSH, TSI keep ordering thyroid cells to produce excess hormone. When this happens, the thyroid gland is no longer under pituitary control. The amount of hormone it produces is dependent on levels of TSI.

TSI are the direct cause of hyperthyroidism in Graves' disease and various immune system chemicals known as cytokines also contribute to the symptoms that occur in this disorder.

Treatment Considerations

At one time, before Graves' disease was found to be an autoimmune disorder, aggressive therapy seemed like a wise option. However, today, now that the autoimmune nature of Graves' disease is well known, at least to most physicians, therapy, using anti-thyroid drugs (ATDs) is aimed at reducing TSI production and helping the immune system heal and recover.

Aggressive therapy using surgery removes some TSI because these antibodies are primarily stored in thyroid tissue. However, TSI levels can increase later if the immune system is still stimulated by environmental triggers to produce TSI.

On the other hand, when radioiodine is used to ablate or destroy the gland, the thyroid gland and immune system cells within the gland perceive this as a foreign attack. Consequently, TSI production dramatically rises. This evokes an increase in hyperthyroidism that persists until most of the thyroid tissue is destroyed. Radioiodine molecules stay within the body for a long time.

Even though RAI molecules have a short-half life, trillions of atoms are administered in the RAI dose. Studies show that these RAI molecules in patients receiving RAI ablation for GD can trigger airport radiation detectors for a minimum of 8 weeks. How long these molecules truly persist isn't known. However, it's known that TSI production persists, peaking within the year and remaining elevated for many years. And it's known that RAI causes a slight but significant increase in thyroid and small bowel cancer in mortality studies. Patients who develop cancer but do not die from it aren't included in these studies so the true long-term effects remain unknown.

Furthermore, as time passes, the immune system begins to produce more blocking than stimulating TSH receptor antibodies. These antibodies block both TSH and TSI from activating the lock on thyroid cells. At about 6 years after radioiodine ablation, atrophic hypothyroidism develops, worsening conditions of radioiodine-induced hypothyroidism. And because both stimulating and blocking TSH receptor antibodies contribute to thyroid eye disease, eye and skin complications often occur at this time. In addition, the condition of thyroid acropachy, a form of elephantiasis, is known to occur up to 30-40 years after radioiodine ablation. Acropachy is a form of soft-tissue swelling primarily of the fingers and toes.

Treatment-induced Hypothyroidism

Hypothyroidism caused by thyroidectomy surgery is usually more predictable. In the recommended procedure of partial thyroidectomy, hypothyroidism develops but it is milder than that seen after RAI ablation. It also tends to improve about 6 months after surgery. Thyroid hormone levels fluctuate for the first year, but usually levels begin to stabilize within a year or two after surgery. After radioiodine, the influences of stimulating and blocking antibodies cause a more dramatic fluctuation in thyroid hormone levels. In addition, these and also T4, T3, and TSH antibodies, can affect laboratory results, making it difficult to accurately determine one's thyroid status.

The goal of therapy using anti-thyroid medications or natural medicine is healing the immune system. When the immune system heals and patients stop producing TSH receptor antibodies, thyroid function returns to normal. Until then, meds are used to lower thyroid hormone levels and assist the healing process. Lifestyle changes are another important part of healing. If stress triggered GD, the immune system cannot heal until measures are taken to reduce the effects of stress. Stress reduction techniques, such as meditation, tai chi, yoga, or any form of mild exercise, promote immune system health. Diets free of chemicals, including subsidized iodine products, also allow the immune system to heal.

There are no shortcuts in treating GD although some people naturally have a shorter and more benign disease course than others. Destroying the gland will eliminate hyperthyroidism and induce hypothyroidism, but this is not a true cure. Nor does it promote healing. If circumstances haven't changed, the immune system will continue on its erratic path, which has been intensified by the radioiodine assault. Consequently, other autoantibodies are frequently produced, and other autoimmune diseases can develop. Aggressive treatment is permanent. There is no going back.

Anti-thyroid drugs and alternative methods work while they're used and then these substances are stopped when they're no longer needed (after remission has been achieved). Isn't this reason enough to ask what the rush is when deciding on treatment?