AUTOIMMUNE HYPOTHYROIDISM

Subtypes and Causes

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Read on to learn about the subtypes, symptoms, diagnosis, treatment, and disease course in autoimmune hypothyroid disorders.

Classifications of Autoimmune Disease

Autoimmune disorders are classified as systemic, which means that they involve multiple bodily organs and systems, or organ-specific, which means that they primarily target one organ. Autoimmune thyroid disorders can cause hypothyroidism, a condition of diminished thyroid function, or hyperthyroidism, a condition of increased thyroid function. Both types of thyroid dysfunction cause specific symptoms and have specific consequences.

Autoimmune Hypothyroidism Subtypes

Autoimmune hypothyroidism is the most common organ-specific autoimmune disorder. Two specific forms of autoimmune hypothyroidism exist: 1) chronic autoimmune thyroiditis, which is also known as Hashimoto's thyroiditis or Hashimoto's disease, and its variants, postpartum and sporadic thyroiditis, and 2) autoimmune atrophic thyroiditis, which is also known as primary myxedema.

Myxedema

Myxedema is a term referring to the skin changes characterized by pitting and swelling (water-logged appearance) associated with hypothyroidism. Before laboratory tests for thyroid function were developed, most hypothyroid disorders were diagnosed on the basis of goiter and myxedema. The severity of autoimmune hypothyroidism varies ranging from subclinical hypothyroidism, which is described in my 5-21-06 article, to fatal conditions of myxedema coma. This article describes the causes, symptoms, diagnosis and treatment of autoimmune hypothyroidism.

Causes

Autoimmune hypothyroidism is caused by a combination of genetic and environmental factors. That is, people with certain immune and organ-specific genes are predisposed to developing autoimmune thyroid disorders when they're exposed to certain environmental triggers.

Several of these genes and environmental triggers have been identified but genetic tests aren't used outside of research since immune system genes aren't specific; they cause predisposition to several different autoimmune disorders. Environmental triggers include:
iodine deficiency, iodine excess, lithium, selenium deficiency, cigarette smoke, sex steroids, trauma, interferon-alpha used in hepatitis C and other disorders, infection with Yersinia and viruses, iodine-rich medications such as amiodarone, and iodine contrast dyes used in imaging tests.

**Symptoms**

Symptoms vary although patients with Hashimoto's disease and its variants are more likely to develop goiter and patients with atrophic thyroiditis typically have a small firm thyroid gland characterized by fibrous or scar tissue. Because hypothyroidism affects all of the body's organs and systems, a variety of different symptoms can occur. Most patients do not have all of these symptoms, but rather develop a constellation of predominant symptoms that can change over time and with treatment.

Depression is one of the earliest symptoms to occur in hypothyroidism, followed by skin and hair changes. Myxedema is one of the most characteristic symptoms and is associated with accumulations of mucopolysaccharides beneath the skin. These mucin deposits may thicken the tongue, surround the heart, leading to congestive heart failure, and lead to diminished function of virtually any organ. The skin in hypothyroidism is cold, dry and pale and the palms may take on a yellow tinge associated with the inability to properly metabolize vitamin A. This hue may also occur on the soles and the folds on each side of the nose. Hair loss is common and the outer third of the eyebrow is usually sparse. Nails may appear thickened, brittle and grow slowly.

Other common symptoms include fatigue, exercise intolerance, shortness of breath, sleep apnea, hypertension, cognitive changes, peripheral and entrapment neuropathy, increased lipid levels, reduced drug and hormone metabolism, joint pain, constipation, weight gain, nutrient deficiencies, diminished lung capacity, neuromuscular weakness, dyscoordination, edema, mineral imbalances, behavioral disturbances, gastrointestinal disturbances, malabsorption, liver dysfunction, anemia, hearing loss, diminished adrenal function, menstrual changes, infertility, diminished libido, decreased adrenergic responses, schizophrenic and affective psychoses, decreased bone metabolism, carpal tunnel syndrome, gait disorder, seizures, and in severe hypothyroidism, progression to myxedema coma.

**Diagnosis**

The type of autoimmune hypothyroidism one has isn't usually distinguished since treatment remains the same although a radioiodine uptake test or ultrasound may be used to diagnose variant types that can spontaneously resolve quickly or move into hyperthyroidism. Patients with Hashimoto's thyroiditis may also move into Graves' disease after months or years of being on thyroid replace hormone. When a move into hyperthyroidism is suspected, tests for TSI, the antibodies found in Graves' disease are usually performed. The primary tests to diagnose hypothyroidism include TSH, FT4, and FT3 levels and tests for thyroid antibodies. In hypothyroidism, the TSH is elevated and
the FT4 and FT3 levels are below the reference range. Positive tests for thyroid antibodies confirm that hypothyroidism is autoimmune.

Blood tests considered obsolete and no longer recommended to diagnose hypothyroidism include the T3 uptake, reverse T3 and the T4 index. The TRH stimulation test is only needed in the rare instances that central hypothyroidism and thyroid hormone resistance are suspected. In these cases, TSH would be low in the presence of low thyroid hormone levels.

Most patients with Hashimoto's thyroiditis have high levels of thyroglobulin and TPO antibodies and about 10 percent of patients have blocking TSH receptor antibodies. People with autoimmune variants, such as postpartum thyroiditis, usually have lower levels of these antibodies. About 60 percent of patients with atrophic thyroiditis have blocking TSH receptor antibodies.

The normal cell destruction or apoptosis of thyroid cells appears to be accelerated in people with Hashimoto's thyroiditis. Tissue changes include lymphocytic infiltration.

**Atrophic thyroiditis**

Atrophic thyroiditis is considered the opposite of Graves' disease in that TSH is blocked from activating thyroid cells. The signal is lost and thyroid hormone fails to be produced. Consequently, thyroid cells fail to grow even when TSH levels become markedly elevated. Tissue changes in atrophic thyroiditis are characterized by fibrosis and stunted cell growth, and hypothyroidism generally progresses to complete thyroid failure.

**Treatment**

Thyroid replacement hormone remains the mainstay for treating hypothyroidism. Both synthetic and porcine glandular formulations of T4 and T3 are available. Because autoimmune disorders tend to wax and wane, frequent laboratory tests are necessary to ensure that an optimal dose is used.

Autoimmune disorders benefit from treatments that help strengthen, not stimulate, the immune system, including a nutrient-rich diet, correction of nutrient deficiencies, and stress reduction techniques. Avoiding environmental triggers and keeping allergies in control are also known to help. Because autoimmune hypothyroidism can resolve or move into hyperthyroidism, regular monitoring of FT4 and FT3 levels is important.