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Commentary

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Pathophysiology of Hashimoto's Thyroiditis and its Treatment

Jaion Cela*
Department of Paediatric Cardiology, University Hospital Münster, Münster, Germany

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Description

Hashimoto's thyroiditis, also known as chronic lymphocytic thyroiditis and Hashimoto's disease, is an autoimmune disease in which the thyroid gland is gradually destroyed. In the early stages, symptoms may not be noticed. Over time, the thyroid gland can enlarge, forming a painless goiter. Some people develop hypothyroidism over time, accompanied by weight gain, fatigue, constipation, depression, hair loss, and general aches and pains. After many years, the thyroid gland usually decreases in size. Potential complications include thyroid lymphoma. Moreover, because untreated Hashimoto's patients often develop hypothyroidism, additional complications can include, but are not limited to, high cholesterol, heart disease, heart failure, high blood pressure, myxedoma, and possible problems with pregnancy.

Hashimoto's thyroiditis is thought to be caused by a combination of genetic and environmental factors. Risk factors include a family history of the disease and the presence of another autoimmune disease. The diagnosis is confirmed by blood tests for TSH, T4 and antithyroid autoantibodies. Other conditions that can cause similar symptoms include Graves' disease and non-toxic nodular warts. Hashimoto's thyroiditis is usually treated with levothyroxine. If hypothyroidism is not present, some may not recommend treatment, while others may provide treatment to try to reduce the size of the goiter. Sufferers should avoid consuming large amounts of iodine; however, adequate iodine is required especially during pregnancy. Surgery is rarely required to treat goiter. Hashimoto's thyroiditis affects about 5% of Caucasians at some point in their lives. It usually starts between the ages of 30 and 50 and is much more common in women than men. The incidence rate seems to be increasing. This disease was first described by the Japanese doctor Hakaru Hashimoto in 1912. It was recognized as an autoimmune disease in 1957.

Pathophysiology

Multiple mechanisms by which the pathology of Hashimoto's thyroiditis develops have been proposed. Various autoantibodies against thyroid peroxidase, thyroglobulin, and TSH receptors may be present, although a small percentage of people may have none of these antibodies. As shown in various twin studies, a percentage of the population can also have these antibodies without developing Hashimoto's thyroiditis. However, antibody-dependent cell-mediated cytotoxicity is an essential factor in apoptosis in Hashimoto's thyroiditis. Activation of cytotoxic T lymphocytes (CD8+ T cells) in response to a cell-mediated immune response driven by helper T lymphocytes (CD4+ T cells) is central to thyrocyte destruction. As is characteristic of type IV hypersensitivity, macrophage recruitment is another effect of helper T cell activation, with Th1 axis lymphocytes producing inflammatory cytokines in thyroid tissue to further activate macrophages and migrate to the thyroid gland for direct effects.

Gross morphologic changes in the thyroid gland are manifested by a general enlargement that is much more nodular and irregular than more diffuse patterns (eg, in hyperthyroidism). While the capsule is intact and the gland itself is still distinct from the surrounding tissue, microscopic examination can give a clearer indication of the level of damage.

Histologically, hypersensitivity is seen as a diffuse infiltration of the parenchyma by lymphocytes, especially plasma B cells, which can often be seen as secondary lymphoid follicles (germinal centers not to be confused with the colloid follicles normally present that make up the thyroid gland). Atrophy of colloid bodies is lined by Gürtle cells, cells with intensely eosinophilic granular cytoplasm, metaplasia from normal cuboidal cells lining thyroid follicles. Severe thyroid atrophy is often manifested by denser fibrous bands of collagen that remain

Contact: Jaion Cela, E-mail: jaioncela@gmail.com

within the thyroid capsule.

Treatment of Hashimoto's thyroiditis

There is no cure for Hashimoto's, but hormone replacement medications can regulate hormone levels and restore normal metabolism. The tablets are available in several different strengths. The exact dose your doctor will prescribe will depend on a number of factors, including;

• Age

• Weight

• Severity of hypothyroidism.

• Other health problems.

• Other medications that may interact with synthetic thyroid hormones.

• Once you start treatment, your doctor will order a lab test called a Thyroid-stimulating Hormone (TSH) test to monitor your thyroid function and make sure you're getting the right dose. Because thyroid hormones work very slowly in the body, it may take several months for your symptoms to go away and your goiter to shrink. However, a large goiter that does not improve may require thyroid removal.