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HASHIMOTO'S THYROIDITIS - AN AUTOIMMUNE DISEASE

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ABSTRACT

Hashimoto's thyroiditis, often known as Hashimoto's illness. In Hashimoto's disease, the thyroid gland is attacked by the immune system, which results in inflammation and impairs the thyroid glands capacity to generate thyroid hormones. The thyroid develops an abundance of lymphocytes, which are white blood cells. Lymphocytes produce the antibodies that set off the autoimmune reaction. Hypothyroidism, or decreased thyroid function, is a frequent consequence of Hashimoto's illness. When the thyroid does not produce enough thyroid hormone to meet the body's needs, hypothyroidism is a condition that develops. Almost every organ in the body is impacted by thyroid hormones, which also govern metabolism and how the body consumes energy. Numerous bodily processes slow down when there is less thyroid hormone.

Keywords: Hashimoto's thyroiditis, Inflammation, Lymphocytes, Hypothyroidism and Autoimmune process.

INTRODUCTION

The most frequent autoimmune thyroid condition and leading cause of hypothyroidism is Hashimoto's thyroiditis (HT), which was first identified in 1912 by Hakaru Hashimoto. The thyroid gland is a butterfly-shaped endocrine gland that is frequently located in the lower front of the neck. The thyroid's function is to produce thyroid hormones, which are then secreted into the blood and distributed to all bodily tissues. The body uses thyroid hormones to use energy, stay warm, and keep the brain, heart, muscles, and other organs functioning normally.[1, 2]

Those most influenced are women. At least ten to one is the ratio of women to men. Most women are diagnosed between the ages of 30 and 50, although some sources claim that this is when it

happens more frequently. Levothyroxine at the recommended dose of 1.6 to 1.8 mcg/kg/day makes up the standard of care. T4 is converted to T3, which is the human body's active form of thyroid hormone. Excessive supplementation can have harmful and morbid effects, including arrhythmias (Atrial fibrillation is the most frequent) and osteoporosis.[3, 4]

A condition known as hypothyroidism, in which the thyroid gland is unable to produce enough hormones due to the immune system's production of antibodies that harm the thyroid gland, is an auto-immune disorder. The enzyme TPO, also known as thyroid peroxidase, is crucial for the synthesis of thyroid hormones. Most people who have Hashimoto's disease are middle-aged women. Men, women, and children of all ages are also affected. Another factor contributing to Hashimoto's disease is heredity.[5]

PATHOPHYSIOLOGY OF HASHIMOTO'S THYROIDITIS [6]

There are a number of theories as to how the pathology of Hashimoto's thyroiditis arises. Although a small percentage of people may not have any of these antibodies, thyroid peroxidase, thyroglobulin, and TSH receptor autoantibodies may all be present. Some people may also have these antibodies without going on to develop Hashimoto's thyroiditis, as evidenced by numerous twin studies. However, in Hashimoto's thyroiditis, antibody-dependent cell-mediated cytotoxicity is a crucial component of apoptosis.

Thyrocyte destruction relies heavily on the activation of cytotoxic T lymphocytes (CD8+ T cells) in response to a cell-mediated immune response mediated by helper T lymphocytes (CD4+ T cells). Helper T cell activation also results in macrophage recruitment, which is a hallmark of type IV hypersensitivity. Th1 axis lymphocytes produce inflammatory cytokines in thyroid tissue, which further activate macrophages and cause them to migrate to the thyroid gland for direct effects. Gross morphologic changes in the thyroid gland are indicated by a general enlargement that is significantly more nodular and irregular than more diffuse patterns (such as in hyperthyroidism). A microscopical examination can provide a clearer picture of the degree of damage, even though the gland's capsule is intact and can still be distinguished from the surrounding tissue.

According to histology, hypersensitivity is characterised by a diffuse infiltration of the parenchyma by lymphocytes, particularly plasma B cells, which are frequently visible as secondary lymphoid follicles (germinal centres should not be confused with the colloid follicles that normally make up the thyroid gland). Colloid bodies that have atrophy are lined by Hurthle cells, which have developed from the normal cuboidal cells that line thyroid follicles and have an intensely eosinophilic granular cytoplasm. The thyroid capsule may still contain thick, fibrous bands of collagen in cases of severe thyroid atrophy.

SYMPTOMS OF HASHIMOTO'S THYROIDITIS

Depression, sluggishness, fatigue, and a cold-sensitivity Constipation, dry, pale skin, and a swollen face; broken nails; loss of hair; expansion of the tongue; being overweight; Aches, weakness, tenderness, and stiffness in the muscles; joint discomfort; bleeding during a period (prolonged bleeding); memory loss. [7]

ETIOLOGY OF HASHIMOTO'S THYROIDITIS

Hashimoto disease's origin is not well understood. The majority of patients develop antibodies to several thyroid antigens, with anti-thyroid peroxidase (anti-TPO) being the most prevalent. Many also develop TSH receptor-blocking antibodies (TBII) and anti-thyroglobulin (anti-Tg) antibodies. These antibodies attack the thyroid tissue, ultimately resulting in insufficient thyroid hormone production. Only a small portion of people, roughly 10% to 15%, have the clinically obvious disease and lack serum antibodies. TPO antibody positives predict the clinical syndrome.[8, 9]

It may be a component of type 2 Polyglandular Autoimmune Syndrome with type 1 DM and autoimmune adrenal deficiency. Numerous other autoimmune conditions, including celiac disease, adrenal insufficiency, and pernicious anaemia, are linked to Hashimoto thyroiditis. Nonthyroidal Autoimmune Diseases are linked to Hashimoto disease, and their prevalence increased after an adult diagnosis. [10, 11]

RISK FACTORS HASHIMOTO'S THYROIDITIS

An increased risk of Hashimoto's disease is linked to the following factors:-

- **Sex:** Hashimoto's disease is much more common in women.
- **Age:** Although Hashimoto's disease can strike at any age, it tends to strike in middle age.
- **Additional autoimmune illnesses:** Your risk of developing Hashimoto's disease increases if you already have an autoimmune condition like lupus, type 1 diabetes, or rheumatoid arthritis.
- **Family history and Genetics:** If there are thyroid conditions or other autoimmune diseases in your family, you are more likely to develop Hashimoto's disease.
- **Pregnancy:** Hashimoto's disease that develops after pregnancy may be influenced by typical changes in immune function during pregnancy. Consuming too much iodine. People who are already at risk for Hashimoto's disease may be triggered by eating too much iodine

- **Exposure to radiation:** People with Hashimoto's disease are more likely to be exposed to high doses of environmental radiation. [12]

DIAGNOSIS OF HASHIMOTO'S THYROIDITIS

Testing thyroid function

The healthcare professional will request blood tests that might include the following to determine whether hypothyroidism is the underlying cause of your symptoms:

TSH test: The pituitary gland creates TSH, or thyroid stimulating hormone. TSH is sent to the thyroid to cause an increase in thyroid hormone production when the pituitary detects low levels of thyroid hormones in the blood. A high blood TSH level is a sign of hypothyroidism.

T-4 tests: Thyroxine (T-4) is the primary thyroid hormone. A low blood level of T-4 supports the results of a TSH test and suggests that the thyroid itself is the source of the issue.

Antibody tests

Hypothyroidism can be caused by a variety of medical conditions. Your doctor will ask for an antibody test to see if Hashimoto's disease is the root of your hypothyroidism. An antibody's primary function is to alert other immune system players to foreign pathogens that must be eliminated. When a person has an autoimmune disorder, their immune system makes errant antibodies that attack the body's healthy proteins or cells. Thyroid peroxidase (TPO), a protein that is crucial for the production of thyroid hormone, is typically the target of an immune system antibody in Hashimoto's disease. TPO antibodies are frequently found in the blood of people with Hashimoto's disease. It may be necessary to perform laboratory tests for additional antibodies linked to Hashimoto's disease.[13,14]

TREATMENT OF HASHIMOTO'S THYROIDITIS

The cornerstone of treating hypothyroidism is thyroid hormone replacement. Levothyroxine sodium, titrated for oral administration, is the preferred medication. It can be administered daily and has a half-life of seven days. To prevent insufficient absorption, it should not be administered along with iron or calcium supplements, aluminium hydroxide, or proton pump inhibitors. For optimal absorption, it is best taken first thing in the morning on an empty stomach.

The usual dose is 1.6 - 1.8 mcg/kg per day; however, it can differ from patient to patient. A regular full dose should be started for people under 50 years old; however, patients with cardiovascular disorders and the elderly should get lesser doses. The beginning dose for people older than 50 years old is 25 mcg/day, with a reevaluation in six to eight weeks. Levothyroxine doses must be raised in patients

with short bowel syndrome in order to maintain a euthyroid state, while thyroxine doses must be increased by 30% during pregnancy.

The evidence in favor of an autoimmune or anti-inflammatory diet is weaker. Leaky gut syndrome, in which the gut mucosa is injured and allows proteins that would not normally enter the circulation through transporters in the gut mucosa, is the idea behind the inflammation. According to one theory, a reaction resembling molecular mimicry takes place, and antibodies are made against the antigens. Unfortunately, there is a chance that the antigen will cause antibodies to develop against thyroid peroxidase because of its strong structural similarity. The idea behind an autoimmune diet is to improve gut health and lessen the severity of the autoimmune reaction. Before this subject is included in the recommendations, further research must be done on it.[15]

CONCLUSION

Patients with Hashimoto's thyroiditis, a common endocrine condition, are more likely to be female than male. An autoimmune disease unique to one organ is called Hashimoto's thyroiditis. Thyroid tissue invasion by lymphocytes and the development of antibodies against thyroid proteins such as thyroid peroxidase, TPO ab, thyroglobulin, and Tgab are its defining features. Hypothyroidism or normal thyroid function may coexist with the existence of Hashimoto's thyroiditis. Information on the pathophysiology, symptoms, etiology, risk factors, diagnosis, and treatment of Hashimoto's thyroiditis is included in the current review.

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