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### Autoimmune Thyroiditis: A Look into Hashimoto’s Disease

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#### Introduction

Thyroid disease is a general term utilized to describe a variety of conditions affecting the thyroid gland, including autoimmune, hypothyroidism, the deficient production of thyroid hormone; hyperthyroidism, the excess production of thyroid hormone; and goiter, the physiologic swelling of the thyroid gland (Choleva, 2001). Thyroid disease is one of the most common endocrine disorders (Berg, 2014). It is a major health concern, and one in which scientists have a thorough understanding of the pathophysiology is vital in continuing advancements to take place in the areas of diagnosis, treatment, and prevention of this disease. (Berg, 2014).

#### Pathophysiological Process

In HD, increased lymphocytic activity leads to the destruction of thyroid follicular cells, either by accelerated apoptosis or as a direct result of T cell activity, concluding in increased lymphocytic infiltration of the thyroid gland (Thompson, 2014). Structural damage to the thyroid gland, coupled with autotoxic antibodies, results in thyroid gland dysfunction, resulting in inadequate hormone production and secretion. Consequently, laboratory findings include decreased thyroxine (T4), possible decreased triiodothyronine (T3), and the presence of circulating autoantibodies, resulting in a hypothyroid state (Thompson, 2014). Symmetrical thyroid gland enlargement ensues with fibrous densities occurring over time, resulting in the presence of glandular nodules (Thompson, 2014).

#### Significance of Pathophysiology

Pathophysiology integrates scientific and clinical research to advance knowledge in the area or particular condition, such as autoimmune thyroiditis. The understanding of both basic and clinical research is essential for significant advancements to take place in the areas of diagnosis, treatment, and prevention of such conditions. This is of particular importance in HD, as the pathophysiology of this disease is currently not entirely understood. As such, although treatment based on speculation is available, there is no cure for this condition (Waren, 2014). As with any disease process, the ultimate goal is health, and with HD, a thorough understanding of the pathophysiology is vital in continuing advancements to one day include successful disease prevention.

#### Case Study

An 87-year-old female presented to her primary care practitioner with nonspecific complaints of fatigue, cold intolerance, weight gain, depression, and difficulty with concentration. Her vital signs were as follows: BP 140/102 mm Hg, HR 68 beats/minute, RR 16 breaths/minute, SpO2 98% on room air, and oral temperature 97.2°F. Upon physical exam, the patient was noted to have mild facial swelling with periorbital edema; dry, scaling skin; thick, brittle fingernails; and an enlarged, multinodular thyroid gland. All other physical exam findings were within normal limits.

#### Signs and Symptoms

Signs and symptoms of Hashimoto’s thyroiditis are directly related to the degree of hypothyroidism experienced. HD-related hypothyroidism typically has an insidious onset, progressing over a period of months or years, eventually leading to manifestations of hypothyroidism, the deficient production of thyroid hormone. HD-related nonspecific symptoms may include the following:

- **Fatigue**
- **Lethargy**
- **Cold intolerance**
- **Dry Skin**
- **Constipation**

As the disease progresses, more severe symptoms may develop, including the following:

- **Cold intolerance**
- **Joint pain**
- **Decreased peripneum**
- **Swollen lymph nodes**
- **Severe fatigue**
- **Dysphagia**
- **Dizziness**
- **Numbness**
- **Tremors**
- **Nonspecificconstitutional symptoms**

In conclusion, HD is a common autoimmune disease of the thyroid gland and is the most common cause of hypothyroidism in the United States, with approximately 6% as some in different parts of the country (Lin, 2014). With left untreated, HD results in many troubles and perhaps debilitating symptoms, as such, early diagnosis of HD is essential to recognize and properly diagnose this chronic health condition.

#### References


### Signs and Symptoms of Hashimoto’s Thymorrhysis

- Fatigue
- Decreased energy
- Ataxic gait
- Hair loss
- Sleep disturbances
- Slowed Movement
- Cold intolerance
- Weight gain
- Periorbital
- Bradycardia
- Hypertension (diastolic)
- Vocal changes or dysphagia

Autoimmune Thyroiditis is an autoinmune thyroid disease of the United States, most frequently occurring as a result of chronic autoimmune thyroiditis, specifically Hashimoto’s Disease (Waren, 2014).

Hashimoto’s disease (HD) is an organ-specific autoimmune condition characterized by the presence of antibodies to various thyroid self antigens (Kristensen, Hegedüs, Madsen, Smith, & Nielsen, 2014). The pathogenesis is not entirely understood at this time; however, it is believed that both environmental and genetic factors may play a role in development of HD (Park, Grywalska, Mariusz, Paterek, & Rolinski, 2015).

In HD, lymphocytic infiltration of the thyroid gland occurs and CD4 T cells produce large amounts of cytokines, including TNF, Th1, Th2, and T cell. Th2 cells activate Th1 and Th17 T cells and cytokines and interleukin-17 (IL-17). Th17 cells activate cytokine-interleukin and macrophages, resulting in the destruction of thyroid follicular cells (Pyzik et al., 2015). T cells play an extensive role in producing B cells and plasmacytoid cells, which produce anti-thyroid antibodies (anti-TPO), antithyroglobulin (anti-Tg), and anti-TSH receptor (aTSHR) antibody, resulting in thyroids (Ji et al., 2014). Th17 cells produce interleukin-17, which leads to the recruitment of neutrophils and subsequent inflammation (Ji et al., 2014).

Increased lymphocytic activity leads to the destruction of thyroid follicular cells, either by way of accelerated apoptosis or as a direct result of T cell activity, concluding in progressive damage to the thyroid parenchyma (Pyzik et al., 2015). An additional subset of CD4 T cells, known as regulatory T cells (Treg cells), work to suppress an overabundant immune response, working in direct contrast to the aforementioned Th17 cells. As such, an imbalance of the Th17/Treg system plays an important role in the development and suppression of HD (Ji et al., 2014).

Structural damage to the thyroid gland, coupled with autotoxic antibodies, results in thyroid gland dysfunction, resulting in inadequate hormone production and secretion. Consequently, laboratory findings include decreased thyroxine (T4), possible decreased triiodothyronine (T3), and the presence of circulating autoantibodies, resulting in a hypothyroid state (Thompson, 2014). Symmetrical thyroid gland enlargement ensues with fibrous densities occurring over time, resulting in the presence of glandular nodules (Thompson, 2014).

Diagnostic testing was ordered for the patient, including 12 lead electrocardiogram (EKG), echocardiogram, and an ultrasound of the thyroid gland. The EKG demonstrated sinus bradycardia with low voltage QRS and nonspecific ST changes. Laboratory findings were as follows: elevated thyroid stimulating hormone (TSH) 10.2 mU/L (reference range 0.2-4.5 mU/L), suppressed T4 1.8 µg/dl (reference range 5.4-11.5 µg/dl), and HD. Thyroid autoantibodies including anti-TPO and anti-Tg were detected. The patient has also noted to have mild anemia, with the hemoglobin and hematocrit measured at 10.8 mg/dl and 32%, respectively (reference range 14 mg/dl and 45%), and Warem, 2014). Additional laboratory studies were within normal limits.

Ultrasoundography (US) of the thyroid gland was performed, and the presence of a diffusely enlarged, heterogeneous gland was found, in addition to multiple hypoechoic nodules ranging in size from 0.5 to 1.3 cm. Coupled with the laboratory and EKG findings, a diagnosis of Hashimoto’s Thyroiditis was confirmed. The patient was subsequently started on levothyroxine therapy and, after a therapeutic dose was achieved by way of serial thyroid hormone levels, the patient reported a significant decrease in symptoms with a complete return to normal activities of daily living.

#### Images

- [Image 1](image1.png): History of thyroiditis in autoimmune thyroiditis: d. n.
- [Image 2](image2.png): Thyroid gland, m. thyroiditis, l. severe thyroiditis

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