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Summer 8-5-2022

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Bhattarai, Ojaswi, "Hashimoto Thyroiditis" (2022). *Nursing Student Class Projects (Formerly MSN)*. 523.
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Hashimoto Thyroiditis

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Introduction

- Hashimoto's thyroiditis, also known as chronic lymphocytic thyroiditis, is an autoimmune disease that causes thyroid under activity, hypothyroidism in which the body's immune system attacks the thyroid gland and causes gradual destruction of thyroid tissue (Mincer & Jialal, 2022).
- The thyroid is an endocrine gland located at the front of the neck that produces both T3 (triiodothyronine) and T4 (thyroxine) which regulate metabolism. The thyroid gland is regulated by hormones produced by the pituitary gland (Winnifred & Milas, 2022).
- Currently, there is no clear understanding of the causes of Hashimoto's thyroiditis, but it is believed to be caused by the combination of genetics, environment, physical activity, immunity, dietary intake, and epigenesis factors that lead to an immunological deficit (Ragusa, et al., 2019).
- Having personally suffered from Hashimoto's thyroiditis and having family members who have been affected, this topic was chosen to introduce this disorder and explore current understanding.

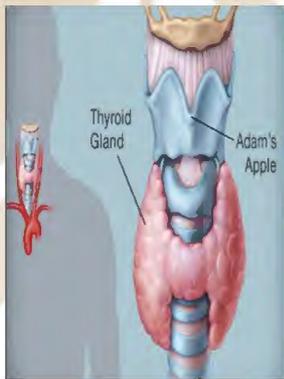


Figure 1: (Dunkin, 2020)

Signs and Symptoms

In the early stages of Hashimoto's disease, a person may not experience any symptoms, but as the disease progresses, one or more of the symptoms of hypothyroidism may appear.

- fatigue and muscle weakness
- insensitivity to cold
- weight gain
- trouble tolerating cold
- joint and stiffness
- constipation
- dry skin or dry, thinning hair
- heavy or irregular menstrual periods or fertility problems
- slowed heart rate
- decreased memory or concentration (Dolan, et al., 2018)



Figure 2: (Hrt.org, 2019)

Pathophysiology

- Studies have shown that genetic variation of the Major Histocompatibility Complex (MHC) antigens is often associated with Hashimoto thyroiditis (Bliddal, et al., 2017).
- The main feature of HT is the Infiltration of thyroid autoantibodies (antithyroid peroxidase and antithyroglobulin antibodies), autoreactive T lymphocytes, natural killer cells, and inflammatory cytokines in the thyroid tissue (Bliddal, et al., 2017).
- When autoreactive CD4+ T-helper cells start to react to thyroid antigens, thyrocyte death occurs following various immunologic mechanisms including binding of antithyroid antibodies, CD8+ cytotoxic T cell-mediated cell death, cytokine-mediated cell death, and induction of apoptosis (Ragusa, et al., 2019).
- The antibodies attach to the thyroid peroxidase enzyme, thyroglobulin, and TSH receptors, which can halt the production of hormones. Afterward, the thyroid gland may undergo fibrosis, and atrophy, and ultimately be replaced (Ragusa, et al., 2019).

Significance of Pathophysiology

- Thyroid autoimmune diseases affect approximately 5% to 6% of the population, with up to 15% of people in their 30s and 50s being affected. The most common autoimmune thyroid condition is HT (Klubo & Wartofsky, 2022).
- Around 1 to 5 % of the population suffers from Hashimoto's thyroiditis, and women between the ages of 30 and 50 are 4 to 10 times more likely to have Hashimoto's disease than men (NIDDK, 2022).
- Prevalence of HT increases in populations with pre-existing autoimmune conditions, women, advanced age, and family history. It is common for Hashimoto's disease to be associated with other autoimmune disorders (Bliddal, et al., 2017).

Diagnosis and Treatment

- Diagnoses are made based on the level of circulating autoantibodies, especially thyroid peroxidase (TPO) and anti-thyroglobulin (Tg), thyroid stimulating hormone (TSH), thyroxine (T4), and classical features of thyroid ultrasounds. Thyroid peroxidase antibody is the most common anti-thyroid antibody found in Hashimoto's thyroiditis, while antibodies against thyroglobulin are frequently also found (Ragusa, et al., 2019).
- The treatment of overt hypothyroidism (elevated TSH and low thyroid hormone levels) consists of thyroid hormone replacement. An appropriate dose of synthetic levothyroxine taken orally can restore normal thyroid hormone levels and improve hypothyroid symptoms (American thyroid association, 2022).

Implications for Nursing Care

- It is essential for nurses to understand the pathophysiology of Hashimoto thyroiditis to effectively diagnose, treat, and educate patients with this condition. Since the symptoms are not very disease-specific and quite vague, recognizing them and acting promptly is the best way to stop the problem from progressing.
- In individuals with long family histories of thyroid disease, as well as in individuals predisposed to the development of other autoimmune disorders, additional diagnostic tests should be conducted by an advanced nurse practitioner as soon as symptoms appear.
- Patients should be educated about medication adherence, follow-up, regular lab work, and other topics to avoid complications like myxedema coma, goiter, and heart disease. Additionally, some patients may develop lymphoma, so regular neck examinations are highly recommended as well.
- It may be necessary to monitor serum TSH levels every 6-8 weeks at the start of treatment or after a dose change until the ideal dose is determined. It is essential to have a nurse practitioner monitor thyroid hormone levels regularly, as some people may become toxic from one standard dose of levothyroxine, resulting in hyperthyroidism (American thyroid association, 2022).

Summary

- Hashimoto's thyroiditis (HT) is an autoimmune disease characterized by thyroid-specific autoantibodies that attack thyroid cells. This causes the body's mechanisms to slow down, leading to fatigue, coldness, weight gain, dry skin, and hair loss. Thyroid autoimmune diseases affect approximately 5% to 6% of the total population.
- Nurse practitioners are in the front line where they educate patients, initiate therapies, prevent complications, and increase the quality of life for patients to live symptoms-free.
- It is imperative for nurse practitioners to know about Hashimoto thyroiditis and be able to diagnose it appropriately since HD manifests numerous unpleasant and sometimes life-threatening symptoms without treatment.

References

