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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 hypothyroidism, the deficient production of thyroid hormone; hyperthyroidism, the excess production of thyroid hormone; and goiter, the physiologic swelling of the thyroid gland (Cihakova, 2001; Warren, 2014). Primary hypothyroidism is the most prevalent thyroid disease in the United States, most frequently occurring as a result of chronic autoimmune thyroiditis, specifically Hashimoto's Disease (Warren, 2014).

Hashimoto's disease (HD), also known as Hashimoto's thyroiditis and human autoimmune thyroiditis, was named after Hakaru Hashimoto, a Japanese medical scientist who first described the condition in 1912 (Cihakova, 2001). HD is a chronic autoimmune condition characterized by the presence of lymphocytic infiltration, serum autoantibodies, and destruction of thyroid tissue; as a result, hypothyroidism often ensues (Liu et al., 2014; Warren, 2014).

According to Rugge, Bougatsos, and Chou (2015), HD has been associated with increased risk for coronary artery disease, congestive heart failure, decreased bone density, and varying negative musculoskeletal, dermatologic, and gastrointestinal effects. As such, HD is a major health concern, and one in which health care practitioners should be well versed.

HD is of particular significance to this author, as my younger sister if afflicted by this condition. As a result, she has undergone multiple biopsies, two surgeries, and countless laboratory tests and medication adjustments .

Pathophysiological Process

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

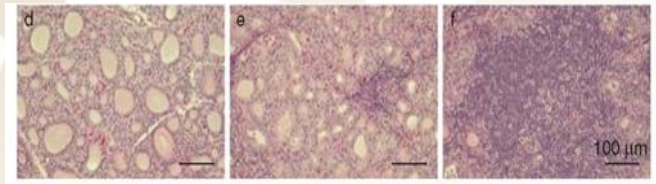
In HD, lymphocytic infiltration of the thyroid gland occurs and $CD4^+$ T cells produce large amounts of cytokines, including Th1, Th2, Th17, and Tfh cells. Th1 and Th2 cells work to produce interferon-gamma and interleukin-4; additionally, Th1 cells activate cytotoxic lymphocytes and macrophages, resulting in the destruction of thyroid follicular cells (Pyziket al., 2015). Th2 cells incite an excessive production of B cells and plasmatic cells which produce autoantibodies, including anti-thyroid peroxidase (anti-TPO), antithyroglobulin (anti-Tg), and anti-TSH receptor (TSHR) antibody, resulting in thyroiditis (Liu et al., 2014). Th17 cells produce interleukin-17, which leads to the recruitment of neutrophils and subsequent inflammation (Liu 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 overzealous 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 of HD (Liu et al., 2014).

Structural damage to the thyroid gland, coupled with autoantibodies precipitates 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 of a particular condition, such as autoimmune thyroiditis. The understanding of basic and complex pathophysiological processes allows 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 process is currently not entirely understood. As such, although treatment based upon symptomatology is available, there is no known cure at this time (Warren, 2014). As with any disease process, prevention is the ultimate goal and, with HD, a thorough understanding of the pathophysiology is vital in continuing advancements to one day include successful disease prevention .



Above: Histology of the thyroid gland in autoimmune thyroiditis: d, normal thyroid gland; e, mild thyroiditis; f, severe thyroiditis
© Yoki et al., 2012

Case Study

A 47 year old female presented to her primary care practitioner with nonspecific complaints of fatigue, cold intolerance, weight gain, mild depression, and sleep disturbances, all of indeterminate onset. She denied fever, chills, or recent illness. Her vital signs were measured as follows: BP 146/102 mmHg, HR 58 beats/minute, RR 16 breaths/minute, SpO2 98% on room air, and oral temperature 97.2º 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, multi-nodular thyroid gland. All other physical exam findings were within normal limits.

Above: An enlarged , multinodular thyroid gland, typical in autoimmune thyroiditis. © Wellness Alternatives , 2013

Diagnostic testing was ordered for the patient, including 12 lead electrocardiogram (EKG), labwork, 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 0.2-5.4 mU/L) and decreased thyroxine (T4) 1.8 µg/dl (reference range 5.4-11.5 µg/dl; Warren, 2014). Thyroid autoantibodies including anti-TPO and anti-Tg were detected. The patient was 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 41%; Warren, 2014). Additional laboratory studies were within normal limits.

Ultrasonography (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 1-3 mm. 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 measurements of TSH and T4 laboratory measurements, the patient reported a significant decline of symptoms with a complete return to normal activities of daily living.

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 to years. Early, nonspecific symptoms may include the following:

- Fatigue
- Lethargy
- Weight gain
- Dry Skin
- Constipation

As the disease progresses, more severe symptoms manifest, including the following:

- Cold intolerance
- Hair loss
- Joint pain
- Decrease perspiration
- Slowed Movement
- Decreased energy
- Depression
- Menstrual irregularities
- Sleep disturbances
- Daytime somnolence
- Vocal changes or dysphagia may also be present as a result of thyroid gland enlargement

Physical exam of a patient with HD may reveal the following findings:

- Bradycardia
- Hypertension (diastolic)
- Thickened, brittle nails
- Diminished deep tendon reflexes
- Periorbital edema and puffy face
- Macroglossia
- Slow, thick speech
- Ataxic gait
- Infertility, sleep apnea, and mild nerve deafness may also be present (Cihakova, 2001).

Nursing Implications

It is important for nursing professionals at all levels to have a basic understanding of Hashimoto's thyroiditis, but it is essential for the advanced practice nurse (APN) to maintain an in-depth knowledge base concerning this disease process. This is of particular importance due to the fact that symptomatology often presents in a nonspecific, furtive manner. As such, in caring for patients with such vague complaints, the APN must be able to understand and apply existing knowledge concerning HD to order and interpret appropriate testing, in which to effectively and accurately make a diagnosis. As an APN, it is also important to recognize the suspected genetic component of HD, therefore potentially conducting appropriate screenings to at risk populations at the onset of any symptoms suspicious of HD (Pyzik et al., 2015).

Conclusion

In conclusion, HD is a common autoimmune disorder of the thyroid gland and the most common cause of hypothyroidism in the United States, with an incidence as high as 6% in some parts of the country (Lee, 2014). If left untreated, HD results in many troubling and perhaps debilitating symptoms; as such, it is important for healthcare providers to maintain an awareness of HD, including the knowledge essential to recognize and properly diagnose this chronic health condition.

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