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 gland's swelling. Hypothyroidism can be caused by a variety of factors, such as Hashimoto's thyroiditis, an autoimmune disease that affects the thyroid gland. The signs and symptoms of hypothyroidism include fatigue, decreased energy, and mood changes. In severe cases, it can result in a range of complications including infertility, sleep apnea, and medication adjustments.

Pathophysiologic Process

Hashimoto's disease (HD) is an organ-specific autoimmune condition characterized by the presence of antibodies to various thyroid self-antigens (Kristensen, Hegedüs, Mahler, Smith, & Brønnum-Hansen, 2015). The pathogenesis of HD is not well understood at this time, but it is believed that both environmental and genetic factors may play a role in development (Pyzik, Cihakova, & Pyziket, 2015). In HD, lymphocytic infiltration of the thyroid gland occurs and CD4+ T cells produce large amounts of cytokines, including Th1, Th2, Th17, and Th cells. Th cells and CD4+ T cells play a role in the development of autoimmune thyroiditis. In addition, Th17 cells activate cytotoxic lymphocytes and macrophages, resulting in the destruction of follicular thyroid cells (Pyzik et al., 2015). Th1 cells elicit an extensive 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 (Liou et al., 2016). Th17 cells produce interleukin-17, which leads to the recruitment of neutrophils and subsequent inflammation (Liou et al., 2016).

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. She denied fever, chills, or recent illness. Her vital signs were measured as follows: BP 140/102 mmHg, HR 168 beats/minute, RR 18 breaths/minute, SpO2 98% on room air, and oral temperature 37.2°F. Upon physical exam, the patient was noted to have mild facial swelling with periorbital edema, dry, scaling skin, thin, 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 to years. Early, non-specific symptoms may include the following:

- Fatigue
- Lethargy
- Dry Skin
- Constipation

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

- Cold intolerance
- Heat intolerance
- Joint pain
- Decreased perspiration
- Slow/Movement
- Increased energy
- Depression
- Menstrual irregularities
- Sleep disturbances
- Daytime somnolence
- Vocal changes or dysphagia

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 mIU/L (reference range 0.25–4.5 mIU/L) and decreased thyroid hormone (Th1) 1.8 mIU/L (reference range 5.4–11.5 µg/dL, p=0.004). Thyroid autoantibodies including anti-TPO and anti-Tg were detected. The patient was also noted to have mild anemia, with a hematocrit and hemoglobin measured at 10.8 g/dL and 32%, respectively (reference range 40–41 g/dL, p=0.0004). Additional laboratory studies were within normal limits.

Ultrasonography 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 and up. 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 function studies and T4 laboratory measures, the patient reported a significant decrease in symptoms with a complete return to normal activities of daily living.

References


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Significance of Pathophysiology

Pathophysiologic integrates scientific and clinical research to advance knowledge in the area of a particular condition, such as autoimmune thyroiditis. The understanding of basic and clinical research is essential for health care providers to maintain an in-depth knowledge base concerning this disease process. This is of particular importance due to the fact that pathophysiology often presents in a non-specific, vague 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 supportive components 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 of up to 7% in some parts of the country (Lee, 2014). As such, it is important for health care providers to maintain an awareness of HD and continue to keep a clinical eye out for its essential to recognize and properly diagnose this chronic health condition.