Hashimoto’s Disease: The Underactive Thyroid Disease

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Introduction

The thyroid gland is part of the endocrine system and has a widespread function that controls multiple organ systems and processes. The main function of the thyroid gland is to produce T3 and T4 hormones (by the release of thyroid stimulating hormone (TSH)) or is not activated successfully by the pituitary gland (by the lack of thyroid stimulating hormone (TSH)). Although, is hyperthyroidism, the thyroid gland contains an increase of circulating thyroid hormones continues, an overall slowing of the person’s metabolism occurs. The underactive thyroid disease is called hypothyroidism (Cherina, 2013).

Pathophysiologic Process and Significance

Although there are several causes for hypothyroidism, Hashimoto’s (HT) is the most common cause of hypothyroidism in United States and is thought to be associated with genetic susceptibility and environmental factors. HT is an autoimmune disease and can present in a woman or a man with or without symptoms. In the initial stage of HT, HT can be so subtle it is left untreated (Davies, 2015).

The Thyroid Gland

HT is caused by an overactive autoimmune response causing thyroid gland destruction. Loss of immune tolerance to the regulatory T cells leads to the production of antibodies directed against thyroid tissue, which causes destruction of the thyroid gland. This destruction inhibits the release of T4 and T3 hormones that are essential for healthy processes. The resultant lack of thyroid hormones leads to a variety of metabolic processes.

The disorder begins with an activated inflammatory process when an individual contacts an antigen antigen that is environmentally (outside diet, toxins, viral infection) presented for HT. This inflammatory process involves the accumulation of the human leukocyte antigen (HLA) class II molecules (APCs) into the thyroid gland. HLA class II molecules then stimulate T helper cells that present antigens-provoking cells such as dendritic cells and macrophages. HLA class II presents antigens from outside of the cell to T-helper cells. This stimulates the multiplication of T-helper cells. T-helper cells then stimulate B-cells to produce antibodies specific to the antigen. When there is an accumulation of HLA class II molecules in the thyroid gland, this causes damage to thyroid antigens and causes the release of specific thyroid proteins that are then seen in the blood as antigen-specific TSH. From here, the APC travels from the thyroid gland to the lymph nodes of the body. When the encoded APC interacts with a T-helper cells, the draining lymph nodes become activated. An autoimmune process against the thyroid gland occurs. This autoimmune process can affect the thyroid gland, resulting in the destruction of thyroid follicles, lymphocytes, and macrophages infiltrate and accumulate in the thyroid gland through the formation, clonal expansion, and maturity of self-reactive T-helper and T-suppressor lymphocytes. This thyroid gland destruction inhibits the release of the thyroid hormones Triiodothyronine (T3) and Thyroxine (T4). This lack of hormone release leads to hypothyroidism and significantly decreases Thyroid (Davies, 2015).

Signs and Symptoms

Since the thyroid gland regulates the entire metabolism, the thyroid gland indirectly affects every cell, tissue, and organ in the body—from muscles, bones, hair, and skin to the digestive track, heart, and brain. Since the thyroid gland is a multisystem function, there are a wide variety of signs and symptoms that may be present with Hashimoto’s disease and/or any form of hypothyroidism. Regardless of which type of hypothyroidism a person may present, the possible signs and symptoms will be the same. Initially, abnormalities of the entire thyroid gland may be (mild and without symptoms), but as the disease progresses the symptoms become more apparent.

Signs and symptoms that can be seen in someone with a hypothyroidism are fatigue, constipation, dry skin, increased sensitivity to cold, muscle weakness, cramps or stiffness, coarse hair, breath intolerance, hoarseness, sore throat, depression, menstrual irregularities, heavy and excessive bleeding. Physical examination usually reveals goiter, weight change, muscle weakness, decreased metabolic processes.

Hashimoto’s thyroid autoantibodies (anti thyroid antibodies) occur against the thyroid gland. In thyroid gland, the pituitary gland (by the release of the thyroid gland hormones) are produced. When the encoded APC interacts with an activated T-helper cells, the draining lymph nodes become activated. An autoimmune process against the thyroid gland occurs. This autoimmune process can affect the thyroid gland, resulting in the destruction of thyroid follicles, lymphocytes, and macrophages infiltrate and accumulate in the thyroid gland through the formation, clonal expansion, and maturity of self-reactive T-helper and T-suppressor lymphocytes. This thyroid gland destruction inhibits the release of the thyroid hormones Triiodothyronine (T3) and Thyroxine (T4). This lack of hormone release leads to hypothyroidism and significantly decreases Thyroid (Davies, 2015).

Additional Sources


References Cited

Glick, A. B., Wodzinski, F., Liu, Y., Zhu, C., & Beck, V. (2012). Treatment for HT depends upon goiter activity and/or if hypothyroidism is present. A person with HT does have hypothyroidism and/or a goiter, continued monitoring should take place. If a person with HT has a goiter but is still free of hypothyroidism, health professionals are provoked to an effort to decrease the size of the goiter. When a person with HT is diagnosed with hypothyroidism (TSH 4-10; T4 4-7), treatment is needed to avoid the goiter becoming severe. When a person with HT will eventually develop hypothyroidism due to the destruction of the thyroid gland, the TSH and T4 may present normally since hypothyroidism has not occurred at this time. For a definitive diagnosis, the goiter must be present for at least 2 years. Antithyroid antibodies are tested to test for severe Santorph antisubstances. This test confirms the presence of autonumthases that mistakenly attack the thyroid tissue. A computerised tomography (CT) scan may also be performed to assess the thyroid gland for size, texture, nodules, and/or inflammation. (NIH, 2013).

Conclusion

In conclusion, HT is characterized by the loss of thyroid follicular cells, hypothyroidism, and the presence of autoantibodies (autoimmune thyroiditis). The thyroid gland is affected by the release of thyroid peroxidase (TPO) and thyroglobulin (Tg). The thyroid tissue is involved as having an infiltration of T-cells and T-cells which are reactive against the thyroid antigens. The lymph system of the thyroid gland becomes involved and this leads to the death of the thyroid gland tissue. "Nonclassic" signs and symptoms of Hashimoto’s thyroiditis (Glick, Wodzinski, Fu, & Levine, W., 2013).