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Hashimoto's Disease: The Underactive Thyroid Disease

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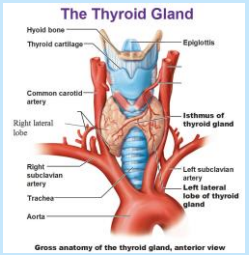
Introduction

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 and secrete two different thyroid hormones: triiodothyronine (T3) and thyroxine (T4), both of which help to meet the metabolic demands of the entire body. When the thyroid gland fails to produce T3/T4 hormones (primary gland failure) or is not activated successfully by the pituitary gland (by the release of thyroid stimulating hormone (TSH)), or hypothalamus, this results in low levels of circulating thyroid hormone. As low levels of the circulating thyroid hormones continue, an overall slowing of the person's metabolism occurs. This underactive thyroid disease is called hypothyroidism (Chiasera, 2013).

Although there are several causes for hypothyroidism, Hashimoto's Thyroiditis (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 a chronic autoimmune disease and can present itself in an aggressive form or a mild form such as silent thyroiditis or postpartum thyroiditis leading to an undiagnosed hypothyroidism. Regardless of the virulence of the disease, HT can become serious if left untreated (Davies, 2015).

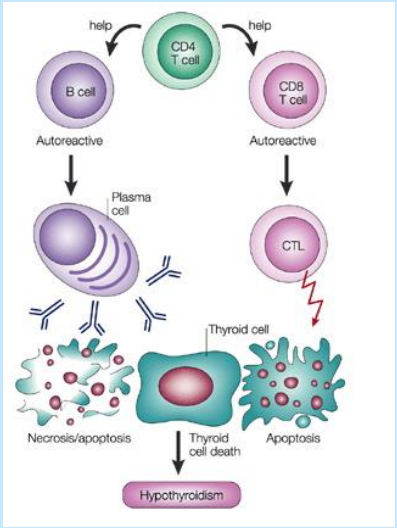


Gross anatomy of the thyroid gland, anterior view

Pathophysiological Process and Significance

HT is caused by an overt autoimmune response causing thyroid gland destruction. Loss of immune tolerance (lack of regulatory T-cells) to normal thyroid cells leads to the production of antibodies directed against thyroid tissue, which causes destruction of the thyroid gland. This destruction inhibits the release of thyroid hormones necessary for the metabolic processes.

The disease begins with an activated inflammatory process when an individual is genetically (familial) and environmentally (iodine diet, toxins, viral infect) predisposed for HT. This inflammatory process initiates the accumulation of the human leukocyte antigen (HLA) Class II antigen-presenting cells (APC) into the thyroid gland. HLA Class II is a set of molecules found on antigen-presenting cells such as dendritic cells and subclasses of macrophages. HLAs present antigens from outside of the cell to T-lymphocytes. This stimulates the multiplication of T-helper cells. T-helper cells then stimulates B-cells to produce antibodies to the specific antigen. When there is an accumulation of HLA Class II molecules in the thyroid gland, this causes damage to thyrocytes and causes the release of thyroid-specific proteins that are then seen on the cell surface of the encoded APC. From here, the APC travels from the thyroid gland to the draining lymph nodes of the thyroid. When the encoded APC interacts with the T-lymphocyte cells and B-lymphocyte cells in the draining lymph nodes, a production of thyroid autoantibodies occur against the thyroid antigens: thyroid peroxidase (TPO) and thyroglobulin (Tg). Once the production of thyroid autoantibodies (anti-Tg antibodies and anti-thyroperoxidase antibodies) occurs, antigen-producing B-lymphocytes, T helper cells (CD4+), and cytotoxic T cells (CD+8), and macrophages infiltrate and accumulate in the thyroid gland through the formation, clonal expansion, and maturation of self-reactive T lymphocytes and B-lymphocytes in the lymph tissue of the thyroid gland. This entire process of autoantibody, cytokine-mediated and apoptotic mechanisms of cytotoxicity leads to the destruction of the thyroid gland. 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 Hashimoto's Thyroiditis (Davies, 2015).



Hashimoto's thyroiditis is one of the most common autoimmune diseases in the United States. The name of this disease was derived from the 1912 pathology report completed by Dr. Hakaru Hashimoto (Caturegli et al., 2013). It is estimated that one in 300 persons in the United States has been diagnosed with some form of hypothyroidism and an estimated 13 million persons have hypothyroidism, but have not been properly diagnosed. Hypothyroidism may not be present until later in life and affects women more than men. Often times, hypothyroidism is ignored or misdiagnosed due to the multiple symptoms that appear similar to other diseases (Gaitonde, Rowley, & Sweeney, 2012).

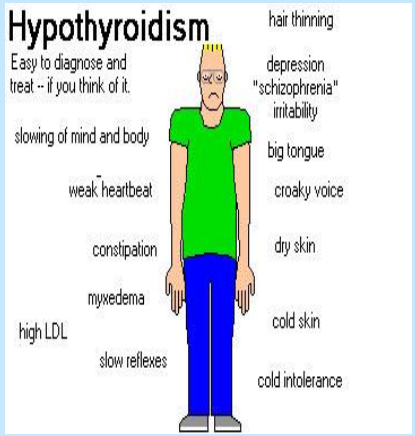
Due to the widespread function of the thyroid gland, an untreated hypothyroidism can lead to multisystem complications including; cardiovascular diseases, pregnancy complications, and even life-threatening conditions. An accurate diagnoses and treatment is critical. Any form of hypothyroidism such as Hashimoto's Thyroiditis is a serious disease and should be treated as such (Mendes, 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, and skin to the digestive tract, heart, and brain. Since the thyroid gland is a multisystem functioning gland, 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 have, the possible signs and symptoms will be the same. Initially, hypothyroidism may present itself in a subclinical manner (mild and without symptoms), but as the disease progresses the symptoms become more apparent (NIH, 2013).

Signs and symptoms that can be seen in someone with a hypothyroid disease are: fatigue, constipation, dry skin, increased sensitivity to cold, muscle weakness, cramps or stiffness, coarse brittle thinning hair, brittle nails, hoarse voice, unintentional weight gain, depression, menstrual irregularities; heavy and excessive bleeding. Physical exam findings that may also be seen are: bradycardia, goiter, diastolic hypertension, cognitive impairment, delayed deep tendon reflexes, edema, altered laboratory results: elevated TSH, decreased Free T4, normal or sometimes decreased Total or Free T3, hyperlipidemia, hyponatremia, and elevated creatine kinase, Hashimoto's specific: Elevated serum concentrations of TPO autoantibodies (Ross, 2015).

In general, these symptoms are due to the decreased production of thyroid hormone. Most of the symptoms are not manifested in the early stages of the disease, but as the disease progresses, the degree of the disease along with clinical manifestations will worsen. When the metabolic rate drops to a critical level, a life threatening emergency known as "Myxedema Coma" can occur. This event is characterized by hypothermia, hypoglycemia, severe bradycardia and altered level of consciousness (Hampton, 2013). Proper diagnoses and treatment is life or death when dealing with hypothyroidism.



Nursing Implications

Since HT and hypothyroidism can present itself in a variety of ways and level of severity, the most important factor is an accurate recognition and diagnosis. Diagnosing should always begin with a thorough physical exam and obtaining a complete medical history. Taking time to interview the patient and truly hear what is being said is crucial. In addition, focusing on one symptom will prevent a healthcare provider from accurately diagnosing someone with HT. The healthcare provider needs to be well educated about hypothyroidism and its presenting symptoms. Again, HT and/or hypothyroidism can easily be mistaken for other medical problems due the thyroid gland being a multisystem gland, which is why it is so important for healthcare providers to be well educated about this disease.

When the physical exam and medical history is complete and hypothyroidism or HT is suspected, diagnostic lab work must be obtained to confirm the diagnosis. Lab work usually begins with a TSH level. Normally, a TSH level above normal is indicative of hypothyroidism. In addition, a T4 level should be obtained and will be lower than normal with hypothyroidism. Although most persons 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 of HT, a person must test positive for serum antithyroid antibodies. This test confirms the presence of autoantibodies that mistakenly attack the thyroid tissue. A computerized tomography (CT) scan may also be performed to assess the thyroid gland for size, texture, nodules, and/or inflammation (NIH, 2013).

Treatment for HT depends upon goiter activity and/or if hypothyroidism is present. If a person with HT does not 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, healthcare providers may treat the person in an effort to decrease the size of the goiter. When a person with HT is positive for hypothyroidism, they must be treated with synthetic T4 (synthetic T4 stays in the body longer than synthetic T3). The most common form of synthetic T4 that is currently being used is Synthroid. Once Synthroid is initiated, healthcare providers must monitor the patient's lab work closely to prevent the counter effect of hyperthyroidism. Usually, lab work is rechecked every 3 months after the initial start of Synthroid and/or if changes are being made to the patient's Synthroid dosage (NIH, 2013). Finally, another very important nursing implications is patient education. It is very important that a patient understands the disease, the importance of the treatment, and most importantly, compliance with taking medication as prescribed. Noncompliance is not an option with HT. Patient compliance is important in preventing extreme hypothyroidism such as Myxedema Coma.

Conclusion

In conclusion, HT is characterized by the loss of thyroid follicular cells, hypothyroidism, and the presence of autoantibodies against tissue-specific antigens such as thyroid peroxidase (TPO) and thyroglobulin (Tg). The thyroid tissue is identified as having an infiltration of T cells and B 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 thyrocytes with the end result of hypothyroidism and Hashimoto's Thyroiditis (Glick, Wodzinski, Fu, Levine, & Wald, 2013).

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