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AUTOIMMUNE HYPOTHYROIDISM

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Annotation: Autoimmune hypothyroidism may be associated with a goiter (Hashimoto's, or goitrous thyroiditis) or, at the later stages of the disease, minimal residual thyroid tissue (atrophic thyroiditis). Because the autoimmune process gradually reduces thyroid function, there is a phase of compensation when normal thyroid hormone levels are maintained by a rise in TSH. Though some patients may have minor symptoms, this state is called subclinical hypothyroidism. Later, unbound T4 levels fall and TSH levels rise further; symptoms become more readily apparent at this stage (usually TSH >10 mIU/L), which is referred to as clinical hypothyroidism or overt hypothyroidism.

Keywords: thyroidit, thyroxine, triiodothyronine, myxedema, Hashimoto's encephalopathy.

The thyroid is a small, butterfly-shaped gland located in the front of the neck that plays a critical role in regulating metabolism, energy levels, and overall hormonal balance.
In autoimmune hypothyroidism, the immune system produces antibodies that target thyroid cells, leading to inflammation and damage to the gland. Over time, this impairs the thyroid's ability to produce enough thyroid hormones, which are essential for various bodily functions.

The mean annual incidence rate of autoimmune hypothyroidism is up to 4 per 1000 women and 1 per 1000 men. It is more common in certain populations, such as the Japanese, probably because of genetic factors and chronic exposure to a high-iodine diet.

ETIOLOGY AND PATHOGENESIS:

In Hashimoto's thyroiditis, there is a marked lymphocytic infiltration of the thyroid with germinal center formation, atrophy of the thyroid follicles accompanied by oxyphil metaplasia, absence of colloid, and mild to moderate fibrosis. In atrophic thyroiditis, the fibrosis is much more extensive, lymphocyte infiltration is less pronounced, and thyroid follicles are almost completely absent. Atrophic thyroiditis likely represents the end stage of Hashimoto's thyroiditis rather than a distinct disorder.

As with most autoimmune disorders, susceptibility to autoimmune hypothyroidism is determined by a combination of genetic and environmental factors, and the risk of either autoimmune hypothyroidism or Graves' disease is increased among siblings. HLA-DR polymorphisms are the best documented genetic risk factors for autoimmune hypothyroidism especially HLA-DR3, -DR4, and -DR5 in Caucasians. A weak association also exists between polymorphisms in CTLA-4, a T cell–regulatory gene, and autoimmune hypothyroidism. Both of these genetic associations are shared by other autoimmune diseases, which may explain the relationshipbetween autoimmune hypothyroidism and other autoimmune diseases, especially type 1 diabetes mellitus, Addison's disease, pernicious anemia, and vitiligo HLA-DR and CTLA-4 polymorphisms account for approximately half of the genetic susceptibility to autoimmune hypothyroidism. Other contributory loci remain to be identified. A gene on chromosome 21 may be

responsible for the association between autoimmune hypothyroidism and Down syndrome. The female preponderance of thyroid autoimmunity is most likely due to sex steroid effects on the immune response, but an X chromosome–related genetic factor is also possible and may account for the high frequency of autoimmune hypothyroidism in Turner's syndrome. Environmental susceptibility factors are poorly defined at present. A high iodine intake may increase the risk of autoimmune hypothyroidism by immunologic effects or direct thyroid toxicity. There is no convincing evidence for a role of infection except for the congenital rubella syndrome, in which there is a high frequency of autoimmune hypothyroidism. Viral thyroiditis does not induce subsequent autoimmune thyroid disease.The thyroid lymphocytic infiltrate in autoimmune hypothyroidism is composed of activated CD4+ and CD8+ T cells as well as B cells. Thyroid cell destruction is primarily mediated by the CD8+ cytotoxic T cells, which destroy their targets by either perforin-induced cell necrosis or granzyme B–induced apoptosis. In addition, local T-cell production of cytokines, such as tumor necrosis factor (TNF), IL-1, and interferon γ (IFN-γ), may render thyroid cells more susceptible to apoptosis mediated by death receptors, such as Fas, which are activated by their respective ligands on T cells. These cytokines also impair thyroid cell function directly and induce the expression of other proinflammatory molecules by the thyroid cells themselves, such as cytokines, HLA class I and class II molecules, adhesion molecules, CD40, and nitric oxide. Administration of high concentrations of cytokines for therapeutic purposes (especially IFN-α) is associated with increased autoimmune thyroid disease, possibly through mechanisms similar to those in sporadic disease.Antibodies to TPO and Tg are clinically useful markers of thyroid autoimmunity, but any pathogenic effect is restricted to a secondary role in amplifying an ongoing autoimmune response. TPO antibodies fix complement, and complement membrane-attack complexes are present in the thyroid in autoimmune hypothyroidism. However, transplacental passage of Tg or TPO antibodies has no effect on the fetal thyroid, which suggests that T cell–mediated injury is required to initiate autoimmune damage to the thyroid.Up to 20% of patients with autoimmune hypothyroidism have antibodies against the TSH-R, which, in contrast to TSI, do not stimulate the receptor but prevent the binding of TSH. These TSH-R–blocking antibodies, therefore, cause hypothyroidism and, especially in Asian patients, thyroid atrophy. Their transplacental passage may induce transient neonatal hypothyroidism. Rarely, patients have a mixture of TSI- and TSH-R–blocking antibodies, and thyroid function can oscillate between hyperthyroidism and hypothyroidism as one or the other antibody becomes dominant. Predicting the course of disease in such individuals is difficult, and they require close monitoring of thyroid function. Bioassays can be used to document that TSH-R–blocking antibodies reduce the cyclic AMP–inducing effect of TSH on cultured TSH-R–expressing cells, but these assays are difficult to perform. TBII assays that measure the binding of antibodies to the receptor by competition with radiolabeled TSH do not distinguish between TSI- and TSH-R–blocking antibodies, but a positive result in a patient with spontaneous hypothyroidism is strong evidence for the presence of blocking antibodies. The use of these assays does not generally alter clinical management, although it may be useful to confirm the cause of transient neonatal hypothyroidism.

CLINICAL PICTURE:

The clinical picture of autoimmune hypothyroidism (Hashimoto's thyroiditis) develops gradually and varies based on the severity and duration of the disease. It is often characterized by a slowing down of the body's metabolic processes due to a decrease in thyroid hormone levels.

Early Stages

Initially, many patients may be asymptomatic or have mild symptoms, but as the thyroid gland becomes progressively damaged, more symptoms appear.

Common Symptoms and Signs:

1. Fatigue and Weakness:

Profound tiredness, even after adequate rest.

Muscle weakness, particularly in the legs.

2. Weight Gain:

Gradual weight gain despite no significant changes in diet or exercise.

Difficulty losing weight due to a slowed metabolism.

3. Cold Intolerance:

Increased sensitivity to cold temperatures.

Feeling cold even in warm environments.

4. Dry Skin and Hair:

Dry, coarse, and pale skin.

Brittle hair that may become thin or fall out.

Hair loss from the outer edge of the eyebrows.

5. Constipation:

Persistent or severe constipation due to reduced digestive activity.

6. Mental Health Changes:

Depression, irritability, or mood swings.

Impaired memory and concentration ("brain fog").

Slowed speech and thinking.

7. Bradycardia (Slow Heart Rate):

Heart rate may slow down due to decreased metabolism.

Mild to moderate bradycardia.

8. Myxedema:

Swelling of the face, especially around the eyes.

Thickened skin with a puffy appearance, especially in the extremities.

9. Goiter (Thyroid Enlargement):

A visibly enlarged thyroid gland in the neck (goiter) due to inflammation.

The goiter can cause a sensation of fullness in the neck, difficulty swallowing, or mild discomfort.

10. Hoarseness:

The voice may become hoarse due to the effects of thyroid inflammation and swelling.

11. Menstrual Irregularities:

Heavy or prolonged menstrual periods (menorrhagia).

Irregular menstrual cycles.

In severe cases, infertility may be a concern.

12. Slowed Reflexes:

Delayed relaxation phase of deep tendon reflexes (e.g., Achilles reflex).

LABORATORY FINDINGS:

High TSH (thyroid-stimulating hormone): The pituitary gland produces more TSH in an attempt to stimulate the failing thyroid.

Low Free T4 (thyroid hormone): A hallmark of hypothyroidism.

Positive **anti-TPO antibodies**: Indicative of autoimmune thyroiditis.

CONCLUSION: Autoimmune hypothyroidism is a condition in which the immune system mistakenly attacks the thyroid gland, leading to decreased production of thyroid hormones (hypothyroidism).

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