AUTOIMMUNE THYROIDITIS

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Abstract. *Autoimmune thyroiditis is an autoimmune disease that affects the thyroid gland. Autoimmune thyroiditis is ten times more common in women than in men. Symptoms usually first appear between the ages of 30 and 50. The overall incidence increases with age in both men and women. Autoimmune thyroiditis is characterized by antibodies to thyroid antigens. The thyroid gland is damaged and gradually grows. Over time, autoimmune thyroiditis leads to hypothyroidism, in which the body lacks thyroid hormones. Hypothyroidism is accompanied by weight gain, dry skin, and increased fatigue.*

Key words: *thyroglobulin, hypothyroidism, euthyroid, hypothyroid, hyperthyroid, Ashkenazi-Gurtel cells.*

АУТОИММУННЫЙ ТИРЕОИДИТ

Аннотация. *Аутоиммунный тиреоидит — это аутоиммунное заболевание, поражающее щитовидную железу. Аутоиммунный тиреоидит встречается в десять раз чаще у женщин, чем у мужчин. Симптомы обычно впервые появляются в возрасте от 30 до 50 лет. Общая заболеваемость увеличивается с возрастом как у мужчин, так и у женщин. Аутоиммунный тиреоидит характеризуется антителами к антигенам щитовидной железы. Щитовидная железа повреждается и постепенно увеличивается. Со временем аутоиммунный тиреоидит приводит к гипотиреозу, при котором организму не хватает гормонов щитовидной железы. Гипотиреоз сопровождается увеличением веса, сухостью кожи и повышенной утомляемостью.*

Ключевые слова: *тиреоглобулин, гипотиреоз, эутиреоид, гипотиреоз, гипертиреоз, клетки Ашкенази-Гюртеля.*

AUTOIMMUNE THYROIDITIS (HASHIMOTO'S DISEASE) is a chronic inflammatory disease of the thyroid gland of autoimmune origin. The disease was first described by Hashimoto in 1912.

The prevalence is 0.1-1.2% in children, 6-11% in women over 60 years of age. The incidence of clinically expressed forms of the disease is 1%. In healthy people in a euthyroid state, subclinical thyroiditis and circulating antibodies in the blood are detected in 10-15%.

ETIOLOGY: 3 groups of etiological factors are distinguished: external, internal and the occurrence of AIT as a "secondary" disease in other diseases.

Internal etiological factors: these include hereditary and etiological factors. Heredity is confirmed by the presence of several people in the family, the simultaneous occurrence of the disease in twins (30-60% in monozygotic, 3-9% in dizygotic), and the presence of other autoimmune diseases (chronic active hepatitis, B12-deficient anemia, type 1 diabetes mellitus, etc.) in one fetus. The genetic marker of AIT is the detection of the HLA system. The hypertrophic form of AIT is often accompanied by HLA DR3, rarely by HLA DR3. The risk of congenital development of AIT is associated with HLA DQW7. Internal provoking factors include disruption of immune and endocrine homeostasis during puberty, menopause, pregnancy, childbirth, and old age.Ташқи этиологик омилларга қуйидагилар киради:

1. Environmental pollution by industrial waste can negatively affect human immune homeostasis and lead to the development of AIT.

2. The use of toxic chemicals in agriculture has an immunotropic effect, and in people who work with them a lot, they enter the body with air and food, causing the development of AIT.

3. Treatment with lithium drugs. Lithium is a thyroid antigen hapten, which enhances the production of antithyroid antibodies and leads to the development of AIT.

4. Long-term excessive iodine intake in genetically predisposed individuals. This occurs as a result of taking high doses of iodine. High doses of iodine are found in amiodarone (cordarone), radiocontrast agents, and a number of antiseptics. Taking a daily dose of iodine does not lead to the development of AIT.

5. The effect of low-dose ionizing radiation.

6. Viral, bacterial and yersiniosis infections.

7. Interferon therapy promotes the expression of HLA-II class molecules in thyrocytes and leads to the development of AIT.

AIT occurs as a “secondary disease” with other diseases of the thyroid gland. These are diffuse toxic goiter, endemic and sporadic goiter, thyroid adenoma and tumor.

PATHOGENESIS:

Autoimmune thyroiditis is the result of a combination of genetic and environmental factors.

The disease is based on a deficiency of the T-suppressor function of lymphocytes, thyroid antigens (thyroglobulin, colloid component and microsomal antigen). When the T-suppressor function of lymphocytes decreases, T-lymphocyte helpers are activated and the production of antibodies to thyroid antigens increases. HLA-DR3 enhances the helper function of T-lymphocytes. When the T-suppressor function decreases, "forbidden" clones of T-lymphocytes appear, which are organ-specific for the thyroid gland. These clones interact with the thyroid gland, causing cytotoxic damage, its antigens are secreted into the blood and antibodies are produced against them. The following types of antibodies are distinguished in AIT:

-antibody to thyroglobulin;

-antibody to the microsomal fraction of the follicular epithelium;

-cytotoxic antibody inhibiting peroxidase activity;

-antibody to thyrotropin hormone receptors;

-growth-stimulating antibody;

-antibody to the colloidal component.

As a result of the interaction of circulating thyroglobulin and antibodies to the microsomal fraction with T-lymphocyte killers, lymphokines (lymphotoxin, chemotaxis factor, tumor necrosis factor) are released, which have a cytotoxic effect, cause inflammation, and damage thyrocytes.

When the process of autoaggression continues for a long time, the function of the thyroid gland decreases and, based on the principle of feedback, the production of thyrotropic hormone increases. This process and growth-stimulating antibody lead to an increase in the size of the thyroid gland (hypertrophic form). However, as a result of the long-term cytotoxic effect of T-lymphocyte killers and antibodies, thyrocytes are damaged, the thyroid gland shrinks in size, fibrosis develops, and hypothyroidism (atrophic form) occurs.

The following changes are observed in histological AIT:

-diffuse (sometimes focal) infiltration of the thyroid gland with lymphocytes and plasma cells;

-destruction of follicles and their basement membrane;

-appearance of large epithelial oxyphilic Ashkenazi cells (cells that degenerate the thyroid epithelium);

-foci of fibrosis are also found along with lymphoid infiltration.

CLASSIFICATION:

I. According to the nosological sign:

-AIT occurs as an independent disease;

-AIT is combined with other thyroid pathologies;

-AIT is a syndrome in other general autoimmune diseases;

-postpartum AIT.

II. Depending on the form:

-hypertrophic (including nodular);

-atrophic.

III. Depending on the functional activity of the thyroid gland:

-euthyroid;

-hypothyroid;

-hyperthyroid.

IV. Depending on the clinical course:

-clinically expressed;

-latent.

V. Depending on the spread of the autoimmune process in the thyroid gland:

-focal;

-diffuse.

CLINICAL:

The disease is 4-7 times more common in women than in men. It can occur at any age, but is most common after the age of 60.

Hypertrophic form - in this form of AIT, cytostimulating antibodies are produced, which increase the growth and size of the thyroid gland, leading to an increase in its function. This form develops gradually, can begin in childhood, and then manifests itself in puberty and old age.

The main complaints of patients:

-enlargement of the thyroid gland;

-difficulty swallowing;

-weakness;

- a feeling of pressure in the neck.

When viewed objectively: the thyroid gland is diffusely enlarged, hard, elastic consistency, not connected to the skin. Later, the hardness of the gland increases, it becomes rough, the symptom of "fluttering" (that is, when one part is palpated, the other part shakes) appears. Pain may be observed when subacute thyroiditis is added. In 5% of patients with a hypertrophic form, an increase in the function of the thyroid gland is observed, and the clinic of thyrotoxicosis occurs and is called "khasi-toxicosis" ("hashi-toxicosis"). In this case, patients complain of rapid heartbeat, feeling of heat, sweating, weight loss, agitation.

Specific features of Khasi-toxicosis:

-it goes wavelike, that is, the patient's condition alternates between improvement and deterioration;

- thyrotoxicosis responds quickly to treatment compared to diffuse-toxic goiter;

-ophthalmopathy usually begins at the beginning of the disease;

- relapses of hyperthyroidism are acute respiratory infections, mental and physical stress, pregnancy, childbirth and abortion.

Later, the hypertrophic form gradually leads to the development of hypothyroidism. It is characterized by an increase in body weight, edema, dryness and flaking of the skin, constipation, bradycardia, memory loss, hair loss, impaired sexual function, anemia, increased TSH and decreased T3, T4. The hypertrophic form is often accompanied by HLA B8 and DR5.

Atrophic form

In this form, the thyroid gland is not palpable, hypothyroidism is observed, this condition was previously called "idiopathic hypothyroidism". This form develops gradually over the years and is diagnosed after the onset of hypothyroidism. This form is characterized by the HLA-DR3 antibody marker, and galactorrhea-amenorrhea may develop due to increased levels of thyrotropin.

Many patients have arterial hypertension.

Focal (focal) form

This form is characterized by damage to one lobe (one lobe is small, hard). Puncture biopsy confirms autoimmune thyroiditis.

Latent form

In this form, there are no clinical signs, the thyroid gland is normal in size, only immunological indicators can confirm the disease. The latent form is often accompanied by nodular goiter.

In all forms of AIT, depending on the functional state of the thyroid gland, there can be: euthyroidism, hyperthyroidism (rarely) and hypothyroidism.

Risk groups for the development of AIT:

1. Patients with diffuse toxic goiter;
2. Patients who have undergone thyroid surgery;
3. Patients with endemic goiter;
4. Patients with galactorrhea-amenorrhea;
5. Patients with diabetes mellitus;
6. Patients with Stein-Leventhal syndrome (sclerocystic ovary syndrome);
7. Patients with allergic and autoimmune diseases;
8. Women aged 40 years and older;
9. Relatives of patients with AIT, DTZ and other autoimmune diseases.

Laboratory and instrumental examination methods

1. Complete blood count: lymphocytosis and ESR may be increased;
2. Biochemical analysis of blood: increased levels of cholesterol, lipoproteins, triglycerides (with the development of hypothyroidism);
3. Immunological examination of blood: decreased number and function of T-lymphocytes, increased levels of immunoglobulins;
4. Ultrasound of the thyroid gland: uneven structure, hypoechoic areas or unencapsulated nodes are visible. The cardinal sign of AIT is a decrease in diffuse echogenicity of the gland tissue.

Ultrasound cannot distinguish AIT and DTB, since a decrease in echogenicity is also observed in DTB. In the hypertrophic form of AIT, ultrasound reveals an increase in the size of the gland, and in the atrophic form, a decrease.

5. Under the control of ultrasound, a percutaneous aspiration fine-needle biopsy of the thyroid gland is performed; several areas and nodes of the gland are punctured. The biopsy reveals plasma cell and lymphoid infiltration, the ratio of small and large nuclear lymphocytes is less than 4.5 microns (normally greater than 7 microns); Ashkenazi-Gürtel (large epithelial oxyphilic cells) oxyphilic cells are detected.

6. Radioisotope scanning of the thyroid gland with radioactive iodine or technetium. This method determines the increase in the size of the gland (in hypertrophic form) or decrease (in atrophic form), the unevenness of the contour, the change in shape (in the norm it is like a "butterfly" shape, but in AIT it resembles a droplet shape), the presence of radiopharmaceutical absorption, the absence of intense absorption in the center.

7. Determination of antithyroid antibodies in the blood. Titers of diagnostic significance: 1:100 and more for thyroglobulin antibodies (detected in 70% of cases), 1:32 and more for the follicular epithelial microsomal fraction (detected in 95% of cases).

8. Radioimmunoassay of hormonal status. In the stage of hyperthyroidism, the amount of T3, T4 in the blood is increased, and the amount of TSH is decreased; in the stage of hypothyroidism, the amount of TSH is increased, and the amount of T3, T4 is decreased; in the case of euthyroidism, the amount of T3, T4 and TTG in the blood is normal.

9. Determining the amount of prolactin in the blood. The amount of prolactin will be increased.

DIFFERENTIAL DIAGNOSIS:

1. With nodular euthyroid goiter - in this case, antithyroid antibodies are not found in the blood; lymph and plasma cell infiltration, Ashkenaz cells are not found in the thyroid punctate.

2. With thyroid tumor - in this case, the node is poorly mobile or immobile, connected to the surrounding tissues, regional lymphadenopathy, undifferentiated cells are found during the puncture of the node with signs of proliferation.

3. With diffuse toxic goiter - in AIT, the signs of thyrotoxicosis are less pronounced than in DTZ, even without the use of thyrostatics, thyrotoxicosis does not progress, and may even spontaneously transition to a euthyroid state, the titer of antithyroid antibodies is high (in DTB, antibodies to thyroglobulin and microsomal fraction are rarely and in low titers).

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