

AUTOIMMUNE THYROIDITIS

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Abstract: in this article we describe autoimmune thyroiditis is an inflammatory disease of the thyroid gland caused by the attack of specific proteins on the functional cells of the body. Pathology is genetic in nature. Symptoms of autoimmune thyroiditis increase as the function of the gland is suppressed, but the first signs of pathology are often confused with manifestations of other somatic diseases. I start drug therapy when the patient's hormonal status changes. Autoimmune thyroiditis is a chronic disease.

Keywords: Autoimmune thyroiditis, manifestations, diagnosis, Hypothyroidism.

This is inflammation that occurs from damage or destruction of thyroid follicles by cells of the immune system (T-lymphocytes). At the same time, infiltrates appear in the gland, and the activity of the gland and the amount of thyroid hormones in the body decrease. Hypothyroidism develops slowly.

The disease is diagnosed in 3% of all thyroid pathologies. Most of the patients are women. In them, this diagnosis occurs 10-20 times more often than in men. There is also a dependence on age - Hashimoto's thyroiditis rarely occurs in people under thirty years of age. Most patients are middle-aged women (30-50 years). Inflammation occurs as a result of a genetic mutation that changes the mechanisms of interaction between killer cells and suppressor lymphocytes. Increased cellular aggression against thyroid tissue. The body's immune system recognizes its own thyroid antibodies and begins the synthesis of specific proteins against functional structures and hormone receptors. As a result of the attack, inflammation begins, thyroid cells are replaced by connective tissue. Hormone-producing function is reduced. In addition to thyroiditis, patients with a genetic predisposition may develop goiter, rheumatoid arthritis, vitiligo, and other diseases.

For the first time, the symptoms and causes of autoimmune thyroiditis of the thyroid gland were described by the Japanese doctor Hashimoto, so the disease was named after him. The prevalence of pathology is 3-4% of the population. Women get sick more often than men.

Causes of autoimmune thyroiditis

Every 10th inhabitant of the planet has antibodies to thyroid agents in their blood. But not all cases develop autoimmune Hashimoto's thyroiditis. Pathological processes occur in the presence of provoking factors:

- mechanical injuries of the thyroid gland;
- frequent stress;
- poor environmental conditions in the area of residence;
- acute bacterial and viral infections, etc.

One of the causes of autoimmune thyroiditis is a weakened immune system. The disease is more susceptible to adolescents, women in the postpartum period and perimenopause.

Classification of autoimmune thyroiditis

According to the nature of disorders and morphology of changes in the tissues of the thyroid gland, several clinical forms of the disease are distinguished:

- Hypertrophic - the tissues of the endocrine organ grow. The gland significantly increases in size, lymphocytes (cells of the immune system) accumulate in the intercellular space. the hypertrophic form of autoimmune thyroiditis is characteristic of the early stages of the disease;
- atrophic - gland tissue is replaced by fibrous formations. this form develops when most of the follicles die.

Phases and symptoms of autoimmune thyroiditis of the thyroid gland:

- euthyroid phase. The hormonal status of the patient is normal. Symptoms of autoimmune thyroiditis of the thyroid gland are absent. The euthyroid phase can last for many years without clinical manifestations;
- subclinical hypothyroidism. The level of thyroid hormones begins to change, TSH is synthesized in large quantities. For the patient, the processes proceed imperceptibly. Changes can be noticed only by blood tests;
- obvious hypothyroidism. The level of T3 and T4 is greatly reduced. The thyroid gland begins to grow, characteristic clinical symptoms of hypothyroidism appear.

There are destructive variants of autoimmune thyroiditis:

- postpartum. the first signs of the disease appear about 2 weeks after birth. the immunity of a woman, suppressed during pregnancy, rapidly increases (the "rebound" phenomenon). increased activity of autoantibodies that destroy thyroid follicles. thyroid hormones enter the bloodstream, causing a state of hyperthyroidism, which then turns into hypothyroidism. there are symptoms of autoimmune thyroiditis in women;
- cytokine-reduced. thyroiditis occurs against the background of taking interferons in the treatment of hepatitis c and other blood diseases;
- painless ("silent"). the causes of autoimmune thyroiditis of the thyroid gland have not been elucidated.
- destructive forms of the disease last up to 1 year and are amenable to conservative treatment. adequate therapy ends with the clinical recovery of the patient. the function of the gland is restored regardless of the cause of its violation.

Symptoms of the disease. Symptoms of autoimmune thyroiditis of the thyroid gland are associated with a violation of the functions of the organ: increased or insufficient production of hormones.

Signs of a decrease in the level of T3 and T4:

- depressed state, depression;
- weight gain;
- swelling of the face, eyelids;
- cold intolerance;
- constipation;

- dry hair and skin;
- decreased libido;
- bradycardia;
- increased blood pressure;
- menstrual irregularities, miscarriages, reduced fertility (characteristic symptoms of autoimmune thyroiditis in women).

Destructive forms of the disease begin with manifestations of thyrotoxicosis:

- weight loss;
- increased sweating;
- nervousness, aggression;
- stool disorders;
- tachycardia, etc.

Protrusion of the eyeballs with thyrotoxicosis does not have time to develop. The level of hormones gradually decreases, symptoms of hypothyroidism appear.

Diagnosis of autoimmune thyroiditis. The disease has no characteristic symptoms, therefore, for differential diagnosis, the patient must undergo a comprehensive examination. First of all appointment with an endocrinologist. The doctor collects an anamnesis, examines the patient, determines the presence or absence of palpable changes in the structure of the thyroid gland.

Laboratory blood tests for autoimmune thyroiditis:

- antibodies to thyroperoxidase (AT-TPO);
- antibodies to thyroid-stimulating hormone receptors (AT-rTTH);
- TSH level, T3, T4.

Ultrasonography. Decreased echo signal from glandular tissue is detected; Scintigraphy. The procedure is prescribed for the differential diagnosis of autoimmune thyroiditis, accompanied by thyrotoxicosis, from other diseases with similar symptoms. The affected thyroid gland does not accumulate contrast or retains it in small quantities; Fine needle biopsy (FNA). Morphological examination of the material reveals plasma cells and lymphocytes in tissues. Hürthle-Ashkenazi cells are characteristic of autoimmune thyroiditis. Differential diagnosis of the chronic and destructive forms of the disease is carried out against the background of hormone replacement therapy. The patient takes drugs for a year until the condition stabilizes. Then the treatment is canceled and the hormonal background of the patient is controlled. If there are no changes in the biochemical parameters of the blood, then the function of the thyroid gland is fully restored, there is no chronization.

Treatment of autoimmune thyroiditis.

Autoimmune pathologies without thyroid dysfunction do not require treatment. The patient should regularly be examined by an endocrinologist, monitor his well-being. It is recommended to spend more time outdoors, lead a healthy lifestyle, adhere to proper nutrition. Conservative treatment is prescribed when signs of hypothyroidism appear. The doctor calculates the dosage of hormonal drugs and prescribes drugs for long-term use. On the background of therapy, the patient's condition

improves significantly. In some cases, drugs are taken for life, which allows stopping fibrous and inflammatory processes, maintaining the quality of life at the same level. Additional intake of potassium iodide is recommended only for residents of endemic areas. Patients in other regions do not need such therapy, since autoimmune thyroiditis is not associated with iodine deficiency. An excess amount of trace elements can, on the contrary, aggravate the course of the disease. As the follicles are destroyed, the functionality of the organ decreases, which, in turn, provokes the development of multiple complications in the body. Autoimmune thyroiditis is diagnosed annually in 3-10% of the population worldwide. Women get sick much more often than the representatives of the stronger half (the incidence of pathology is ten times higher than in men). Among all gland pathologies, Hashimoto's goiter accounts for 20–30% of cases. The highest risk age is 40–70 years. If in the 60s of the last century the disease occurred in isolated cases, today it is one of the main pathologies of the thyroid gland. Doctor Hashimoto, conducting histological studies in his patients, revealed several changes in the structure of the organ: extensive diffuse plasmacytic infiltration with the formation of multiple centers of cell division; fading of the functions of thyroid follicles, caused by the growth of connective tissue; replacement of differentiated tissue cells with other differentiated cells (metaplasia).

Why such failures occur in the body, scientists cannot explain, but doctors have identified several reasons that contribute to the development of the pathological process. Chronic autoimmune thyroiditis (Hashimoto's thyroiditis) is a non-infectious inflammatory disease of the thyroid gland, in which the immune system is aggressive towards its cells. Autoimmune thyroiditis is a common type of thyroiditis that leads to a decrease in the functional capacity of the thyroid gland (primary hypothyroidism). The lack of thyroid hormones negatively affects the general state of health. It is characterized by symptoms of hypothyroidism and a painless increase in the size of the gland. The causes of development are often a genetic predisposition and concomitant autoimmune diseases. This pathology is characterized by the production of antibodies to its own cells by the immune system. Antibodies destroy the tissues of the gland, reducing its functional ability, causing an inflammatory process. The disease has a chronic course.

Thyroid hormones have a strong influence on the entire body - they are responsible for metabolism, growth, development, synthesis of certain hormones, etc. Their deficiency or excess can lead to serious organ diseases. AIT is several times more common in women than in men. The prevalence increases with age. The average age of patients is from 40 to 50 years, but younger people are increasingly getting sick. Thyroiditis is often found in individuals with chromosomal mutations - Down's syndrome, Turner's syndrome, Klinefelter's syndrome. Often, thyroid pathologies are found in family members of the patient. Chronic thyroiditis is sometimes associated with the presence of another autoimmune disease (Addison's disease, type 1 diabetes mellitus, hypoparathyroidism, pernicious anemia, rheumatoid arthritis, systemic lupus erythematosus, Schmidt's syndrome). The main complication of autoimmune thyroiditis is hypothyroidism. And hypothyroidism can lead to the development of myxedema - edema of the whole body with dysfunction of organs and systems. The disease occurs in two forms: atrophic (the size does not change) and hypertrophic (a goiter appears - a painless increase in the size and density of the thyroid gland). Both forms can occur with subclinical or overt hypothyroidism. The course is chronic, slowly progressive and usually leads to sustained hypothyroidism. Very rarely,

exacerbations occur with a sudden increase in size and palpation of the thyroid gland, as well as systemic manifestations of inflammation.

Any folk recipes to improve the condition of the thyroid gland must be agreed with the doctor. In some cases, homeopathic remedies are approved as an addition to complex treatment. The prognosis for autoimmune thyroiditis is favorable. Hormone replacement therapy in adequate dosages can prevent complications. Autoimmune thyroiditis of the thyroid gland is a serious disease that has an autoimmune nature of development. The disease is of hereditary origin and is associated with the synthesis of antibodies that are foreign to the thyroid tissue. These cells, thyrocytes, damage the gland. The disease is more common among the female half and is quite common in endocrinology. In pathology, there is a constant inflammation of the thyroid tissue, with the destruction of the follicles and follicular cells of the thyroid gland. As practice shows, the disease does not have symptoms in patients. It is possible to identify the disease thanks to analyzes, ultrasound of the thyroid gland, the results of histology during a biopsy of the material. Therapy consists in adjusting the functioning of the gland and stopping autoimmune disorders. Clinical signs of autoimmune thyroiditis usually begin with a painless enlargement of the thyroid gland or a feeling of fullness in the neck. The clinical picture of hypothyroidism in the euthyroid and subclinical phases is absent. In some, relatively rare cases, an increase in the volume of the gland occurs in the euthyroid phase, which rarely reaches significant degrees. The patient may complain of a "lump in the throat". Over time, inflammation affects the tissues of the gland, which leads to hypofunction. In people with hyperthyroidism, tachycardia, nervousness, and heat intolerance are observed at the initial stage. Normally, hormones increase the production of heat from brown fat. With hypothyroidism, the processes are inhibited, the patient experiences a constant feeling of cold. Pathology forces the body not to spend calories on energy processes, but to store them in the form of adipose tissue. As a result, the patient feels constant weakness, and body weight grows even with a small amount of food eaten. As a result of changes in metabolic processes, the body begins to destroy muscle tissue for energy, so the patient complains of constant fatigue. Hair follicles contain many stem cells responsible for tissue regeneration, with hypothyroidism, regeneration processes slow down. Deficiency of thyroid hormones can be manifested by sleep disorders, decreased intelligence, headaches due to increased intracranial pressure. Often, patients go to the doctor with clinical manifestations of osteochondrosis of the thoracic or cervical spine: weakness and muscle pain in the hands, tingling, burning in the fingers. As a result of the examination, it turns out that the symptoms were caused by hypothyroidism. Dysfunction of the thyroid gland is often "disguised" as a violation of the cardiovascular system, there may be hypertension, arrhythmia, bradycardia.

At the initial stages of the destruction of the gland, symptoms of hyperthyroidism can be observed: increased sweating, weight loss with increased appetite, tachycardia, chills, nervousness. But the clinic of hyperthyroidism is quickly replaced by symptoms of hypothyroidism. With AIT, pregnancy is not contraindicated with normal thyroid function and compensated hypothyroidism. If hypothyroidism has not yet developed, AIT, as a rule, does not have severe symptoms, the disease goes unnoticed. Often, pathology is detected by chance, during preventive examinations.

Causes. Autoimmune thyroiditis is characterized by hereditary predisposition. The disease is much more common in women than in men. An autoimmune reaction is the production of antibodies against proteins in the body. The disease develops against the background of a genetically determined defect in the immune response, leading to T-lymphocyte aggression against one's own thyrocytes, ending in their destruction. Histologically, lymphocytic and plasmacytic infiltration, oncocyctic transformation of thyrocytes (formation of Hürthle-Ashkenazi cells), destruction of follicles are determined. The reason, most often, is a genetic predisposition to such reactions or the encounter of the body with viruses, the proteins of which are similar to the proteins of thyroid tissues. Often, the development of the disease is associated with hormonal changes or severe psycho-emotional stress.

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