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MODERN METHODS OF DIAGNOSIS OF AUTOIMMUNE THYROIDITIS

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Autoimmune thyroiditis is a group of organ-specific autoimmune thyroidopathies caused by a genetically determined defect in immune tolerance to thyroid antigens, resulting in autoimmune damage to the thyroid gland [22,23,24].

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Antibodies produced by the immune system begin to mistake thyroid cells for foreign cells. By acting on hormone-active thyroid cells, the antibodies cause destructive changes in thyrocytes. As a consequence, thyroid function decreases and thyroid hormone production decreases, which leads to increased synthesis of thyroid hormone (TTH) and the development of hypothyroidism. Against the background of autoimmune thyroiditis is also possible and temporary increase in hormone production - hyperthyroidism (thyrotoxicosis). Autoimmune thyroiditis is considered a hereditary disease, which is confirmed by the data on frequent cases of the disease in close relatives [1,2,3].

Patients with autoimmune thyroiditis often suffer from other autoimmune diseases of somatic and endocrine genesis: diffuse toxic goiter, myasthenia gravis, infiltrative

(autoimmune) ophthalmopathy, collagenosis, lymphoid cell hypophysitis, Sjögren's syndrome, alopecia, vitiligo [22,23,24].

The occurrence of the disease may be preceded by any effects that lead to disruption of the integrity of the structure of the thyroid gland and penetration of thyroid antigens into the bloodstream (various infectious diseases, inflammatory processes, less often trauma to the thyroid gland or thyroid surgery. Factors that can provoke autoimmune thyroiditis can also be: environmental degradation; iodine deficiency or excess; radioactive contamination and so on. It has been found that the incidence of autoimmune thyroiditis is significantly higher in regions with high selenium deficiency in soils. AIT occurs in 3-4% of the world's population. AIT is 10-15 times more often registered in female individuals. Peaks of morbidity are observed in puberty, after pregnancy, childbirth, abortion, at the age of over 35 years, in perimenopause and postmenopause [19,20,21].

The prevalence of the disease in children is 0.1-1.2%. Family history of AIT occurs in 25-30% of cases.

Clinical manifestations are diverse: from asymptomatic forms to obvious clinical manifestations - depending on the functional state of the thyroid gland. Complaints of discomfort, a feeling of ‘pushing’ in the area of the anterior surface of the neck, ‘hoop feeling’, are quite common. There is no parallelism between the severity of the disease, the size of the goitre and the severity of these sensations [16,17,18].

The most important laboratory criterion of AIT is the presence of diagnostic concentrations of autoantibodies to thyroid peroxidase (AT to TPO), which are detected in 90-100% of cases. The level of AT to TPO closely correlates with the degree of lymphoplasmocytic infiltration of the thyroid gland and indirectly indicates the intensity of the autoimmune process in this disease. Less specific are antibodies to thyroglobulin (AT to TH), which are detected in 80-100% of cases. Antibodies to thyroid hormone receptor (AT to pTH) are detected in 20-36% of AIT patients. They can be stimulating and blocking. Thyroid function in this case depends on the balance of different types of antibodies. Thyroid-stimulating antibodies stimulate the synthesis of thyroglobulin and thyroid hormones, but do not significantly affect thyroid growth. Blocking antibodies, binding to the TTG

receptor, block thyroid hormone stimulation of growth and functional activity of thyrocytes [13,14,15].

Indications for determination in AIT patients: - AT to TPO:

1) to verify the diagnosis of AIT;

2) to clarify the clinical and immunological decompensation of AIT [10,11,12].

- AT to thyroglobulin:

1) in complex diagnostics of AIT in parallel with AT to TPO;

2) for indirect assessment of thyroid autoaggression severity;

3) in screening of autoimmune thyroid pathology.

- ATT to TTG receptor:

1) in case of recurrent thyrotoxicosis;

2) to decide on the start and duration of thyrotoxic therapy;

3) for differential diagnosis of Graves' disease and an independent form of AIT [7,8,9].

'Big' diagnostic signs, the combination of which allows to establish the diagnosis of AIT, are: -primary hypothyroidism (manifest or persistent subclinical), -increased thyroid volume (more than 18 ml in women and more than 25 ml in men), -the presence of antibodies to thyroid tissue and/or ultrasound signs of autoimmune pathology. The diagnosis of AIT cannot be established solely on the basis of palpation of the thyroid gland, as well as the detection of an increase or decrease in its volume. In the absence of at least one of the 'big' diagnostic signs, the diagnosis of AIT is only probabilistic [4,5,6].

The last point is particularly important, because today the detection of antibodies against the thyroid gland or detection of almost any changes in its echogenicity is often considered as a pathognomonic sign of AIT. At the same time, the existing methods of antibody detection are not always perfect, the assessment of gland echogenicity may be rather subjective, but, most importantly, there are no catamnestic data on natural changes in the thyroid gland state, for example, in people with established hypoechogenicity of the gland or minimally enlarged gland size [1,2,3].

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