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CLINICAL AND CYTOLOGICAL DIAGNOSIS OF CHRONIC THYROIDITIS

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Autoimmune thyroiditis (AIT) is one of the most common diseases of the thyroid gland (thyroid), with more than 80 years having passed since its description by the Japanese surgeon Hashimoto [6,7,8].

Keywords: Thyroid gland, autoimmune thyroiditis, genetics, factors.

Currently, AIT is understood as a chronic organ-specific disease of the thyroid, characterized by lymphoid infiltration of its tissue arising due to autoimmune factors [5].

However, to date, there are a number of unresolved problems regarding AIT:

-the etiology and pathogenesis of the disease are not sufficiently clear; there is no single universally accepted classification of AIT;

-There are no clear criteria for diagnosis; there is no pathogenetic therapy of the disease, and approaches to symptomatic therapy are ambiguous.

There is no doubt that AIT is a genetically determined disease, which is realised when exposed to environmental factors. Observations of identical twins show that

they have concurrent AIT in 3-9% of cases, and in identical twins - in 30-60% of observations [1,14,17,18,19].

Genetic predisposition to the development of AIT is confirmed by the fact of its association with certain antigens of the HLA system; more often with HLA DR3 and DR5. According to Japanese researchers, the highest risk of AIT is associated with HLA DQW7 antigens [22,23,24].

It should be noted that HLA antigens are markers of a number of autoimmune diseases, so it is impossible to consider them as a 'disease gene'. It can only be a matter of congenital predisposition to a certain type of autoimmune reactions. The risk of developing AIT is largely determined by the age and sex of the patient. The ratio of women to men suffering from AIT at the age of 40-50 years is 10-15:1; in children there is 1 boy for every 3 girls with AIT. AIT rarely occurs in children younger than 4 years of age, with a peak incidence in children in the middle of puberty [19,20,21].

The prevalence of AIT in children is 0.1-1.2%, and in women over 60 years of age it reaches 10%. In addition to sex and age, environmental factors play a significant role in the development of AIT. In the literature, the question of the relationship between iodine intake and the risk of developing thyroiditis is debated. Experimental and clinical studies show that long-term intake of excessive iodine can lead to an increased incidence of AIT in individuals with a genetic predisposition to it. We are talking about very high doses of iodine - tens and hundreds of milligrams [16,17,18].

Hypothyroidism and, less frequently, thyrotoxicosis are the most typical symptoms of AIT. However, these clinical signs are very non-specific, as most patients with AIT have euthyroidism. In the hypertrophic form of AIT, an enlarged thyroid gland with a dense, often heterogeneous structure may be detected (palpatorily and/or visually). Such patients are often diagnosed with various forms of goitre. Functional state of the thyroid. During the evolution of the autoimmune process, the function of the thyroid undergoes stage changes with frequent outcome in hypothyroidism [13,14,15].

The cytological picture of AIT is represented by a diverse cellular composition, including histiocytes, endothelial cells, fibroblasts, macrophages, plasma and mast tissue cells, neutrophils, eosinophils, giant multinucleated cells of the foreign body

cell type. However, lymphoid elements, follicular epithelial cells and Ashkinazi cells (Gurtle) predominate. Two types of cytograms of Hashimoto's thyroiditis are distinguished. The first type is characterised by a mottled cellular composition with a predominance of mature lymphocytes. Ashkinazi cells (Gurtle) make up 8-10%. In the second type, lymphoblasts predominate, with many reticular cells. Ashkinazi (Gurtle) cells account for 40-60% [10,11,12].

Epithelial elements are arranged in single-layered layers, bundles or dense clusters (like parenchymatous areas). Increased proliferation of epithelium leads to the formation of papillae and gland-like structures with colloid in the centre. A feature of Ashkinazi cell morphology in Hashimoto's thyroiditis is cellular polymorphism with large differences in the nuclear-cytoplasmic ratio, as well as large nuclei and nuclear polymorphism. Caution should be exercised when Ashkinazi cells with signs of monomorphism (abundance of individual cells, clusters, microfollicles, papillary structures with absence or few lymphoplasmacytic component) predominate in the aspirate, as differential diagnosis with follicular B-cell tumour, papillary B-cell carcinoma is very difficult. Colloid is more often scanty, but is almost always present. Complexes of cells with squamous cell differentiation (a reflection of metaplasia as a result of the duration of the process) may also be determined. In some cases, the aspirate is represented exclusively by lymphoid cells [5,6,7,8,9].

Thus, patients with AIT, as a rule, have elevated levels of antibodies to TH, TPO and rarely - elevated levels of antibodies to the TTG receptor. In order to diagnose AIT, it is recommended to simultaneously determine antibodies to TH and TPO. The presence of both antibodies in the blood in diagnostic titres is a serious indication of either the presence or high risk of autoimmune pathology [3,4].

Puncture biopsy of the thyroid in some cases can be used for the purpose of differential diagnostics of AIT and other diseases. However, cytological diagnosis of AIT requires a sufficient amount of puncture material and high qualification of the morphologist performing the study [1,2].

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