THYROIDITIS AND RHEUMATOID ARTHRITIS AND IMMUNOLOGICAL PROCESSES IN THEM

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Abstract : *Rheumatoid arthritis is one of the most common non-organ-specific autoimmune diseases. The thyroid gland is one of the organs that can be damaged by autoimmune. Patients with rheumatoid arthritis may be at increased risk of developing thyroid disease.*

Key words: *thyroid gland, T4, T3, Autoimmune diseases, Rheumatoid arthritis.*

INTRODUCTION

The study of the pathogenesis of autoimmune diseases of the thyroid gland, which occupies a special place in the group of endocrine pathologies due to its high prevalence, is one of the urgent problems of modern medicine. The triggering mechanisms of their development are still unknown. In non-thyroid pathology, changes in the state of the thyroid gland, for example, in the development of the "non-thyroid syndrome", can trigger the synthesis of opposing cytokines by immunocytes, which can then lead to a loss of tolerance to thyroid autoantigens. It is thought that mast cells through their Toll-like receptors can affect the secretory activity of thyrocytes and thereby induce the synthesis of opposite cytokines and subsequently lose utotolerance. Mast cells established in the thyroid gland during its autoimmune pathology can egulate the functional activity of immunocytes and endocrinocytes by molecular separation of secretory material. However, it is not clear which of the mechanisms of mast cell activation dominates in autoimmune thyroid diseases, whether the effect of thyroid hormone levels on this activation is primary or changes in the state of the thyroid gland are secondary. In order to clarify these issues, the functional state of mast cells and production characteristics of opposing cytokines (IL-1b, IL-10, IFNy, TNFa) were studied in experimental thyrotoxicosis and hypothyroidism. Recombinant interleukin-2 was introduced to strengthen the immune system of one of the groups of experimental animals with thyrotoxicosis. Specific changes in the IFNγ/IL-10 ratio depending on the thyroid status indicated the importance of opposing cytokine balances in the development of different variants of this pathology. A significant increase in Th1-marker cytokines at the organ level in thyrotoxicosis proves that thyroid hormones are directly involved in immunoregulatory processes, which is confirmed by the focal infiltration of mast cells in the thyroid gland against the background of a significant increase in anti-inflammatory cytokines. systemic and organ levels. The hypothesis that thyroid hormones can be taken up by the cells of the immune system is supported by the existence of a strong correlation between the opposite cytokines in the target organ and

the level of thyroid hormones in the peripheral blood. The regulatory role of interleukin-2 in maintaining the balance of opposing cytokines and changing the direction of the vector of the immune response in the case of thyroid changes has been revealed. Based on research, the important role of mast cells and the balance of opposite cytokines in the pathogenesis of thyroid gland dysfunction became more clear. Further studies are required to clarify the mechanisms of interaction between thyroid hormones and immunocytes.

Rheumatoid arthritis is the most common inflammatory disease of the joints, characterized by erosive symmetrical polyarthritis combined with systemic immune-inflammatory lesions of internal organs (lungs, liver, heart, peripheral nervous system, skin). One of the most severe and common inflammatory diseases of the joints. Rheumatoid arthritis is a common disease that occurs in about 1% of the general population in all countries and all peoples of the world. Both a child and an elderly person can have rheumatoid arthritis, but women of an active age are more sick (women are sick with rheumatoid arthritis 3 times more often than men). The highest stage of the disease occurs between 40 and 55 years of age. The cause of the disease is unknown. It is only known that some people are genetically predisposed to rheumatoid arthritis, but the disease is not directly passed from parents to children. Smoking has been shown to increase the risk of developing rheumatoid arthritis. In 20-30% of patients, the disease begins after an infection, most often nasopharyngeal. However, the long-term search for a specific microorganism that causes rheumatoid arthritis has not been successful, so there is no reason to consider this disease infectious. Another important factor that triggers the development of the disease is severe emotional stress (quarrel, divorce, exams, etc.). At the same time, in about a third of patients, the first symptoms appear in complete health without any reason.

Materials and Methods: Inflammation in RA has been found to be based on a malfunction of the immune system. For some reason, the cells of the immune system are activated and begin to produce special regulatory proteins - anti-inflammatory cytokines, which cause an inflammatory reaction and damage the cells of the joint membrane, the inner lining of blood vessels, bone tissue. Causes tissue damage and others. As a result, a self-sustaining inflammatory process is formed. The inflammation in RA is the result of an overactive (albeit misguided) immune system. Therefore, rheumatoid arthritis can be included in the group of autoimmune diseases - that is, in conditions where the immune system actively works against its own body. They found that thyroid dysfunction was more common in people with RA. Hypothyroidism, meaning an underactive thyroid gland, was present in 16 percent of people with RA compared to 11.7 percent of controls. Hyperthyroidism is also common in people with RA. Hyperthyroidism. This can cause the following symptoms: nervousness, tremors (hand tremors), rapid heartbeat, frequent bowel movements. One of the most common causes of hyperthyroidism is an autoimmune condition called Graves' disease. RA is also an autoimmune disease. Studies have shown that the two conditions may be related—a higher prevalence of RA in people with Graves'

disease and vice versa. However, experts are not sure why. A 2019 study found that the cause of increased thyroid disease in people with RA is unclear. However, autoimmune conditions may share the same underlying pathology, which is another word for abnormal physiological processes. This may explain why a person with one autoimmune disease may have others.

Results and their analysis: Currently, RA is considered as a chronic systemic inflammatory disease, which affects not only the joints and periarticular tissues, but also the autonomic and central nervous system, the endocrine system, and their relationships. Additional, complications of the underlying disease [10]. Clinicians are aware of the frequent manifestations of pathology of the endocrine gland function in patients with RA, such as goiter, hypothyroidism, chronic adrenal insufficiency, etc. Thyroid hormones (thyroid gland) increase the activity of metabolic processes, stimulate lipogenesis, increase lipogenesis. absorption of glucose by fat and muscle tissue, activates gluconeogenesis and glycogenolysis [9]. Thyroid hormones (triiodothyronine - T3, thyroxine - T4) enhance both the resorption and synthesis of bone tissue and the production of glycosaminoglycans and proteoglycans in connective tissue. An increase in their amount in the body leads to the acceleration of bone tissue metabolism due to an increase in the number and activity of osteoclasts, and also stimulates osteoblastic function, which is expressed in the increase of bone formation markers in the blood [1, 11, 10]. With a lack of thyroid hormones, the activity of adenylate cyclase in synovial membranes increases, which increases the production of hyaluronic acid by fibroblasts, which leads to the accumulation of synovial fluid in the joints and causes the clinical appearance of synovitis [10, 2]. Enzyme immunoassay (ELISA), which is used to determine the level of antibodies to the thyroid gland, is a fast, sufficiently sensitive and reliable method for diagnosing its autoimmune lesion. In clinical practice, it is common to test antibodies directly to thyroid tissue, TSH receptors and thyroperoxidase antibodies [5, 6]. Autoantibodies to thyroid hormones specifically directed against T3 and T4 (THAAA) are less common. They have been known since 1956 J. Robbins et al. [7] first described the presence of γ-binding γ-globulin in papillary thyroid adenocarcinoma treated with iodine. S. Sakata, M. Matsuda, T. Ogawa, et al. [2, 18, 19, 20] also studied antibodies to thyroid hormones and the predominance of autoantibodies (anti-T3 and anti-T4) in the majority of the population is unusual, but their presence is increased in patients with hypothyroidism. Came to the conclusion that it is possible, hyperfunction of the thyroid gland and in autoimmune diseases not related to the pathology of the thyroid gland. According to the findings of D. Thomas et al. [9, 20], even if there are no clinical or biochemical changes in the thyroid gland, the presence of these antibodies reflects the early stages of the pathological process.

Conclusions: In the existing literature, there is little information about the processes of antibody formation to thyroid hormones in rheumatic diseases.

Therefore, if we consider that the detection of direct antibodies to thyroid hormones can be a test for the diagnosis of autoimmune thyroid disease, the possibility of their

detection, as well as their role in patients with RA and thyroid It was necessary to study the effect on the function.

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