ISSN: 2582-4686 SJIF 2021-3.261, 2022-2.889, 2023-5.384, 2024-6.875 ResearchBib IF: 9.948 / 2024

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-5, ISSUE-10

CHARACTERISTICS OF ENDOTHELIAL DYSFUNCTION IN PATIENTS WITH AUTOIMMUNE THYROIDITIS

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Keywords: Autoimmune thyroiditis, hypothyroidism, hyperthyroidism, endothelial dysfunction, coagulation, hemostasis, thrombosis, fibrinolysis, thyroid hormones, sICAM-1.

Abstract: This article provides information on the characteristics of von Willebrand factor, endothelin-1, sICAM-1 markers that change as a result of endothelial dysfunction in patients with autoimmune thyroiditis.

Introduction.

Autoimmune thyroiditis (AIT) is a chronic disease in which the thyroid gland cells gradually become inflamed. The functionality of the thyroid gland is disrupted because it is affected by autoantibodies. Therefore, the disease is described as autoimmune. The exact causes of the development of AIT are not fully understood, but specialists have found ways to significantly slow down the progression of the pathology.[8,2]

Most of the coagulation factors involved in the process of coagulation are synthesized under the influence of thyroid hormones. Disturbances in hemostasis are observed at various stages in patients with thyroid gland pathology.[4.10]

Hyperthyroidism is a clinical syndrome that occurs as a result of an increase in thyroid hormone levels, which can be caused by excessive production of these hormones (Graves' disease, thyroid gland adenoma, pituitary adenoma). It is characterized by tremors, emotional instability, heat intolerance, sinus tachycardia, rapid heart rate, hypertension, increased appetite, and weight loss. Many researchers associate hyperthyroidism and the increase in thyroid hormones with a state of hypercoagulability. This hypothesis supports the possibility that this hyperthyroidism may be a risk factor for venous thromboembolism. Because patients with this condition may experience thromboembolic events.

Endothelium is a metabolically active tissue that synthesizes various biological active substances. In patients with hyperthyroidism, the levels of adhesive molecules such as ICAM-1, VCAM-1, E-, L-, P-selectins, and endothelin-1, thrombomodulin, and other endothelial molecules increase [7,1].

Damage to the endothelial layer disrupts the coagulation cascade and opens up the factor that participates in primary hemostasis, von Willebrand multimers. Injured endothelial cells release endothelin-1, an anti-inflammatory peptide, and a potent vasoconstrictor from large and small arteries and veins. Additionally, under the influence of vasoconstrictors, the given peptide increases the concentration of intercellular adhesion molecule 1 (ICAM-1) and vascular cell adhesion molecule 1

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(VCAM-1) on endothelial cells. Endothelial dysfunction alters blood vessel homeostasis, leading to vasodilation and increased local aggregation of cells.[5,2]

The main part.

Autoimmune thyroiditis affects a total of 98 patients, with 27 patients in Group I, 34 patients in Group II, 26 patients in Group III, and 11 patients in Group IV.

Analysis of the distribution of patients by age and sex shows that there are more women among patients. Women are almost twice as rare. The control group included 20 patients. Among the patients aged 12-17 years in Group 1 there were 31 (18.8%) women and 20 (13.3%) men, 36 (29.1%) women and 24 15.8%) men, 18 (8.5%) women and 8 (6.1%) men aged 30-49 years, as well as 16 (4.8%) women and 12 (3.6%) men.

All complaints of patients with autoimmune thyroiditis were analyzed. Patients may experience goiter, fatigue, lack of energy, memory loss, apathy, depression, facial swelling, weight gain or loss without apparent reason, tachycardia, feeling cold, hair loss, menstrual irregularities, and so on. In some cases, the thyroid gland may initially produce excessive hormones, leading to symptoms of hyperthyroidism (excess hormones), but the clinical presentation quickly transitions to hypothyroidism symptoms. The clinical manifestations of Hashimoto's thyroiditis vary depending on the nature of the disease. Initially, patients may exhibit symptoms of hyperthyroidism because the initial destruction of thyroid gland cells can release thyroid hormones. However, as a result of the autoimmune antibody reaction, patients eventually develop symptoms of hypothyroidism. These symptoms are variable and can affect almost any organ system in the body.

In 98 patients with autoimmune thyroiditis who developed hypothyroidism, a significant increase in the level of AT-TPO hormone was observed. When the level of AT-TPO was determined in patients: in Group I, 27 patients had AT-TPO levels ->79 IU/ml, in Group II, 34 patients had AT-TPO levels ->200 IU/ml, in Group III, 26 patients had AT-TPO levels ->110 IU/ml, in Group IV, 11 patients had AT-TPO levels ->65 IU/ml, and in the control group, AT-TPO levels were <32 IU/ml. The levels of AT-TPO are presented in diagram form based on the table below.

The increase in AT-TPO hormone in the body is a sign of the presence of autoimmune thyroid disease. AT-TPO antibodies play a crucial role in diagnosing thyroid peroxidase diseases. IgG1 and IgG4, produced by B lymphocytes infiltrating the thyroid gland, respond to AT-TPO.

In addition, to determine the indicator of endothelial damage in AIT, s-ICAM1, Endothelin-1, and von Willebrand factor were studied. The disruption of the main functions of the endothelial layer leads to endothelial dysfunction. The main indicators of identifying endothelial dysfunction are considered to be the levels of molecules produced in it. These include von Willebrand factor, endothelin-1, and sICAM-1 (adhesion molecules) [3,9].

Conclusion:

In conclusion, it revealed significant changes in parameters of the hemostasis system and signs of endothelial dysfunction in patients with autoimmune thyroiditis. Among the key findings are Willebrand factor (vWF), endothelin-1, sICAM-1, and D-dimer, which indicate a prothrombotic state, as well as platelet hyper reactivity and endothelium-dependent changes. This study highlights the importance of considering hemostatic disturbances and endothelial dysfunction in patients with autoimmune thyroiditis. As a result of this research, 2 proposals are presented. Firstly, in patients with autoimmune thyroiditis hypothyroidism and hyperthyroidism, disorders of endothelial dysfunction were evident. As a result, signs of hyper coagulation and hypo coagulation disappear.

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Secondly, as a result of the research, it is recommended to control the endothelial dysfunction and regulate the indicators of thyroid hormones in the treatment of vascular complications in patients. Correlation of thyroid function and hemostasis system should be performed and managed.

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