



**VASCULAR ENDOTHELIAL DYSFUNCTION IN AUTOIMMUNE
THYROIDITIS: CLINICAL AND PATHOPHYSIOLOGICAL
CHARACTERISTICS**

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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 [1,7,11].

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 (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

The present study demonstrated substantial alterations in hemostatic parameters accompanied by clear evidence of endothelial dysfunction in patients with autoimmune thyroiditis. Elevated levels of von Willebrand factor (vWF), endothelin-1, sICAM-1, and D-dimer were identified as key indicators reflecting a prothrombotic tendency, together with increased platelet reactivity and endothelium-dependent vascular changes.

These findings emphasize the clinical relevance of assessing both hemostatic imbalance and endothelial impairment in individuals with autoimmune thyroiditis. The results further indicate that endothelial dysfunction is evident in patients with autoimmune thyroiditis at both hypothyroid and hyperthyroid stages, contributing to disturbances in coagulation homeostasis, including manifestations of hypercoagulability and, in some cases, hypocoagulability.

Based on the obtained data, two practical implications can be proposed. First, evaluation of endothelial function should be incorporated into the routine assessment of patients with autoimmune thyroiditis. Second, optimization of thyroid hormone levels alongside monitoring of endothelial markers and hemostatic indices is essential for the prevention and management of vascular complications. Continuous assessment of the relationship between thyroid function and the hemostatic system is therefore recommended as part of comprehensive clinical management.

REFERENCES:

1. Bekchanova N.I. Hemostatic changes in patients with autoimmune thyroiditis // *Zamonaviy tibbiyot yosh olimlar nigohida*. Tashkent 2022 №3 B804
2. Bekchanova N.I. Qalqonsimon bezi kasalliklarida gemostaz o'zgarishlari. // *Nazariy va Klinik tibbiyot jurnali*. Tashkent 2022 №5 B51
3. Bekchanova N.I., Babadjanova Sh.A., Hemostatic changes in pregnant women with thyroid disease // *Nazariy va Klinik tibbiyot jurnali* Tashkent 2022 №5 B48
4. Bekchanova N.I., Babadjanova Sh.A., Zaynutdinova D.L., Nuriddinova N.F. (2024) Hemostasis and Endothelial dysfunction in autoimmune thyroiditis: Insights



from hypothyroid patients. *MedForum: International Conference on Patient-Centered Approaches to Medical Intervention 2024* (28) 241-243.

5. Bekchanova, N. I. (2025). THE ROLE OF ENDOTHELIAL MARKERS (sICAM-1, ENDOTHELIN-1, VON WILLEBRAND FACTOR) IN THE ASSESSMENT OF VASCULAR DYSFUNCTION IN AUTOIMMUNE THYROIDITIS AT THE HYPOTHYROID STAGE. *Central Asian Journal of Medicine*, (7), 21-23.

6. Yuldashova, S. U. Bekchanova NI MODERN LABORATORY TECHNOLOGIES. *Zamonaviy klinik laborator tashxisi dolzarb muammolari*, v, 1, 49-50.

7. Bekchanova, N. I. (2024). HEMATOLOGIC MANIFESTATIONS OF HYPOTHYROIDISM.

8. Bekchanova, N. I. (2022). *Hemostatic changes in autoimmune thyroiditis* (Doctoral dissertation, Toshkent).

9. Bekchanova, N. I. (2022). *Gipotireoz bemorlarda gemostaz o'zgarishlari* (Doctoral dissertation, Ўзбекистон, Термиз).

10. Chernova E.V. Willebrand Factor // Bulletin of The North-West State Medical University Named After I.I. Mechnikov. - 2018. - Т. 10. - № 4. - С. 73-80. <https://doi.org/10.17816/Mechnikov201810473-80> Received:23.10.2018

11. Grin N.O., Serebryakova O.V., Serkin D.M. "Markers of Endothelial Dysfunction in Hypothyreosis" *Eni Transbaikal Medical Bulletin*, No. 3/2021 Doi: 10.52485/19986173_2021_3_70

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