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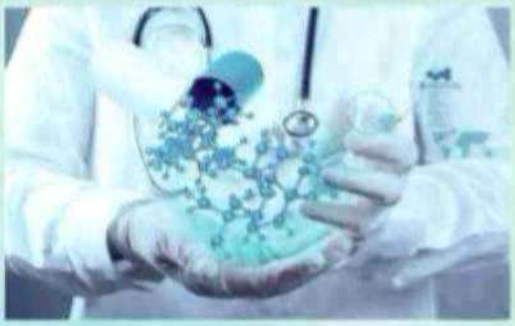

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FEATURES OF THE LIPID PROFILE AND VON WILLEBRAND FACTOR LEVELS IN PATIENTS WITH AUTOIMMUNE THYROIDITIS AS MARKERS OF CARDIOVASCULAR RISK

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✓ ***Resume***

Background. Autoimmune thyroiditis (AIT) is one of the most common thyroid disorders and a leading cause of primary hypothyroidism. In addition to impaired thyroid function, patients with AIT often develop lipid metabolism disorders and endothelial dysfunction, both of which may contribute to increased cardiovascular risk.

Objective. To assess the characteristics of the lipid profile and von Willebrand factor (vWF) levels in patients with different functional phases of autoimmune thyroiditis and to determine their significance in the formation of cardiovascular risk.

Materials and methods. The study included 113 patients with autoimmune thyroiditis and 94 apparently healthy individuals who formed the control group. All participants underwent assessment of blood lipid parameters, including total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, and the atherogenic coefficient. The level of von Willebrand factor was measured as a marker of endothelial injury and vascular-platelet hemostasis. Statistical analysis was performed using standard methods of biomedical statistics.

Results. Progression of thyroid insufficiency in AIT was accompanied by deterioration of the lipid profile. Patients demonstrated increased total cholesterol, triglycerides, low-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, and atherogenic coefficient, along with decreased high-density lipoprotein cholesterol. These changes were most pronounced in subclinical and overt hypothyroidism. Significant changes in von Willebrand factor levels were also observed, reflecting the development of endothelial dysfunction and disturbances in vascular-platelet hemostasis.

Conclusion. Lipid metabolism disorders in patients with autoimmune thyroiditis are associated with changes in von Willebrand factor levels and may be considered one of the mechanisms contributing to increased cardiovascular risk. Combined assessment of lipid profile parameters and endothelial dysfunction markers may help identify high-risk patients and optimize preventive strategies.

Keywords: autoimmune thyroiditis; dyslipidemia; von Willebrand factor; endothelial dysfunction; hypothyroidism; cardiovascular risk; lipid profile.

Relevance

Autoimmune thyroiditis is a chronic organ - specific autoimmune disease of the thyroid gland characterized by lymphocytic infiltration of thyroid tissue and gradual decline in its functional activity. In the overall structure of thyroid pathology, AIT occupies one of the leading positions and remains the most frequent cause of primary hypothyroidism in iodine-sufficient regions [4, 5, 7, 15].

In recent years, increasing attention has been paid not only to hormonal disturbances in AIT but also to the systemic metabolic consequences of the disease. Thyroid hormone deficiency has a significant effect on lipid metabolism, vascular remodeling, and endothelial function. Even subclinical hypothyroidism may be accompanied by atherogenic changes in the lipid spectrum, which subsequently increase the probability of cardiovascular complications [6, 8, 9, 14].

Thyroid hormones regulate the synthesis, transport, and catabolism of lipids. A decrease in thyroid hormone levels reduces the hepatic expression of low-density lipoprotein receptors, slows cholesterol clearance, and promotes the accumulation of atherogenic lipid fractions in the bloodstream. As a result,

patients with hypothyroidism frequently demonstrate hypercholesterolemia, elevated low-density lipoprotein cholesterol, increased triglycerides, and a higher atherogenic coefficient [2, 3, 6].

Alongside dyslipidemia, signs of endothelial dysfunction may develop in autoimmune thyroiditis. Endothelial dysfunction is currently regarded as an early component of the cardiovascular continuum. The endothelium contributes to the regulation of vascular tone, coagulation, inflammatory response, and vascular permeability. Chronic inflammation, oxidative stress, and metabolic abnormalities reduce endothelial cell activity and increase the risk of vascular complications [10, 11].

Von Willebrand factor is one of the informative laboratory markers of endothelial injury. This glycoprotein is synthesized by endothelial cells and megakaryocytes, mediates platelet adhesion to the vascular wall, and stabilizes coagulation factor VIII. Changes in vWF concentration are considered indicators of endothelial damage and impaired hemostatic balance [12].

Although numerous studies have addressed lipid disorders and endothelial dysfunction in thyroid diseases separately, the relationship between these abnormalities across different functional phases of autoimmune thyroiditis remains insufficiently clarified. This determines the relevance of further investigation of lipid profile characteristics and von Willebrand factor levels in this category of patients.

Purpose of the study

To study blood lipid profile parameters and von Willebrand factor levels in patients with different clinical phases of autoimmune thyroiditis and to evaluate their role in the formation of cardiovascular risk.

Materials and methods

The study was conducted at the outpatient department of the Third Clinic of Tashkent Medical Academy. A total of 113 patients with autoimmune thyroiditis in different functional phases of the disease were examined. The control group included 94 apparently healthy individuals of comparable age.

The diagnosis of autoimmune thyroiditis was established on the basis of clinical findings, thyroid ultrasound examination, thyroid-stimulating hormone (TSH), free thyroxine (free T4), and antibodies to thyroid peroxidase.

According to the functional status of the thyroid gland, patients were divided into four groups: euthyroidism - 65 patients; subclinical hypothyroidism - 25 patients; overt hypothyroidism - 15 patients; and hyperthyroidism - 8 patients.

All participants underwent blood lipid profile assessment, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and the atherogenic coefficient. To evaluate vascular endothelial status, von Willebrand factor levels were determined using an immunological method.

Statistical processing of the results was performed using methods of variation statistics. Quantitative variables are presented as mean and standard error of the mean (M±m). Differences were considered statistically significant at $p<0.05$.

Result and discussions

Analysis of lipid profile parameters in patients with autoimmune thyroiditis demonstrated that the severity of metabolic disorders depended on thyroid functional status. With increasing thyroid insufficiency, a gradual deterioration of the lipid profile was observed, indicating the formation of atherogenic changes and an increase in cardiovascular risk.

The most unfavorable lipid metabolism changes were found in patients with subclinical and overt hypothyroidism. These groups showed higher levels of total cholesterol, LDL-C, VLDL-C, triglycerides, and atherogenic coefficient, together with lower HDL-C values.

As shown in Table 1, patients with autoimmune thyroiditis had pronounced changes in the lipid profile, and the degree of these changes was associated with thyroid functional status. In the euthyroid group, total cholesterol was 5.22±0.97 mmol/L, exceeding the control value of 4.81±0.38 mmol/L. In subclinical hypothyroidism, total cholesterol increased to 5.87±1.39 mmol/L, while in overt hypothyroidism it reached the highest value of 6.42±1.17 mmol/L.

A similar pattern was observed for LDL-C. In the control group, LDL-C was 2.66±0.58 mmol/L, whereas in euthyroid AIT it increased to 3.20±0.99 mmol/L. In subclinical hypothyroidism and overt hypothyroidism, LDL-C reached 4.09±1.20 mmol/L and 4.21±1.36 mmol/L, respectively. These findings indicate a progressive increase in the atherogenic potential of the lipid profile as thyroid insufficiency develops.

Table 1. Lipid profile parameters in patients with autoimmune thyroiditis in different phases of the disease

Parameter	Control (n=94)	Euthyroidism (n=65)	Subclinical hypothyroidism (n=25)	Overt hypothyroidism (n=15)	Hyperthyroidism (n=8)
Total cholesterol, mmol/L	4.81±0.38	5.22±0.97	5.87±1.39*	6.42±1.17*	4.58±0.67
Triglycerides, mmol/L	1.08±0.48	1.23±0.39	1.42±0.81*	1.64±0.35*	1.19±0.29
HDL-C, mmol/L	1.66±0.38	1.37±0.29	1.14±0.31*	1.17±0.56*	1.88±0.23
LDL-C, mmol/L	2.66±0.58	3.20±0.99	4.09±1.20*	4.21±1.36*	2.81±0.46
VLDL-C, mmol/L	0.49±0.22	0.56±0.18	0.80±0.29*	0.62±0.22*	0.55±0.19
Atherogenic coefficient	1.90±0.70	3.02±0.98	4.11±1.25*	4.26±1.29*	2.74±0.38

*p<0.05 compared with the control group.

HDL-C levels, in contrast, decreased in the hypothyroid phases of AIT. While the control group had an HDL-C level of 1.66±0.38 mmol/L, this parameter decreased to 1.14±0.31 mmol/L in subclinical hypothyroidism and to 1.17±0.56 mmol/L in overt hypothyroidism. A decline in the anti-atherogenic lipoprotein fraction may further contribute to the risk of vascular complications.

The atherogenic coefficient was 1.90±0.70 in the control group. In patients with AIT, it was higher and reached 3.02±0.98 in euthyroidism, 4.11±1.25 in subclinical hypothyroidism, and 4.26±1.29 in overt hypothyroidism. These results confirm the presence of atherogenic remodeling of lipid metabolism in patients with autoimmune thyroiditis, particularly in thyroid insufficiency.

Thus, autoimmune thyroiditis, especially in the phases of subclinical and overt hypothyroidism, is associated with lipid metabolism disorders manifested by increased total cholesterol, LDL-C, VLDL-C, triglycerides, and atherogenic coefficient, together with reduced HDL-C.

Endothelial Function Assessed by von Willebrand Factor

To assess endothelial dysfunction, von Willebrand factor was measured in the examined patients. This parameter is considered one of the informative markers of endothelial damage and vascular-platelet hemostasis.

Table 2. Von Willebrand factor levels in patients with autoimmune thyroiditis (M±m)

Group	von Willebrand factor, %
Control group (n=94)	106.74±24.85
Euthyroidism (n=65)	105.40±23.44
Subclinical hypothyroidism (n=25)	93.75±26.80*
Overt hypothyroidism (n=15)	40.38±7.09*
Hyperthyroidism (n=8)	172.11±15.94*

*p<0.05 compared with the control group.

The analysis showed that in patients with preserved thyroid function, von Willebrand factor levels were almost comparable to those of the control group. However, with the development of thyroid insufficiency, a progressive decrease in this parameter was observed.

The most pronounced changes were recorded in patients with overt hypothyroidism, in whom von Willebrand factor levels decreased more than 2.5-fold compared with the control group. This finding may indicate substantial impairment of vascular endothelial function and hemostatic processes.

In contrast, patients with hyperthyroidism demonstrated a marked increase in von Willebrand factor levels up to 172.11±15.94%, which may be associated with endothelial activation and the formation of a hypercoagulable state.

Relationship Between Lipid Disorders and Endothelial Dysfunction

The obtained data indicate parallel progression of dyslipidemia and endothelial dysfunction in patients with autoimmune thyroiditis. As thyroid insufficiency increased, total cholesterol rose from 5.22±0.97 mmol/L in euthyroidism to 6.42±1.17 mmol/L in overt hypothyroidism. A similar trend was observed for LDL-C, which increased from 3.20±0.99 mmol/L to 4.21±1.36 mmol/L.

At the same time, von Willebrand factor levels decreased from 105.40±23.44% in euthyroidism to 40.38±7.09% in overt hypothyroidism. These findings suggest that deterioration of the lipid profile is accompanied by impairment of vascular endothelial function.

The most pronounced abnormalities were recorded in patients with overt hypothyroidism. Therefore, the combination of dyslipidemia and endothelial dysfunction may be regarded as an important pathogenetic mechanism contributing to increased cardiovascular risk in autoimmune thyroiditis.

Discussion

The results of the present study demonstrate a close pathogenetic relationship between lipid metabolism disorders and endothelial dysfunction in patients with autoimmune thyroiditis. As hypothyroidism progresses, dyslipidemia becomes more pronounced and is characterized by increased total cholesterol, triglycerides, LDL-C, VLDL-C, and atherogenic coefficient.

The observed lipid abnormalities can be explained by reduced lipoprotein lipase activity, decreased expression of hepatic LDL receptors, and slowed cholesterol catabolism under conditions of thyroid hormone deficiency. These mechanisms contribute to the accumulation of atherogenic lipoprotein fractions and may accelerate atherosclerotic vascular remodeling [2, 3, 6].

Simultaneously, deterioration of endothelial function occurs, as reflected by changes in von Willebrand factor levels. Chronic autoimmune inflammation, oxidative stress, and atherogenic lipid alterations exert damaging effects on endothelial cells and create conditions for vascular dysfunction. Endothelial dysfunction, in turn, may contribute to impaired regulation of vascular tone, inflammation, thrombogenicity, and microcirculatory homeostasis [10, 11, 12].

The findings of this study suggest that lipid disorders and von Willebrand factor changes develop in parallel with progression of thyroid insufficiency. The combination of dyslipidemia and endothelial dysfunction may therefore be considered a unified pathogenetic mechanism underlying increased cardiovascular risk in patients with autoimmune thyroiditis, especially in subclinical and overt hypothyroidism.

From a clinical perspective, these results emphasize the need for broader cardiovascular risk assessment in patients with autoimmune thyroiditis. Evaluation should not be limited to thyroid hormone levels alone. Lipid profile testing and, when clinically justified, assessment of endothelial dysfunction markers may improve early identification of high-risk patients and allow timely preventive interventions.

Conclusion

1. Patients with autoimmune thyroiditis demonstrate significant lipid metabolism abnormalities, the severity of which depends on the functional status of the thyroid gland.
2. Progression of thyroid insufficiency is accompanied by a statistically significant increase in total cholesterol, triglycerides, LDL-C, VLDL-C, and the atherogenic coefficient, together with a decrease in HDL-C.
3. The most pronounced atherogenic changes in the lipid profile are observed in patients with overt hypothyroidism, indicating a high probability of atherosclerotic vascular damage and cardiovascular complications.

4. Changes in lipid profile are accompanied by impairment of vascular endothelial function, manifested by significant changes in von Willebrand factor levels.
5. Reduced von Willebrand factor levels in hypothyroid phases of autoimmune thyroiditis may reflect endothelial dysfunction and disturbances in vascular-platelet hemostasis.
6. Combined determination of lipid profile parameters and von Willebrand factor levels may be used for early identification of patients with increased cardiovascular risk and for improving preventive strategies in autoimmune thyroiditis.

Practical Recommendations

1. Regular monitoring of lipid profile parameters is recommended for patients with autoimmune thyroiditis, regardless of the presence or absence of clinical manifestations of hypothyroidism.
2. In patients with subclinical hypothyroidism, an expanded assessment of cardiovascular risk factors should be performed, including lipid profile testing and evaluation of endothelial dysfunction markers when available.
3. Determination of von Willebrand factor may be considered as an additional laboratory marker for assessing endothelial status in patients with autoimmune thyroiditis.
4. Patients with a combination of dyslipidemia and endothelial dysfunction require dynamic follow-up by an endocrinologist and timely correction of detected metabolic abnormalities.

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