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STUDY OF THE FUNCTIONAL STATE OF THE THYROID GLAND AND DISEASES OF ITS PARENCHYMA IN WOMEN WITH METABOLIC SYNDROME

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Abstract

This article evaluates the functional state of the thyroid gland (TG) in women with metabolic syndrome through hormonal examinations, as well as pathological changes in the TG tissue using ultrasound and histological analysis. According to the results obtained, an increase in thyroid volume was observed in Group 1 (150 women with metabolic syndrome) compared to the control group: Grade I goiter was recorded in 37.3% of cases, and Grade II goiter in 62.7% of cases. Analysis of the functional state of the TG confirmed that cases of subclinical and overt hypothyroidism were significantly higher in women of Group 1 compared to Group 2 (women without metabolic syndrome).

Keywords: Thyroid gland, thyroiditis, hypothyroidism, nodular goiter, metabolic syndrome.

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Relevance of the study

In recent years, the dramatic increase in the prevalence of metabolic syndrome (MS) on both a global scale and within our republic has become one of the major challenges in modern medicine. Insulin resistance, dyslipidemia, and visceral obesity—the core components of MS—trigger systemic inflammation and hormonal imbalance in the body.

Endocrine and metabolic changes have a direct impact on thyroid gland function. Specifically, compensatory hyperinsulinemia, characteristic of MS, exerts a mitogenic effect on receptors (IGF-1) in the thyroid gland, accelerating proliferative processes (nodular and diffuse goiter). At the same time, inflammatory markers (IL-6, TNF-α) released from visceral adipose tissue activate oxidative stress and autoimmune inflammation (autoimmune thyroiditis) in the thyroid tissue, resulting in the development of subclinical and overt hypothyroidism. In endemic regions with iodine deficiency, such as the Fergana Valley, the comorbid occurrence of these pathologies leads to increased disease severity. Therefore, creating a system for early detection and comprehensive assessment of the risk of thyroid diseases against the background of metabolic disorders is one of the most urgent tasks in preventive medicine.

Purpose of the study

To study the functional state of the thyroid gland and parenchymal diseases in women with metabolic syndrome.

Objects and methods of the study

The study involved an analysis of clinical data from 200 women aged 35–60 years who sought outpatient treatment at the Fergana branch of the Republican Specialized Scientific and Practical Medical Center of Endocrinology (RSSPMCE) between 2024 and 2026.

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Particular attention was paid to hormonal assessment of the thyroid gland's functional state and ultrasound (US) examination of its parenchyma to identify specific pathologies. Based on these indicators, the patients were divided into two groups:

- **Group 1:** 150 women with metabolic syndrome.
- **Group 2 (control):** 50 women without metabolic syndrome.

All patients underwent a general clinical examination, palpation of the thyroid gland, biochemical blood analysis, CLIA (chemiluminescent immunoassay) hormonal testing (T3, T4, TSH, Anti-TPO), and thyroid ultrasound analysis.

Results:

According to the WHO classification, thyroid Grade 0 was not observed in any of the patients in Group 1 (women with metabolic syndrome). Grade I thyroid enlargement was recorded in 56 patients (37.3%), and Grade II thyroid enlargement was recorded in 94 patients (62.7%).

In the Group 2 control group (women without metabolic syndrome), thyroid Grade 0 was identified in 13 patients (26%), Grade I in 20 patients (40%), and Grade II thyroid enlargement in 17 patients (34%) (Table 1).

Table 1 Thyroid enlargement degree based on WHO classification and palpation results in examined patients

№ Group		Thyroid Enlargement Degree			Total
		Grade 0	Grade I	Grade I	
Group 1	Women with metabolic syndrome (n=150)	0 (0%)	56 (37%)	94 (63%)	150 (100%)
Group 2	Women without metabolic syndrome (n=50)	13 (26%)	20 (40%)	17 (34%)	50 (100%)
	Total	3 (2%)	76 (42%)	111 (56%)	200 (100%)

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In the next stage, the thyroid hormone levels and thyroid status of patients in groups 1 and 2 were assessed based on TSH, T3, T4, and Anti-TPO levels (Table 2).

Table 2 Functional state of the thyroid gland in the examined patients

Groups	Euthyroidism	Subclinical Hypothyroidism	Overt Hypothyroidism	Subclinical Hyperthyroidism	Total
Women with metabolic syndrome (n=150)	75 (50%)	55 (36,6%)	12 (8%)	8 (5,4%)	150 (100%)
Women without metabolic syndrome (n=50)	41 (82%)	9 (18%)	0	0	50 (100%)

As shown in Table 2, in Group 1 (women with metabolic syndrome), 75 (50%) were in a euthyroid state, 55 (36.6%) had subclinical hypothyroidism, 12 (8%) had overt hypothyroidism, and 8 (5.4%) had subclinical hyperthyroidism. In Group 2 (women without metabolic syndrome), 41 (82%) were euthyroid, and 9 (18%) had subclinical hypothyroidism. Overt hypothyroidism and subclinical hyperthyroidism were not observed in any of the women in the control group. The results indicate that in women with metabolic syndrome, the euthyroid state is compromised due to metabolic slowdown, leading to a transition toward hypo- or hyperthyroid states.

In the next stage, pathologies detected in the thyroid gland via ultrasound (US) were compared and evaluated. Analysis of Group 1 revealed: nodular goiter in 6 (5.3%) patients, mixed goiter in 11 (7.3%) patients, multinodular goiter in 12 (8%) patients, and autoimmune thyroiditis in 67 (44.6%) patients.

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In the control group (Group 2), nodular goiter was found in 2 (4%) women, mixed goiter in 2 (4%), autoimmune thyroiditis in 9 (18%), and multinodular goiter in 2 (4%) women. (Table 3).

Table 3 Pathologies of the thyroid gland detected by ultrasound (US) in examined patients

Groups	Nodular Goiter	Mixed Goiter	Multinodular Goiter	Autoimmune Thyroiditis
Women with metabolic syndrome (n=150)	6 (5,3%)	11 (7,3%)	12 (8%)	67(44.6%)
Women without metabolic syndrome (n=50)	2 (4%)	2 (4%)	2 (4%)	9 (18%)

These results demonstrate an increase in nodular changes, specifically multinodular goiter, in the thyroid tissue of women with a history of metabolic syndrome. Furthermore, it was determined that the presence of metabolic syndrome in this group led to an increase in autoimmune diseases of the thyroid tissue. These findings suggest a negative impact of metabolic syndrome on thyroid tissue in conditions of iodine deficiency. We believe that iodine deficiency plays a significant role in these changes, and additional studies providing further evidence should be conducted in this regard. The results obtained in both groups indicate an increase in multinodular and mixed types of changes in thyroid tissue. This raises the question of whether this condition is a complication of iodine deficiency or a complication of metabolic syndrome.
Here is the translation of the "Conclusion" section into English:

Conclusion

The results of this study demonstrate that thyroid gland pathologies are significantly more prevalent in women with metabolic syndrome (MS) compared

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to the control group. Based on our data, the correlation between metabolic disorders and thyroid disease can be explained by three primary factors:

Firstly, the direct impact of insulin resistance. The hyperinsulinemia identified in our study activates growth factors (IGF-1) within the thyroid cells (thyrocytes). This mechanism stimulates the enlargement of the gland and the formation of nodules. The 94 cases of Grade II goiter observed in our study are a direct consequence of this proliferative process.

Secondly, the role of visceral adipose tissue as a source of chronic inflammation. The 44.6% incidence of autoimmune thyroiditis (AIT) observed in our study is not incidental. Pro-inflammatory mediators released by visceral fat tissue impair the immune system's self-tolerance, leading to autoimmune attacks on thyroid tissue. This finding is highly consistent with international research (e.g., Biondi et al., 2024), which highlights the link between systemic inflammation and thyroid autoimmunity.

Thirdly, the regional factor. In the iodine-deficient environment of the Fergana Valley, the presence of metabolic syndrome appears to accelerate the progression of thyroid pathology significantly. The fact that the control group (women without MS) exhibited more stable thyroid parameters suggests that the root of the problem lies not only in iodine deficiency but also in the patient's metabolic status.

In conclusion, our findings suggest that endocrinologists should not focus solely on thyroid hormone replacement (TSH, T4) when treating these patients. It is essential to simultaneously manage body weight, monitor insulin levels, and control systemic inflammation. Without addressing the metabolic syndrome, hormone therapy alone may be insufficient to stop the progression of nodular goiter.

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