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DISORDERS OF HEMATOPOIESIS IN HYPOTHYROIDISM AND HYPERTHYROIDISM

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Abstract
The article considers the influence of hypo- and hyperthyroidism on hematopoiesis processes. It is shown that hypothyroidism is characterized by normo- and microcytic anemia, caused by decreased erythropoiesis and impaired iron metabolism. Hyperthyroidism is characterized by relative polycythemia, changes in the leukocyte and platelet levels. It is emphasized that timely correction of thyroid status in most cases leads to normalization of blood parameters, however, some patients require additional therapy and examination to exclude concomitant pathologies.

Keywords: Hypothyroidism, hyperthyroidism, hematopoiesis, anemia, erythropoiesis, thyroid hormones, thyroid gland.

Introduction

The scientific novelty of this article is that for the first time a comprehensive analysis of hematopoiesis disorders in hypo- and hyperthyroidism has been carried out taking into account pathophysiological mechanisms, clinical manifestations and laboratory features. The work systematizes modern data on the influence of thyroid hormones on various links of hematopoiesis, identifies characteristic diagnostic markers and gives practical recommendations for the correction of the detected disorders, which allows us to clarify approaches to the management of patients with thyroid pathies.

Thyroid function has a direct impact on hematopoiesis processes. Thyroid hormones (T4 and T3) are key regulators of energy metabolism, as well as cell growth and differentiation processes, including hematopoietic precursors in the bone marrow. Thyroid status disorders can alter iron metabolism, erythropoietin production, and bone marrow activity, which ultimately affects peripheral blood parameters [1].

Clinical and experimental data indicate a direct relationship between thyroid dysfunction and hematological changes. Hypothyroidism is often associated with the development of anemia, which in most cases is normocytic and normochromic. However, microcytic forms caused by iron metabolism disorders may also occur. Hyperthyroidism may be accompanied by erythrocytosis or

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relative polycythemia. Disturbances in the leukocyte and platelet germ are also observed [2] .

Autoimmune thyroid diseases (Graves' disease, autoimmune thyroiditis) are often combined with other immune-mediated hematological disorders, such as autoimmune hemolytic anemia and thrombocytopenia. This fact emphasizes the importance of a comprehensive approach to assessing the hematological status in patients with thyroid disorders. Therefore, studying the effect of hypo- and hyperthyroidism on hematopoiesis is of high clinical importance for timely diagnosis and effective correction of concomitant blood disorders. Restoration of normal thyroid status is a key element in the treatment of secondary hematological complications.

The influence of thyroid hormones on the hematopoietic system is mediated by several pathophysiological mechanisms, which include direct effects on the bone marrow, indirect regulation through erythropoietin, as well as effects on metabolic and immunological processes:

1. Direct effect on bone marrow. Thyroid hormones (T3 and T4) participate in the regulation of the cell cycle and differentiation of hematopoietic precursors, especially erythrocytes and myeloid cells. Experimental studies confirm that triiodothyronine (T3) stimulates erythroid cell proliferation by acting on thyroid hormone receptors (TRα and TRβ) in the nuclei of bone marrow cells [3]. Accordingly, in hypothyroidism, a decrease in T3 and T4 levels leads to a decrease in this stimulation, which is manifested by anemia.

2. Indirect influence via erythropoietin . Erythropoietin (EPO) is one of the key mediators in this process. Thyroid hormones increase the expression of EPO in the kidneys, thereby enhancing erythropoiesis. Consequently, hypothyroidism is accompanied by a decrease in EPO concentration, while thyrotoxicosis increases its level. This mechanism partially explains the development of anemia in hypothyroidism and, conversely, erythrocytosis in hyperthyroidism.

3. Metabolic and nutritional factors. Thyroid dysfunction also affects the metabolism of iron, vitamin B12, and folic acid. Hypothyroidism reduces intestinal iron absorption, and autoimmune gastritis often develops, which leads to impaired absorption of vitamin B12. These factors significantly increase the risk of developing iron- and B12-deficiency anemia.

4. Immunological mechanisms. Autoimmune diseases of the thyroid gland (Graves' disease, autoimmune thyroiditis) are often associated with other

autoimmune cytopenias, including autoimmune hemolytic anemia and thrombocytopenia [4]. In rare cases, severe hypothyroidism can lead to pancytopenia due to the combined effects of metabolic deficiencies and autoimmune reactions.

Table 1 - The main mechanisms of influence of thyroid hormones on hematopoiesis

Mechanism	Hypothyroidism	Hyperthyroidism
Direct effect on bone marrow	Decreased stimulation of erythroid cells → normocytic / microcytic anemia	Increased erythroid cell proliferation
Effect on erythropoietin (EPO) production	Decreased EPO production → anemia	Increased EPO production → relative polycythemia
Metabolism of iron and vitamins	Impaired absorption of iron, B12, folates → iron- and megaloblastic anemia	Increased iron metabolism, sometimes deficiency with high catabolism
Immunological mechanisms	Association with autoimmune anemia, pancytopenia	Association with autoimmune thrombocytopenia

Thyroid dysfunction leads to characteristic changes in the blood system, which have their own characteristics in hypothyroidism and hyperthyroidism.

In hypothyroidism, the most common hematological manifestation is anemia. According to meta-analyses, patients with clinical hypothyroidism have a significantly higher risk of developing anemia than the general population [5].

Types of anemia in hypothyroidism:

- normocytic normochromic anemia is the most common type, which reflects direct suppression of erythropoiesis and decreased production of erythropoietin;
- microcytic hypochromic anemia develops with concomitant iron deficiency caused by impaired absorption;
- megaloblastic anemia is less common and is caused by a deficiency of vitamin B12 or folic acid, often associated with autoimmune gastritis.

In rare cases, severe and prolonged hypothyroidism can lead to pancytopenia, which is explained by combined bone marrow suppression and autoimmune processes [6].

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Hematological changes in hyperthyroidism are more variable:

1. Some patients may have relative polycythemia due to increased erythropoietin production [2]. However, paradoxical conditions such as anemia due to increased catabolism or nutritional deficiency are also possible.
2. Leukopenia or lymphocytosis is often noted. Platelet disorders may develop, including thrombocytopenia or, less commonly, thrombocytosis, especially if hyperthyroidism is autoimmune in nature (eg, in Graves' disease).

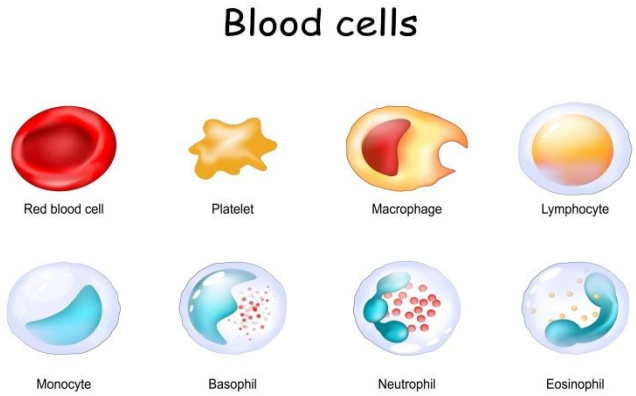

Fig. 1 - The main components of blood

Thus, clinical manifestations of hematopoiesis disorders in hypo- and hyperthyroidism differ not only in frequency but also in character. In hypothyroidism, anemic syndrome dominates, whereas in hyperthyroidism, both stimulation of erythropoiesis and the development of cytopenias of various origins are possible. The involvement of leukocyte and platelet sprouts in both conditions confirms the systemic effect of thyroid hormones on the bone marrow. For ease of generalization of key laboratory and clinical changes in various thyroid dysfunctions, Table 2 is presented, which lists the main manifestations and their pathogenetic features.

Table 2 - Clinical and laboratory changes in hypo- and hyperthyroidism

Indicator / manifestation	**Hypothyroidism**	**Hyperthyroidism**
Erythrocyte sprout	Normocytic / normochromic anemia; microcytic anemia (iron deficiency); megaloblastic anemia (B12/ folates)	Relative polycythemia; sometimes iron deficiency anemia
Leukocytes	Leukopenia or neutropenia may occur.	Leukopenia, lymphocytosis
Platelets	Thrombocytopenia (rare)	Thrombocytopenia, less commonly thrombocytosis
Rare conditions	Pancytopenia in severe hypothyroidism	Immune cytopenias (eg, Graves' disease)

Detection of hematological disorders in patients with thyroid dysfunction requires a comprehensive approach, including both routine and specialized laboratory tests. The stages of the diagnostic process include:

1. Complete blood count (CBC). The first step is to perform a CBC to assess the presence of anemia, leukocyte or platelet changes. In hypothyroidism, normocytic or microcytic anemia is most often detected, and in hyperthyroidism - relative polycythemia or cytopenia.

2. Assessment of nutritional and biochemical markers. Serum iron, ferritin, total iron -binding capacity, and vitamin B12 and folate levels are tested to determine the cause of anemia. Deficiencies of these nutrients are closely associated with thyroid dysfunction and may worsen anemia.

3. Hormonal examination. To verify the nature of thyroid dysfunction, the concentrations of thyroid stimulating hormone (TSH) and free fractions of T4 and T3 are determined. A combined assessment of thyroid status and blood parameters allows for differential diagnosis of anemia associated with thyroid dysfunction from other possible causes.

4. Specialized and immunological tests. If the autoimmune nature of the disease is suspected, antibodies to the thyroid are additionally examined. peroxidase (AT-TPO) and antibodies to the TSH receptor. In case of suspected autoimmune cytopenias, the Coombs test is performed and hemolysis markers are assessed.

5. Invasive methods. In the presence of pronounced and persistent cytopenias that are not explained by thyroid pathologies, a consultation with a hematologist and a bone marrow puncture/biopsy may be required to exclude primary hematological diseases.

Table 3 - Diagnostic approaches for hematopoiesis disorders in patients with hypo- and hyperthyroidism

Diagnostic stage	Research	Target
Primary screening	Complete blood count (Hb , erythrocytes, leukocytes, platelets, erythrocyte indices)	Detection of anemia and cytopenias
Blood biochemistry	Serum iron, ferritin , TIBC, B12, folates	Determination of deficiencies associated with anemia
Hormonal tests	TSH, free T4 , free T3	Confirmation of hypo- or hyperthyroidism
Immunology	AT-TPO, AT- rTSH , Coombs test , hemolysis markers	Diagnosis of autoimmune disorders
Invasive methods	Bone marrow puncture/biopsy	Exclusion of primary blood diseases

Treatment of hematological disorders associated with thyroid dysfunction should be comprehensive and bidirectional. First, it is necessary to restore normal thyroid status. Second, concomitant causes of cytopenias, such as iron, vitamin B12, or folate deficiency, should be diagnosed and corrected simultaneously.

The standard approach to treating hypothyroidism is replacement therapy with levothyroxine . In many cases, this results in normalization of hematological parameters, but the effect depends on the underlying cause of the anemia. If the anemia is caused solely by thyroid dysfunction, blood parameters often recover once euthyroidism is achieved. However, if iron or B12 deficiency is also present, simultaneous replacement therapy of these deficiencies significantly increases the effectiveness of treatment [7].

Practical recommendations:

1. Initiate levothyroxine therapy according to clinical guidelines, taking into account age and cardiovascular risks.
2. If iron deficiency is detected, prescribe iron preparations (orally or parenterally), observing the time interval with taking levothyroxine .

Correction of thyrotoxicosis with antithyroid drugs, radioiodine therapy, or surgery usually results in normalization of most associated hematological abnormalities. The choice of treatment depends on the cause and associated factors. It is important to remember that radioiodine therapy itself may cause changes in blood counts, so careful monitoring is necessary [8]. In addition to restoring thyroid status, specific treatment of identified hematological abnormalities may be required:

- iron deficiency anemia requires standard therapy with iron preparations;
- B12/ folate deficiency is corrected by vitamin replacement therapy [9];
- autoimmune cytopenias require specialized treatment (corticosteroids, immunoglobulins). In some cases, normalization of thyroid function promotes remission, but more often an independent hematological approach is necessary;
- in case of persistent and pronounced cytopenias, a consultation with a hematologist is indicated and, if necessary, a bone marrow puncture to exclude primary hematological diseases [10].

In most cases, hematological disorders secondary to thyroid dysfunction are reversible with timely and adequate treatment. However, there are exceptions:

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- in the presence of other causes of anemia (for example, primary iron deficiency or myelodysplastic syndrome), normalization of thyroid status may be insufficient;
- in elderly patients with subclinical hypothyroidism, the effect on hemoglobin may be absent, which requires an individual approach;
- outcome of autoimmune diseases Cytopenia depends on the severity of the immune process and the response to specialized therapy.

Table 4 - Treatment tactics and prognosis for hematologic disorders associated with thyroid dysfunction

Problem	Basic treatment	Additional/symptomatic therapy	Forecast
Hypothyroidism with mild anemia (thyroid -mediated)	Replacement therapy with levothyroxine (achieving euthyroidism).	As a rule, no specific hematological therapy is required; CBC monitoring.	Good, often normalizes Hb in euthyroidism.
Hypothyroidism + iron deficiency anemia	L-thyroxine + correction of iron deficiency (oral/parenteral).	Take into account the intervals of administration (iron can reduce the absorption of L-thyroxine).	Good with combination therapy; restoration of ferritin stores takes weeks to months.
Subclinical hypothyroidism in the elderly with anemia	The decision is individual; treatment with L-thyroxine does not always increase Hb in the elderly.	Investigate other causes of anemia and treat as indicated.	The outlook depends on the underlying causes; the effect of L-thyroxine is not guaranteed.
Hyperthyroidism with cytopenias	Elimination of thyrotoxicosis (ATD/RAI/ thyroidectomy).	Supportive therapy (in severe cases, transfusion, immunotherapy for autoimmune cytopenias).	Often reversible in euthyroidism; variable in immune processes.
Severe/persistent cytopenia	Treatment of the underlying cause of hypo /hyperthyroidism + urgent hematological assessment (BM puncture).	Specific hematological therapy as indicated (corticosteroids, immunoglobulins, cytostatic therapy, etc.).	Dependent on the underlying disease; requires a multidisciplinary approach.
After radioiodine therapy (monitoring)	Thyroid status monitoring ; correction based on results.	Monitoring of CBC in the presence of symptoms; treatment of complications according to standards.	Most changes are reversible; temporary or long-term changes are possible (monitoring is essential)

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Based on the presented data, it is possible to formulate key practical recommendations aimed at optimizing the diagnosis and treatment of hematological disorders in patients with thyroid disorders:

1. It is recommended to perform a complete blood count both at the stage of primary diagnostics and at subsequent control visits in all patients with detected thyroid dysfunction. This will allow timely detection of anemia, as well as changes in the leukocyte and platelet links.
2. When anemia is detected in a patient with hypothyroidism, it is necessary not only to begin replacement therapy with levothyroxine, but also to conduct a parallel examination to exclude and correct concomitant causes, such as iron deficiency, vitamin B12 or folic acid.
3. In cases of persistent or severe cytopenias that cannot be corrected after normalization of the thyroid status, patients should be referred to a hematologist. This is necessary to exclude primary bone marrow diseases or complex autoimmune syndromes that require specialized treatment.

Thus, the relationship between thyroid function and hematopoiesis has significant clinical significance. Adequate diagnostics and timely correction of thyroid pathologies , as well as concomitant nutritional and immunological disorders, is the basis for successful treatment of secondary hematological complications. This approach allows not only to normalize blood parameters, but also to improve the general condition and prognosis for the patient.

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