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COURSE OF ANEMIA IN DIFFUSE TOXIC GOITER

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Abstract:

This article describes the causes, clinical course, and specific features of anemia in patients with diffuse toxic goiter (thyrotoxicosis). It analyzes the effects of thyroid hormone overproduction on accelerated metabolism, impaired absorption of iron and vitamin B12, and the role of autoimmune factors in the development of anemia. The importance of timely diagnosis and correction of anemia alongside the treatment of thyrotoxicosis to improve patients' quality of life is emphasized.

Keywords: Diffuse toxic goiter, thyrotoxicosis, anemia, thyroid gland, metabolism, iron deficiency.

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Relevance of the topic. Diffuse toxic goiter (DTG), or thyrotoxicosis, remains one of the most common and serious disorders of the endocrine system among thyroid diseases. DTG is characterized not only by hormonal imbalance but also by adverse effects on the functional state of all organs and systems, particularly the cardiovascular, nervous, and hematopoietic systems.

Changes in the blood system, particularly the development of anemia of varying severity, are frequently observed in patients with thyrotoxicosis. Anemia associated with DTG has a multifactorial origin and may result from excessive acceleration of metabolism, impaired gastrointestinal absorption of iron, vitamin B12, and folic acid, as well as autoimmune processes. The manifestations of anemia are often masked by the general symptoms of thyrotoxicosis, such as weakness, palpitations, and rapid fatigue, making timely and accurate diagnosis more difficult. The coexistence of anemia and thyrotoxicosis further aggravates the general condition of patients and reduces their quality of life.



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Aim of the study. To investigate the mechanisms of anemia development and the characteristics of its clinical course in patients with diffuse toxic goiter, and to develop recommendations for the timely detection of this condition and improvement of treatment effectiveness.

MAIN SECTION

Pathogenetic mechanisms of anemia development in diffuse toxic goiter

The development of anemia in diffuse toxic goiter (DTG) is a complex, multifactorial process based on the following pathological mechanisms:

Hydremia (increase in plasma volume): In thyrotoxicosis, the circulating blood volume increases mainly due to expansion of the plasma volume. As a result, relative hemodilution occurs, and erythrocyte and hemoglobin values decrease artificially (pseudoanemia).

Shortening of erythrocyte lifespan: A sharp increase in serum thyroid hormone levels (T3 and T4) disrupts the stability of erythrocyte membranes and leads to their premature destruction (g hemolysis).

Disturbances of iron and vitamin metabolism: Hypermetabolism markedly increases the body's requirements for iron, vitamin B12, and folic acid. At the same time, gastrointestinal hypermotility (diarrhea) and achylia, which are characteristic of thyrotoxicosis, reduce the absorption of these substances.

Autoimmune mechanisms: Because DTG is an autoimmune disease, autoantibodies may be produced not only against the thyroid gland but also against erythroid precursor cells or the parietal cells of the gastric mucosa (anti-



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intrinsic factor antibodies). This may cause autoimmune hemolytic anemia or vitamin B12 deficiency anemia.

2. Clinical features of the combined course of anemia and thyrotoxicosis

In clinical practice, the coexistence of DTG and anemia is characterized by mutual amplification of their symptoms (a syndrome of mutual aggravation):

Clinical manifestations	DTG-only group	DTG + anemia group
Cardiovascular system	Tachycardia, awareness of heartbeat	Persistent tachycardia, shortness of breath, high risk of myocardial dystrophy
Neurological status	Tremor, nervousness, excessive sweating	Dizziness, tinnitus, tendency to faint
Skin and mucous membranes	Warm, moist, hyperemic skin	Pale-yellowish, dry skin and brittle nails

The subjective symptoms of anemia, such as weakness and rapid fatigue, are often masked by the main manifestations of thyrotoxicosis. Therefore, physicians must pay particular attention to laboratory findings.

Modern approaches to diagnosis and treatment

Effective correction of anemia in patients with DTG requires a two-pronged treatment strategy:

Laboratory diagnosis: In addition to a complete blood count, serum ferritin, transferrin, vitamin B12, and reticulocyte levels must be measured to determine the specific type of anemia.



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Etiotropic therapy (treatment of thyrotoxicosis): After euthyroidism is achieved using antithyroid medications such as thiamazole or propylthiouracil, hemoglobin levels normalize spontaneously in most patients.

Symptomatic and pathogenetic treatment: Depending on the type and severity of anemia, iron preparations, administered orally or parenterally, cyanocobalamin, and folic acid are added to the treatment plan. During treatment, the toxic effects of antithyroid medications on the bone marrow, including the risk of leukopenia and agranulocytosis, must be continuously monitored.

CONCLUSION

The investigation of the course of anemia associated with diffuse toxic goiter (DTG) led to the following conclusions:

Multifactorial pathogenesis: The development of anemia in patients with DTG is associated not only with hormonal disturbances but also with profound changes in homeostasis, including hydremia (blood dilution), shortening of erythrocyte lifespan, increased requirements for iron and vitamin B12 due to hypermetabolism, and autoimmune processes.

Syndrome of mutual aggravation: The coexistence of anemia and thyrotoxicosis significantly worsens the clinical condition of patients. Symptoms of anemia, such as weakness and pallor, are often concealed by manifestations of hypermetabolism; nevertheless, this combination sharply increases the risk of cardiovascular complications such as hypoxia and myocardial dystrophy.



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Effectiveness of comprehensive treatment: The basis of successful anemia treatment in DTG is the rapid achievement of euthyroidism (normal thyroid hormone levels) using antithyroid medications. After euthyroidism is achieved, blood parameters recover spontaneously in many cases. However, in moderate and severe anemia, pathogenetic therapy with iron and vitamin B12 preparations must be administered concurrently, and the risk of leukopenia must be monitored throughout treatment.

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