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Clinical-Laboratory Diagnostics of Diffuse Toxic Goiter

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Abstract: The article provides detailed information about the effects of diffuse toxic goiter on human life and body, their treatment with several different methods, laboratory diagnostics and assessment of hormonal changes.

Key words: diffuse toxic goiter, anemia, thyroid gland, autoimmune, thyrotoxicosis, nonthyroidal illness, exophthalmos.

Diffuse toxic (poisonous) goiter (DTG) is an autoimmune disease characterized by a persistent increase in QB thyroid hormones under the influence of antibodies to TTG receptors with the development of thyrotoxicosis and diffuse damage to the thyroid gland. DTG and thyrotoxicosis are diagnosed according to MKB-10 as follows:

- E 05 – thyrotoxicosis.
- E05.0 - diffuse goiter with thyrotoxicosis.
- E05.1 – single nodular toxic goiter with thyrotoxicosis.
- E05.2 – multinodular toxic goiter with thyrotoxicosis [16].

According to various authors, the incidence of DTG is 15-50 people per 1000 people per year. In most cases, DTG occurs in people aged 20-40. The incidence of DTG in men and women is 7-10%, and 3% in children. Girls and boys have the same frequency of encounters. DTG develops under the influence of genetic predisposition and negative factors. Predisposing factors include genetic disorders in the immune system leading to T-suppressor deficiency. The development of DTG is associated with the carrier gene STL44 (cytotoxic T-lymphocyte 4 antigen), as well as haplotypes such as HLA B8, HLA DR3, HLA -DR W3 [24].

The negative factors causing the development of DTG are divided into internal (stress) and external effects (level of iodine intake, viral and bacterial infection, smoking). Based on the classification of diffuse toxic goiter, there are signs such as the volume of QB and the severity of thyrotoxicosis.

Currently, in practical endocrinology, the classification of DTG, which is common to all thyroidological conditions, is used (Table 1).

Table 1.
DTG classification (World Health Organization 2001)

Goiterlevel	**Signs**
Level 0	No goiter
Level 1	The size of the distal phalanx of the thumb increases, or there are no enlarged nodes in the QB.
Level 2	The lump is palpated and visually identified

According to the degree of severity of thyrotoxicosis, mild, medium and severe types are distinguished (Table 2).

Table 2. Classification of thyrotoxicosis according to severity

Severitylevel	**Basiccriteria**
Subclinical (mild)	The clinical manifestation is not observed or weak; TTG is slightly reduced; free T4 and T3 are slightly increased
Manifesto (medium weight)	Extended clinical presentation of thyrotoxicosis; TTG decreases; free T4 and T3 are increased
Complicated (severe)	Manifestations of thyrotoxicosis with complications: tremor, arrhythmia, heart failure, relative adrenal

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	insufficiency, dystrophic changes of parenchymatous organs, psychosis, lack of body weight; TTG decreases sharply; free T4 and T3 increase dramatically

In addition, cases of decompensation, compensation, and relapse are distinguished against the background of hyperthyroidism treatment, which show the effectiveness of the treatment [17].

It should be noted that the terms "thyrotoxicosis" and "hyperthyroidism" cannot be completely synonymous. Thyrotoxicosis is a clinical laboratory syndrome, a pathological reaction of the body occurs due to an increase in the amount of thyroid hormones in organs and tissues. Hyperthyroidism (hyperthyroidism) can be a partial thyrotoxicosis condition, increased QB secretion is manifested by a physiological and pathological increase in the amount of thyroid hormones. For example, thyrotoxicosis without hyperthyroidism from excessive intake of thyroid hormone drugs, and hyperthyroidism without thyrotoxicosis in pregnancy is of some importance [22].

In the second half of pregnancy, the pulse is 160/min. and children born with threotoxicosis are often congenitally disabled, while healthy children are born with a small weight of 2-2.5 kg. At the same time, microcephaly and expansion of cerebral ventricles, exophthalmos are observed.

In half of the children born with congenital thyrotoxicosis, palpation of the scrotum and compression of the upper respiratory tract is observed. They have increased excitability, increased activity in their movements, increased sweating and increased appetite. Many babies lose weight due to vomiting and diarrhea, and it is difficult to develop the sucking reflex. At the same time, hepatosplenomegaly and jaundice are observed. In adults, the classic Nikolaev triad (goiter, tachycardia, exophthalmos) studied by Karl Bazedov occurs in 50% of patients. DTG is often characterized by increased appetite, weight loss, sweating, tachycardia and palpitations, nervousness, tremors of hands, general and muscle weakness, rapid fatigue and a number of other symptoms (Table 3) [16].

Table 3. Clinical presentation of diffuse toxic goiter

Organandsystem	**Signs**
Metabolism	Acceleration of basic metabolism with the development of catabolic syndrome: loss of body weight, poor heat transfer, excessive sweating, etc.
Eyes	Fistula dilation, upper eyelid retraction, exophthalmos, conjunctival edema, ophthalmoplegia
Heart-blood vessel system	Sustained sinus tachycardia, extrasystole, atrial fibrillation, increased systolic and diastolic blood pressure, increased heart tone, extracardiac and cardiac murmurs, myocardiodystrophy, heart failure
Nervoussystem	Emotional lability, anxiety, sleep disorders, rapid weight loss syndrome, small tremor, hyperreflexia
Skinand nail	Hyperhidrosis, thinning and loss of hair and cuticles, pretibial plate migration, onycholysis (nail plate migration), scar spots, vitiligo spot, hyperpigmentation of palmar layers.
Gastrointestinaltract	Increased appetite, diarrhea, toxic damage to the liver (recurrent enlargement, pain, jaundice)
Reproductivesystem	Menstrual cycle disorders (up to amenorrhea), in men - gynecomastia (optional), impotence, decreased fertility
Endocrinesystem	Carbohydrate intolerance, adrenal insufficiency (relative)
Locomotorapparatus	Myopathy, muscle atrophy, osteopenia/osteoporosis, increased blood alkaline phosphatase and calcium
Respiratorysystem	Increasedbreathingrate
Thermoregulation	Subfebrile

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Pretibial myxedema-associated immunopathy with DTG	One or two clear dark red-blue lines on the medial anterior surface of the calf, hypoxia. Patients with ophthalmopathy are usually registered.
Thyroid acropathy – combined immunopathy with DTG	In men, there is thickening of the phalanx of the fingers, which causes swelling of the dense tissue phalanx and new formation of periosteal bone tissue.

DTG patients are characterized by a warm and moist palm, soft and tender skin, small tremors of the tips of the fingers extended forward (Marie's symptom), tremors of the eyelids that are not fully closed (Rosenbach's syndrome). Patients are mostly women and look young for their age.

In most DTG patients, palpation of the thyroid gland reveals an enlarged thyroid gland. In this, not only the size of the QB is determined, but also its surface, consistency, uniformity, mobility and pain. Factors such as obesity, "short" neck, neck musculature expressed in men make it difficult to palpate QB. Palpation is performed with the patient's head slightly bent forward [23]. UTT is one of the most reliable thyroid screening methods. Examination of the QB with UTT is carried out in the position of the patient's back with a small roller under the shoulder. The UTT report provides information on the location, linear size, structure, focal changes and volumetric changes (about their location, size and exostructure), as well as the exostructure and size of regional lymph nodes.

In the laboratory examination, it was found that the level of TTG in the blood was reduced in DTG. At the same time, T3 and T4 increase, in the "destructive" form of thyrotoxicosis, a significant predominance of T4 over T3, characteristic of healthy people, is preserved [17].

The rate of anemia in patients with DTG is high [3-5]. Anemia is caused by metabolic disorders in DTG [20, 21]. As DTG severity increases, anemia severity also increases [1]. In some patients, iron metabolism is intact and anemia is normochromic [7, 10, 12, 18, 19]. As the level of thyrotoxicosis increases, the osmotic resistance of erythrocytes decreases, as a result, anemia increases [6-9,13].

The development of thyrotoxicosis in DTG leads to an increase in endothelial dysfunction and thrombotic complications [2]. Disruption of protein metabolism in DTG is associated with increased catabolism [11, 14, 15].

Treatment of DTG patients can be divided into two main stages.

- Stage I - elimination of thyrotoxicosis and its manifestations. Thyrostatic therapy with β-blockers and drugs from the thionamide group is usually recommended for this.
- Stage II - elimination of the disease. One of the ways to achieve long-term remission of DTG is determined: continuation of conservative therapy with thyrostatics in a supportive dose or radical treatment of QB (surgical treatment or use of radioactive iodine). In many patients, the first stage is 3-4 weeks, and the second stage is 12-18 months after stopping the drug [16].

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