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Diffuse Toxic Goiter

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Annotation: Diffuse toxic goiter (synonyms: Graves' disease, Basedow's disease, hyperthyroidism, Perry's disease, Flajani disease) is an autoimmune disease caused by excessive secretion of thyroid hormones by diffuse thyroid tissue, which leads to poisoning with these hormones - thyrotoxicosis. The disease is characterized by the presence of autoantibodies that bind to thyroid-stimulating hormone receptors (TSH-R) located on the surface of thyroid cells. As a result of stimulation of the receptors, there is increased production of thyroid hormones T3 and T4, which leads to the development of symptoms of hyperthyroidism. It is more often observed in women.

Keywords: thyrotoxicosis, thyroxine, triiodothyronine, exophthalmos, glaucoma.

Diffuse toxic goiter is an autoimmune disease of the thyroid gland that develops in genetically predisposed individuals, characterized by diffuse enlargement and hyperfunction of the thyroid gland, as well as toxic damage to organs and systems (thyrotoxicosis) as a result of excessive production of thyroid hormones. The disease is more common in women aged 20-50 years (the ratio of women to men is 5:1.7:1).

ETIOLOGY AND PATHOGENESIS:

Diffuse toxic goiter is currently considered a hereditary autoimmune disease and is transmitted from mother to child in a multifactorial (polygenic) way. The role of the genetic factor in the development of DTB has been proven:

- occurrence of the disease in several people in one family;
- Detection of HLA antigens and antithyroid antibodies in close relatives of a patient with DTB;
- If one of the twins has DTB, the probability of the other twin developing the disease is 60%.

DTB is often accompanied by HLA-B8, DR3, DW3 antigens. The HLA-V8 antigen increases the risk of developing LTB by 2.6 times, the HLA-DR3 and HLA-DR3 antigens by 5.9 and 3.9 times.

Mental trauma, infectious and inflammatory diseases, traumatic brain injury, diseases of the nose and throat are the driving factors in the development of DTB.

The main pathogenetic factors of DTB

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1. Congenital deficiency of T-suppressor function of lymphocytes with development of autoimmune reactions to thyroid antigens.
2. Expression of HLA-DR antigens on the surface of thyrocytes. Induction of this expression occurs under the influence of gamma interferon and interleukins produced by leukocytes. As a result, thyrocytes become antigen-recognizing cells, and T-lymphocytes perceive them as foreign.
3. Formation of prohibited ("forbidden") clones of T-lymphocytes (according to Wolpe's theory) under conditions of insufficiency of T-suppressor function of lymphocytes, which behave as T-helper lymphocytes and produce antibodies against components of the thyroid gland. Currently, thyrotropin is produced on the surface of thyrocytes. This population of antibodies is

heterogeneous and represents a mixture of antibodies to thyrotropin receptors and agonists, antagonists and nonagonists of these receptors (Drexhage, 1996). In the pathogenetic development of DTB, 2 types of antibodies are distinguished in this group - long-acting thyrostimulant (LATS factor) and thyroid growth-stimulating immunoglobulins (OSI, RSI-growth-stimulating immunoglobulins). LATS factor-G belongs to the group of immunoglobulins, with a molecular weight of 150,000 DU. Interacts with thyrotropin receptors and stimulates the thyroid gland function. In this case, the production of thyroid hormones T3 and T4 increases sharply and the clinical picture of toxic goiter (thyrotoxicosis) develops. OSI (RSI) interacts with IFR-1 or S-somatomedin receptors, causing an increase in the thyroid gland. In DTB, antibodies to other thyroid antigens are also detected: to thyroglobulin, to the colloid antigen, to the microsomal fraction and the nuclear component.

4. Increased sensitivity of the cardiovascular system to the effects of catecholamines as a result of excessive production of thyroid hormones. This leads to tachycardia, increased blood pressure and other changes in the cardiovascular system.
5. Increased conversion of thyroxine to triiodothyronine in the periphery. Since triiodothyronine is more biologically active than thyroxine, it deepens the clinical picture of thyrotoxicosis.
6. Development of adrenal insufficiency due to increased catabolism of glucocorticoids.

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PATHOGENESIS OF OPHTHALMOPATHY:

Ophthalmopathy is one of the most important clinical symptoms of DTB. Ophthalmopathy occurs as a result of autoimmune damage to the extraocular muscles that move the eye. The eye muscle antigen is a thyrotropin receptor located in endomysial fibroblasts. The interaction of antibodies with antigens leads to an increase in the production of glycosaminoglycans and other components of connective tissue in the retrobulbar cell, which leads to the development of edema and then fibrosis. A major role in the development of ophthalmopathy is played by the formation of a forbidden cytotoxic clone of T-lymphocytes, damaging the retrobulbar cell.

CLINICAL PICTURE:

Main complaints of patients

- strong mental agitation, sensitivity, anxiety, overexertion, inability to concentrate;
- feeling of pressure in the neck; difficulty swallowing;
- constant rapid heartbeat;
- constant diffuse sweating;
- constant feeling of warmth;
- hand tremors, interfere with work, writing; sometimes patients say that their mood has changed;
- progressive weight loss despite a good appetite;
- general muscle weakness;
- strabismus, tears, photophobia;

Objective:

- patients move too much;
- emotional lability, sudden mood swings, hasty speech;
- diffuse enlargement of the thyroid gland of various sizes; The thyroid gland is soft, sometimes hard-elastic, systolic murmur is rarely heard over it. The severity of the disease does not depend on

the size of the goiter. Even when the thyroid gland is not very large, severe thyrotoxicosis can develop.

- the skin is soft (thin, elastic), hot, moist, hyperemic. The fingers and heels are hot, unlike in NCD; formation of skin pigmentation as a manifestation of adrenal insufficiency. Some patients have pretibial myxedema - the skin in the knee and heel area is compacted, thickened, brownish-carrot in color, coarse hair growth on the skin of the knee - a symptom of "pig skin". Pretibial myxedema develops as a result of excessive accumulation of mucopolysaccharides in the skin.
- weight loss due to the catabolic and lipolytic action of thyroid hormones;
- muscles atrophy, their strength and tone decrease.

Muscle weakness (thyrotoxic myopathy) is associated with the catabolic action of thyroid hormones and can be generalized and local. This is often observed in the muscles of the thigh and trunk. In rare cases, short-term muscle paralysis is observed. When the euthyroid state is restored, muscle weakness disappears.

The changes observed in the eye and surrounding tissues are specific in nature, the following symptoms are observed:

- shine of the eyes;
- Graefe's symptom: formation of sclera between the upper eyelid and the colored membrane when moving an object from top to bottom;
- Kocher's symptom: formation of sclera between the upper eyelid and the colored membrane when moving an object from bottom to top;
- Dalrymple's symptom: formation of sclera between the upper eyelid and the colored membrane when moving an object horizontally;
- Rosenbach's symptom: trembling of the eyelids with closed eyes;
- Geoffroy's symptom: decreased opening and closing of the eyes;
- Moebius's symptom: decreased convergence due to changes in the internal rectus muscle;
- Stasinsky's, or "red intersection" symptom: the vessels of the sclera seem injected *expansion of the ocular field, reminiscent of a state of surprised gaze;

niche The movement of these veins up, down, left and right across the retina resembles a red cross-section with the pupil in the middle. Ophthalmopathy is one of the severe complications of thyrotoxicosis and is characterized by metabolic disorders of extraocular tissues, development of exophthalmos, and dysfunction of the oculomotor muscles. Severe progressive ophthalmopathy leads to loss of vision. Ophthalmopathy is often bilateral, but at the onset of the disease it may be unilateral. Symptoms of ophthalmopathy:

- exophthalmos;
- bulging eyelids and flattening of the palpebro-orbital fold;
- conjunctivitis (swelling and redness of the conjunctiva, lacrimation, photophobia);
- dysfunction of the oculomotor muscles (dysfunction of the eyeball to the sides);
- dysfunction of the eyelids, dry cornea with pronounced exophthalmos, trophic changes and development of keratitis. The addition of infection causes a purulent process in the eye, resulting in sympathetic inflammation of the second eye;
- Increased intraocular pressure (glaucoma) is also observed with partial exophthalmos with subsequent atrophy of the optic nerve.

There are 4 levels of ophthalmopathy:

Grade I - partial exophthalmos, eyelid edema;

Grade II - I-degree + minor changes in the conjunctiva + partial dysfunction of the oculomotor muscles;

III degree - pronounced exophthalmos + pronounced conjunctivitis + obvious changes in the muscles of eye movement + mild corneal damage + initial signs of optic nerve atrophy;

IV degree - obvious trophic changes in the conjunctiva, cornea and optic nerve with the risk of loss of the eye and vision.

THYROTOXICOSIS SEVERITY (V.G. Baranov)

Mild level

- symptoms of thyrotoxicosis are less pronounced, neurotic symptoms and sensitivity prevail;
- weight loss up to 10%;
- tachycardia up to 100 beats per minute, blood pressure and heart boundaries unchanged;
- no symptoms of ophthalmopathy;
- performance is preserved or slightly lost.

Moderate level of complexity

- symptoms of thyrotoxicosis are clearly expressed;
- body weight decreased from 10% to 20%;
- tachycardia from 100 to 120 beats per minute; the borders of the heart are shifted to the left, systolic blood pressure is higher by 130-150 mm.cm; diastolic blood pressure is normal or slightly reduced;
- obvious ophthalmopathy;
- decreased performance.

Severity level

- all symptoms of thyrotoxicosis are clearly expressed, there are signs of severe damage to internal organs (liver, heart);
- body weight has decreased by more than 20%; cachexia;
- tachycardia over 120 beats per 1 minute, the borders of the heart are dilated, often fluctuating arrhythmia and circulatory failure, systolic blood pressure 150-160 mm.sym.ust, diastolic blood pressure is significantly reduced;
- ophthalmopathy is clearly expressed;
- obvious changes in the nervous system;
- complete loss of working capacity.

CONCLUSION

Diffuse toxic goiter (DTG) is considered a life-threatening disease because it results in irreversible changes in all organs and tissues of the human body, especially in the heart, blood vessels, nervous system and skeleton of the patient.

Used literature.

1. Ra'no, D. (2024). SEMIZLIK. *AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI*, *3*(3), 140-147.
2. Нарзуллаева, У. Р., Самиева, Г. У., & Пардаева, З. С. (2020). Pathogenetic aspects of verified risk factors such as arterial hypertension and dyslipidemia in the development of chronic heart failure. American Journal of Medicine and Medical Sciences, 10(10), 776-779.
3. Жураева, Д. Н., & Нарзулаева, У. Р. (2020). Эркак ва аёлларда уч шохли нерв невралгияси кечишининг параклиник хусусиятлари. ЖУРНАЛ НЕВРОЛОГИИ И НЕЙРОХИРУРГИЧЕСКИХ ИССЛЕДОВАНИЙ, 1(1).
4. Narzulyeva, U., & Ismoilova, N. (2023). DETECTION OF EATING BEHAVIOR DISORDERS IN STUDENTS BEFORE THE EXAM USING THE DEBQ QUESTIONNAIRE. Наука и инновация, 1(15), 112-114.
5. Narzulaeva, U. (2023). PATHOGENETIC MECHANISMS OF MICROCIRCULATION DISORDERS. International Bulletin of Medical Sciences and Clinical Research, 3(10), 60–65. Retrieved from https://researchcitations.com/index.php/ibmscr/article/view/2811
6. Narzulaeva Umida Rakhmatulloevna and Rakhmatova Fotima Ulugbekovna, "PATHOGENETIC MECHANISMS OF DISORDERS IN THE HEMOSTASIS SYSTEM OBSERVED IN PATIENTS INFECTED WITH COVID-19", IEJRD - International Multidisciplinary Journal, vol. 7, no. ICMEI, p. 3, Feb. 2023.
7. Narzulaeva, U. (2023). PATHOGENETIC SIGNIFICANCE OF HYPERLIPIDEMIA IN THE CLINICAL COURSE OF ARTERIAL HYPERTENSION. International Bulletin of Medical Sciences and Clinical Research, 3(11), 86-91.
8. Narzulaeva, U. (2023). PATHOGENETIC SIGNIFICANCE OF HYPERLIPIDEMIA IN THE CLINICAL COURSE OF ARTERIAL HYPERTENSION. International Bulletin of Medical Sciences and Clinical Research, 3(11), 86-91.
9. Нарзуллаева, У., Самиева, Г., & Пардаева, З. (2022). ПАТОФИЗИОЛОГИЯ РЕПЕРФУЗИОННОГО ПОВРЕЖДЕНИЯ МИОКАРДА. Журнал вестник врача, 1(2), 155–158. https://doi.org/10.38095/2181-466X-2020942-154-157
10. Самиева, Г., Нарзулаева, У., & Самиев, У. (2023). Течение артериальной гипертензии у жителей засушливого региона. Каталог монографий, 1(1), 1–108. извлечено от https://inlibrary.uz/index.php/monographs/article/view/27456
11. Nematilloyevna, X. M., & Salohiddinovna, X. Y. (2024). LOTIN TILI VA TIBBIYOT TERMINOLOGIYASINI O'QITISHDA TALABALARDA MOTIVATSIYANI KUCHAYTIRISH YO'LLARI. *Modern education and development*, *10*(1), 38-48.
12. Nematilloyevna, X. M., & Salohiddinovna, X. Y. (2024). LOTIN TILI SIFATLARI VA DARAJALARI YASALISHINING MUHIM XUSUSIYATLARI. *Modern education and development*, *10*(1), 16-26.
13. Nematilloyevna, X. M., & Salohiddinovna, X. Y. (2024). FARMATSEVTIKADA DORI PREPARATLARI NOMLARIDA MA'NOLI BO'LAKLARNING QO'LLANILISHI. *Modern education and development*, *10*(1), 49-59.
14. Xalimova, Y. S. (2024). Morphology of the Testes in the Detection of Infertility. *Journal of Science in Medicine and Life*, *2*(6), 83-88.
15. Хафизова, М. Н., & Халимова, Ю. С. (2024). ИСПОЛЬЗОВАНИЕ ЧАСТОТНЫХ ОТРЕЗКОВ В НАИМЕНОВАНИЯХ ЛЕКАРСТВЕННЫХ ПРЕПАРАТОВ В ФАРМАЦЕВТИКЕ. *Modern education and development*, *10*(1), 310-321.

16. Хафизова, М. Н., & Халимова, Ю. С. (2024). МОТИВАЦИОННЫЕ МЕТОДЫ ПРИ ОБУЧЕНИИ ЛАТЫНИ И МЕДИЦИНСКОЙ ТЕРМИНОЛОГИИ. *Modern education and development*, *10*(1), 299-309.
17. Халимова, Ю. С., & Хафизова, М. Н. (2024). ОСОБЕННОСТИ СОЗРЕВАНИЕ И ФУНКЦИОНИРОВАНИЕ ЯИЧНИКОВ. *Modern education and development*, *10*(1), 337-347.
18. Saloxiddinovna, X. Y., & Ne'matillaevna, X. M. (2024). FEATURES OF THE STRUCTURE OF THE REPRODUCTIVE ORGANS OF THE FEMALE BODY. *Modern education and development*, *10*(1), 322-336.
19. Nematilloyevna, X. M., & Salohiddinovna, X. Y. (2024). LOTIN PREFIKSLARI ANATOMIK TERMINLAR YASALISHIDA ASOSIY KOMPONENT SIFATIDA. *Modern education and development*, *10*(1), 27-37.
20. Бакиева, М. Ш., Рустамова, Ш. Р., Рахмонов, Т. О., Шарипова, Н. Н., & Мухитдинова, Х. С. (2022). Гипотензивное действие алкалоида бензоилгетератизина на функциональную активность гладкомышечных клеток аорты крысы. *AcademicResearchJournalImpactFactor*, *7*.
21. Samixovna, M. K. (2024). MORPHOLOGICAL DATA OF THE ORGANS OF HEMATOPOIESIS AND HEMATOPOIESIS. Лучшие интеллектуальные исследования, 14(5), 66-74.
22. Samixovna, M. K. (2024). Morphologic Changes in Red Blood Cells. ResearchJournalofTraumaandDisabilityStudies, 3(3), 178-186.
23. Samixovna, M. K. (2024). MORPHOLOGICAL FEATURES OF POSTPARTUM CHANGES IN UTERINE MEMBRANES. SCIENTIFIC JOURNAL OF APPLIED AND MEDICAL SCIENCES, 3(4), 277-283.
24. Samixovna, M. K. (2024). Current Data on Morphological and Functional Characteristics of the Thyroid Gland in Age Groups. JournalofScienceinMedicineandLife, 2(5), 77-83.
25. Samixovna, M. X. (2024). AYOL ORGANIZMI REPRODUKTIV ORGANLARINING RIVOJLANISH XUSUSIYATLARI. *Modern education and development*, *10*(1), 226-238.
26. Samixovna, M. X. (2024). OITS KASALLIGI, TA'RIFI VA KASALLIKNING KELIB CHIQISH SABABLARI. *Modern education and development*, *10*(1), 239-254.
27. Мухиддинова, Х. С. (2024). РАЗВИТИЕ ЯИЧНИКОВ, ИХ МОРФОЛОГИЯ И ОСОБЕННОСТИ ФУНКЦИОНИРОВАНИЕ. *Modern education and development*, *10*(1), 255-265.
28. Мухитдинова, Х. С. (2024). СОВРЕМЕННЫЕ ВЗГЛЯДЫ НА РАЗВИТИЕ БАКТЕРИАЛЬНОГО ВАГИНОЗА У ЖЕНЩИН ФЕРТИЛЬНОГО ВОЗРАСТА. *Modern education and development*, *10*(1), 202-212.
29. Мухитдинова, Х. С. (2024). ЗАБОЛЕВАЕМОСТЬ СПИДОМ, МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ БОЛЕЗНИ. *Modern education and development*, *10*(1), 213-125.
30. Эргашева, Г. Т. (2024). ОСЛОЖНЕНИЯ САХАРНОГО ДИАБЕТА 2 ТИПА ХАРАКТЕРНЫ ДЛЯ КОГНИТИВНЫХ НАРУШЕНИЙ. *TADQIQOTLAR. UZ*, *30*(3), 112-119.
31. Tokhirovna, E. G. Studying the Causes of the Relationship between Type 2 Diabetes and Obesity. *Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN*, 2456-6470.
32. Эргашева, Г. Т. (2024). ФАКТОРЫ РИСКА РАЗВИТИЯ САХАРНОГО ДИАБЕТА 2 ТИПА. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *36*(5), 70-74.
33. Tokhirovna, E. G. (2024). Risk factors for developing type 2 diabetes mellitus. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *36*(5), 64-69.

34. Toxirovna, E. G. (2024). GIPERPROLAKTINEMIYA KLINIK BELGILARI VA BEPUSHTLIKKA SABAB BO'LUVCHI OMILLAR. *Лучшие интеллектуальные исследования*, *14*(4), 168-175.

35. Toxirovna, E. G. (2024). QANDLI DIABET 2-TUR VA O'LIMNI KELTIRIB CHIQARUVCHI SABABLAR. *Лучшие интеллектуальные исследования*, *14*(4), 86-93.

36. Tog'aydullayeva, D. D. (2024). Embrional Davrda Gemopoez Va Unda Jigar Va Taloqning Roli. *Journal of Science in Medicine and Life*, *2*(6), 132-134.

37. Tog'aydullayeva, D. D. (2024). Occurrence of Combination Diseases in Ischemic Heart Disease and Metabolic Syndrome and their Diagnosis. *Journal of Science in Medicine and Life*, *2*(6), 126-131.

38. Tog'aydullayeva, D. D. (2024). Occurrence of Combination Diseases in Ischemic Heart Disease and Metabolic Syndrome and their Diagnosis. *Journal of Science in Medicine and Life*, *2*(6), 126-131.