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DIFFUSE TOXIC GOITER: ETIOLOGY, PATHOGENESIS, CLINICAL MANIFESTATIONS, AND SYSTEMIC EFFECTS

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Abstract: Diffuse toxic goiter (DTG), also known as Graves' disease, is an autoimmune disorder of the thyroid gland characterized by diffuse enlargement of the gland and excessive production of thyroid hormones. This condition leads to thyrotoxicosis, which affects multiple organs and systems. DTG predominantly occurs in women of working age and has a strong genetic and immunological background. The present article reviews the etiology, pathogenesis, clinical features, ophthalmopathy, systemic involvement, and severity classification of diffuse toxic goiter.

Keywords: diffuse toxic goiter, Graves' disease, thyrotoxicosis, autoimmune thyroid disease, ophthalmopathyIntroduction

Diffuse toxic goiter is one of the most common autoimmune endocrine disorders. It is characterized by hyperfunction of the thyroid gland due to the production of autoantibodies that stimulate thyroid hormone synthesis. Excessive secretion of triiodothyronine (T3) and thyroxine (T4) results in thyrotoxicosis, which leads to significant metabolic, cardiovascular, neurological, and ocular complications. Early recognition of the disease is essential to prevent severe systemic damage.

Etiology and Pathogenesis

Diffuse toxic goiter is considered a multifactorial autoimmune disease with a strong genetic predisposition. The role of heredity is confirmed by familial clustering, association with specific HLA antigens (HLA-B8, DR3, DW3), and a high concordance rate among monozygotic twins.

Triggering factors such as psychological stress, infectious and inflammatory diseases, head trauma, and disorders of the upper respiratory tract contribute to disease onset in genetically predisposed individuals. The central pathogenic mechanism involves a congenital defect in T-suppressor lymphocyte function, leading to loss of immune tolerance.

Autoantibodies directed against thyroid-stimulating hormone (TSH) receptors play a key role in disease development. These antibodies stimulate thyroid hormone synthesis independently of pituitary regulation, resulting in persistent hyperthyroidism. Increased peripheral conversion of T4 to the more biologically active T3 further aggravates thyrotoxicosis. Additionally, accelerated glucocorticoid metabolism may lead to relative adrenal insufficiency.

Pathogenesis of Ophthalmopathy

Ophthalmopathy is a characteristic and clinically significant manifestation of diffuse toxic goiter. It develops due to autoimmune inflammation of extraocular muscles and retrobulbar connective tissue. TSH receptor expression on orbital fibroblasts leads to antibody-mediated activation, resulting in accumulation of glycosaminoglycans, tissue edema, and subsequent fibrosis.

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Progressive ophthalmopathy may cause exophthalmos, impaired ocular motility, corneal damage, and, in severe cases, optic nerve compression with vision loss.

Clinical Manifestations

Patients with diffuse toxic goiter typically present with neuropsychiatric symptoms such as irritability, emotional lability, anxiety, restlessness, and impaired concentration. Cardiovascular symptoms include palpitations, persistent tachycardia, and arrhythmias. Heat intolerance, excessive sweating, hand tremor, and unexplained weight loss despite increased appetite are common.

Physical examination reveals diffuse enlargement of the thyroid gland, warm and moist skin, muscle weakness, and signs of thyrotoxic myopathy. Ocular findings include eyelid retraction, conjunctival edema, excessive tearing, photophobia, and exophthalmos.

Systemic Involvement

Diffuse toxic goiter affects multiple organ systems. The cardiovascular system is involved in almost all patients, with tachycardia, systolic hypertension, atrial fibrillation, and heart failure developing in severe cases. The nervous system exhibits tremors, hyperreflexia, and increased excitability.

Gastrointestinal manifestations include increased intestinal motility and diarrhea. Hepatic involvement may lead to fatty degeneration and, in advanced cases, cirrhosis. Long-standing thyrotoxicosis causes bone demineralization and osteoporosis due to enhanced calcium resorption. In severe disease, signs of adrenal insufficiency may appear.

Severity Classification of Thyrotoxicosis

Based on clinical presentation, diffuse toxic goiter is classified into mild, moderate, and severe forms.

Mild form is characterized by minimal symptoms and preserved working capacity.

Moderate form presents with evident thyrotoxicosis, weight loss, and cardiovascular changes.

Severe form is marked by pronounced systemic involvement, cachexia, organ dysfunction, and complete loss of working ability.

Special Clinical Forms

In elderly patients, diffuse toxic goiter often presents atypically, with predominant muscle weakness, weight loss, and cardiac arrhythmias, while classical hyperexcitability may be absent. T3-thyrotoxicosis represents a variant in which triiodothyronine levels are elevated despite normal thyroxine concentrations. In men, the disease tends to progress more rapidly and is often associated with severe ophthalmopathy and resistance to conservative therapy.

Conclusion

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Diffuse toxic goiter is a complex autoimmune disease with widespread systemic effects. Its pathogenesis involves genetic susceptibility, immune dysregulation, and autoantibody-mediated thyroid stimulation. Due to its multisystem involvement and potential for severe complications, early diagnosis and timely treatment are crucial to improving prognosis and quality of life in affected patients.

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