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An Overview of Grave's Disease

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Editorial

An autoimmune condition that affects the thyroid is known as Graves' disease, commonly referred to as toxic diffuse goitre. It usually causes hyperthyroidism and is one of its main causes. It frequently causes an enlarged thyroid as well. Irritability, muscle weakness, trouble sleeping, a rapid pulse, a poor tolerance for heat, diarrhoea, and accidental weight loss are some of the signs and symptoms of hyperthyroidism. Pretibial myxedema, a thickening of the skin on the shins, and Graves' ophthalmopathy, which results in protruding eyes, are two additional symptoms that may be present. Between 25 and 80 percent of those who have the disease experience eye issues [1].

Although the precise aetiology of the illness is unknown, it is thought to be a result of a combination of hereditary and environmental factors. If a person has a family member who has the illness, they are more likely to be impacted. There is a 30% probability that the other twin will also have the condition if one twin is Stress, whether it is physical or emotional, infection, or giving birth, can cause disease to manifest. People who have rheumatoid arthritis and type1 diabetes are more susceptible to being affected. Smoking raises the chance of getting sick and may make eye problems worse. The condition is brought on by a thyroid-stimulating immunoglobulin (TSI) antibody, which functions similarly to thyroid stimulating hormone (TSH). The thyroid gland produces too many thyroid hormones as a result of these TSI antibodies. Blood tests and radioiodine uptake may be used to confirm the diagnosis when the diagnosis is suspected based on the symptoms. Blood tests typically reveal elevated T3 and T4, low TSH, elevated radioiodine uptake throughout the thyroid, and TSI antibodies. [2].

Thyroid surgery, medicine, and radioiodine therapy are the three available treatments. Iodine-131 is consumed as part of radioiodine therapy, where it is concentrated in the thyroid and kills it over the course of several weeks to months. Synthetic thyroid hormones are used to treat the ensuing hypothyroidism. While other treatments are working, people may have some symptom relief from medications such beta blockers or methimazole, which are antithyroid drugs. Another alternative is to have the thyroid surgically removed. Additional treatments may be necessary for eye issues. About 0.5 percent of males and 3 percent of females will eventually develop Graves' disease. Women experience it almost 7.5 times more frequently than men do. Although it can start at any age, it frequently begins between the ages of 40 and 60. In the United States, it is the most typical cause of hyperthyroidism (about 50 to 80 percent of cases). The illness bears Robert Graves' name, an Irish surgeon who first reported it in 1835.There are also other earlier descriptions [3].

The chief exceptions to this rule are Graves' ophthalmopathy, goitre, and pretibial myxedema. Virtually all of the signs and symptoms of Graves' disease are caused by the direct and indirect effects of hyperthyroidism (which are caused by the autoimmune processes of the disease). Insomnia, hand tremor, hyperactivity, hair loss, excessive sweating, oligomenorrhea, itching, heat intolerance, weight loss despite increased appetite, diarrhoea, frequent urination, palpitations, periodic partial muscle weakness or paralysis in people, especially those of Asian descent, and warmth and moistness of the skin are the main symptoms of the resulting hyperthyroidism. A diffusely enlarged (typically symmetric), nontender thyroid, lid lag, excessive lacrimation from Graves' ophthalmopathy, heart arrhythmias like sinus tachycardia, atrial fibrillation, and premature ventricular contractions are additional symptoms that may be discovered during a physical examination. Although the specific aetiology is unknown, it is thought to be a result of both hereditary and environmental factors. There is a theoretical mechanism by which exposure to extreme stressors and high levels of subsequent distress, such as PTSD (Post Traumatic Stress Disorder), could increase the risk of autoimmune disease and cause an aggravation of the autoimmune response that results in Graves' disease, but more conclusive clinical evidence is required [4].

There is a genetic propensity for Graves' disease, and certain persons are more likely to acquire TSH receptor activating antibodies as a result of this predisposition. Human leukocyte antigen DR, particularly DR3, seems to be important. No obvious genetic abnormality with a single-gene aetiology has been identified as of yet. Thyroglobulin, thyrotropin receptor, protein tyrosine phosphatase nonreceptor type 22 (PTPN22), and cytotoxic T-lymphocyte-associated antigen 4 are a few of the genes thought to be involved. Antigenic mimicry, a phenomena that can result from viral or bacterial infections, is a possibility because Graves' disease is an autoimmune condition that manifests unexpectedly, frequently later in life. Because of its structural resemblance to the human thyrotropin receptor, the bacterium Yersinia enterocolitica has been linked to the development of thyroid autoimmunity in people who are genetically predisposed to it. It was hypothesised in the Y. enterocolitica and Graves' illness might be related. The function of Y. enterocolitica has come under scrutiny more lately. Another possible cause is the Epstein-Barr virus. An autoimmune condition known as Graves' disease occurs when the body makes antibodies that are directed against the thyroid-stimulating hormone receptor, a protein that is present in all healthy people. (Antibodies to the thyroid hormones T3 and T4 as well as to thyroglobulin may also be generated.

These antibodies bind to the TSHr and persistently stimulate it, which results in hyperthyroidism. The thyroid gland's thyroid follicular cells, which are the cells that generate thyroid hormone, express the TSH receptor, and continuous stimulation leads to abnormally high levels of T3 and T4 synthesis. This leads to the clinical signs and symptoms of hyperthyroidism as well as the goitre, which is an enlargement of the thyroid gland. The idea that the thyroid gland and the extraocular muscles share a common antigen that is recognised by the antibodies has been put out as an explanation for the infiltrative exophthalmos that is frequently observed. The extraocular muscles would expand as a result of antibodies attaching to them. The infiltration of antibodies under the skin, which results in an inflammatory response and subsequent fibrous plaques, has been used to explain the "orange peel" skin [5].

Conflict of Interest

None.

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