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Commentary

An Update on the Management of Graves Thyroidal and Extra Thyroidal Disease

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DESCRIPTION

This article provides an update on the diagnosis and therapy of thyroidal and extra thyroidal Graves' disease patients (GD). The goal is to avoid repeating known and broad information. As a result, this update emphasizes contemporary breakthroughs, such as the serological accurate and quick detection of GD, as well as its globally recognized preferred medical therapy. It describes the Insulin-like Growth Factor-1 Receptor (IGF-1R) as a new auto antigen in Graves' disease and Graves Orbitopathy (GO).

In addition, this brief commentary provides innovative, more disease-specific therapies for both GD and GO, with a particular emphasis on the recently FDA-approved IGF-1R blocking monoclonal antibody teprotumumab. Finally, the treatment of GD and GO during the COVID-19 pandemic is reviewed.

The prevalence of GD in the general population is estimated to be 1% to 1.5%, with an annual incidence of 20 to 30 new cases per 100,000 people. African Americans are more likely to be affected. Familial clustering, bad life events, excessive iodine consumption, and smoking are among genetic and environmental variables that lead to GD. GD is a systemic autoimmune illness caused by circulating autoantibodies (Abs) that bind to the Thyrotrophic Receptor (TSH-R), causing thyroid hormone synthesis and release, Thymocyte proliferation, and thyroid gland hypertrophy. In individuals suspected of having hyperthyroidism, the stimulatory TSH-R-Ab (TSAb) is the causal agent in GD, with concomitant signs and symptoms. When in interaction with the TSH-R, TSAb directly raises intracellular cyclic adenosine 5′ -monophosphate, which may be measured in a chemiluminescent bioassay.

Furthermore, TSAbs cause oxidative stress in GD in a direct and particular manner.

SUBCLINICAL GRAVES' DISEASE

Endogenous subclinical GD and totally suppressed serum TSH have been linked to cardiovascular morbidity and death. In the next three years, the incidence of progression from subclinical to overt hyperthyroidism was 30%, especially in individuals with chronic TSH-R-Ab. As a result, therapy of subclinical GD is suggested in all GD patients, especially those over 65, and especially in those with totally suppressed blood TSH levels. ATDs are the initial line of therapy.

SYMPTOMATIC MANAGEMENT AND ANTICOAGULATION

Cardiac symptoms, including as sinus tachycardia, supraventricular arrhythmia, and atrial fibrillation in the elderly, are common in Graves's hyperthyroidism patients. As a result, in the early stages of GD, beta-adrenergic inhibition (e.g., propranolol or bisoprolol) is recommended. Propranolol at high dosages (160-200 mg) inhibits both thyroid hormone release and T4 deiodination in the peripheral. Anticoagulation is recommended for elderly individuals with supraventricular arrhythmias, particularly those over 65 years old with atrial fibrillation.

THYROIDECTOMY

Total TX is indicated in GD patients with (1) persistent hyperthyroidism and/or thyrotoxicosis after a first course of ATD, (2) suspected thyroid cancer, (3) a large thyroid gland (>50-60 mL) and compressive symptoms, (4) nodular goiters and active GO, (5) concurrent hyperparathyroidism, and (6) pregnancy (second trimester). Prior to TX, ATDs are essential, especially in severe hyperthyroidism, to minimize perioperative problems and uncontrolled thyroid dysfunction. Furthermore, a potassium iodide solution administered before to TX reduces blood supply to the thyroid and intraoperative blood loss. According to guidelines, a high-volume surgeon significantly decreases the failure rate to less than 1%, making TX a cost-effective therapy for GD. TX may cause laryngeal edema, recurrent laryngeal nerve damage, hypocalcaemia, hemorrhage, and Hypoparathyroidism. With competent, high-volume surgeons, the prevalence of the aforementioned problems remains low (153-155) and the chances of transitory hypocalcaemia and recurrent laryngeal nerve damage are 10%and 1%, respectively. Calcium replacement significantly reduces the incidence of perioperative hypocalcaemia. Finally, postoperative levothyroxine replacement is based on a body mass index or weight-adapted calculation.

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