

DIFFUSE TOXIC GOITER AND ITS TREATMENT

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Annotation

Goiter simply refers to the enlargement of the thyroid gland. It can be due to various causes with the dietary iodine deficiency being the most common cause worldwide. Diffuse toxic goiter consists of a diffusely enlarged, vascular gland with rubber-like consistency. Microscopically, the follicular cells are hypertrophic and hyperplastic with little colloid in them. Lymphocytes and plasma cells infiltrate into the gland and can ultimately aggregate into lymphoid follicles.

Key words: Iodine deficiency, autoimmune disorders, smoking, hereditary pattern, Radioactive Iodine Therapy, antithyroid drug, surgery.

The following are potential commonly seen causes of a goiter development: Iodine deficiency, autoimmune disorders, smoking, hereditary pattern, medications, like for example lithium, iodides, interferon-alpha among others, radiation therapy, and infections. Diffuse toxic goiter consists of a diffusely enlarged, vascular gland with rubber-like consistency. Microscopically, the follicular cells are hypertrophic and hyperplastic with little colloid in them. Lymphocytes and plasma cells infiltrate into the gland and can ultimately aggregate into lymphoid follicles. All cases of diffuse toxic goiter are not Graves disease and there may be various non-autoimmune causative processes although the majority of cases are autoimmune in nature. In Graves disease, antibodies are directed towards the thyroid-stimulating hormone receptor (TSHr) which is present on thyroid follicular cells. The chronic stimulation of these receptors results in the production of excess amounts of T3 and T4 hormones and causes the enlargement of the thyroid gland that eventually results in a goiter.

The treatment modalities for diffuse toxic goiter include:

Antithyroid Drugs (ATD)

1. The antithyroid drug options are propylthiouracil, methimazole, and carbimazole.
2. The American Thyroid Association (ATA) and American Association of Clinical Endocrinology (AACE) recommend methimazole as the preferred drug for Graves disease, except in patients with adverse reactions to the drug or women in



the first trimester of pregnancy. It is preferred over propylthiouracil due to better efficacy, longer half-life and duration of action, and the ease of using it as a once a day dosing.

3. There are two different regimens to administer ATDs. The first is titration, where the dose of ATD is tapered to the lowest possible dose when the euthyroid state is achieved. The second regimen is based on block-and-replace method, where a high dose of ATD is given adjunct with thyroxine replacement to ultimately maintain a euthyroid state.

4. A drawback of ATD therapy is the risk of recurrence, especially in the first year after stopping therapy. Studies reported a 50% to 55% risk of recurrence, with poor prognostic factors pointing to this direction including severe hyperthyroidism, large goiter, high T3: T4 ratios, persistently suppressed TSH, and high baseline concentrations of TRAb.

5. Rare but major side effects of ATD therapy include agranulocytosis, hepatotoxicity, and vasculitis.

Radioactive Iodine Therapy (I-31, RAI) RAI is the commonest modality to treat Graves disease in the United States and it is a very safe and effective form of treatment.

1. The absolute contraindications of this therapy include pregnancy, breastfeeding, and severe uncontrolled thyrotoxicosis.

2. It can be administered in liquid or capsule forms, and fixed-dose therapy is as effective as calculated dose therapy based on the volume of the gland, iodine uptake, etc.

3. Patients must discontinue all iodine-containing medication and be on iodine restricted diet to ensure effective uptake of RAI.

4. ATD therapy needs to be discontinued preferably a week before the use of RAI and it can be resumed a few days after administering RAI, if needed.

5. Potential side effects include the risk of developing hypothyroidism, and in rare occasions transient radiation-induced hyperthyroidism or worsening of thyroid-associated ophthalmopathy (TAO).

6. While hypothyroidism is actively screened at subsequent follow-ups, prednisone can be used to prevent the progression of mild TAO.

7. Patients have to be counseled about lifelong follow-up for either disease recurrence or for the development of hypothyroidism and they will benefit from prompt treatment if diagnosed with either abnormality.

Surgery



1. Thyroidectomy is the most successful form of therapy for a diffuse toxic goiter, with total thyroidectomy being more successful than sub-total thyroidectomy with equivalent side effects.
2. Due to the side effects associated with the use of general anesthesia, recurrent laryngeal nerve palsy, vascular complications, and hypothyroidism, surgical intervention is usually the last line of treatment.
3. It is preferred in patients who are unable to tolerate antithyroid medications or RAI treatment, or in patients with compressive symptoms due to a bigger size of the goiter.

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