Goiter

A goiter is a diffuse or nodular enlargement of the thyroid gland that does not result from an inflammatory or neoplastic process and is not associated with abnormal thyroid function. Endemic goiter is defined as thyroid enlargement that occurs in more than 10% of a population, and sporadic goiter is a result of environmental or genetic factors that do not affect the general population. Lithium treatment has been associated with the development of goiter.

More than 2.2 billion people worldwide have some form of iodine deficiency disorder. Twenty-nine percent of the world's population lives in a region that has iodine deficiency (primarily in Asia, Latin American, central Africa, and regions of Europe). Of those at risk, 655 million were known to have goiter. In Bangladesh, most patients of goiter are residing in the northern districts. The districts of middle and southern parts of the country are less prevalent of goiter.

As reported by the World Health Organization (WHO), the United Nations Children's Fund (UNICEF), and the International Council for the Control of Iodine Deficiency Disorders (ICCIDD), the absence of iodine deficiency (ie, median urine iodine >100 mg/dL) is associated with a goiter prevalence of less than 5%; mild iodine deficiency (ie, median urine iodine 50-99 mg/dL), with a goiter prevalence of 5-20%; moderate iodine deficiency (ie, median urine iodine 20-49 mg/dL), with a goiter prevalence of 20-30%; and severe iodine deficiency (ie, median urine iodine 20-49 mg/dL), with a goiter prevalence of greater than 30%.

Endemic goiters arising from iodine deficiency are associated with sometimes immense thyroid hypertrophy, hypothyroidism, and cretinism. Sporadic goiters are generally asymptomatic and found either by a clinician's physical examination or by the patient's observation of neck enlargement. Occasionally, the goiter may produce symptoms caused by pressure on anterior neck structures, including the trachea (wheezing, cough, globus hystericus [anterior neck pressure]), the esophagus (dysphagia), and the recurrent laryngeal nerve (hoarseness).

Diffuse and nodular goiter is more common in women than in men. According to the best estimate, the incidence of goiter in women is 1.2-4.3 times as great as that in men.

Sporadic goiter from dyshormonogenesis, a genetic error in proteins that are necessary for thyroid hormone synthesis, occurs during childhood. Endemic goiter due to iodine deficiency occurs during childhood, with the goiter's size increasing with age.

The thyroid gland usually grows outward because of its location anterior to the trachea. Occasionally, the thyroid wraps around and compresses the trachea and/or esophagus or extends inferiorly into the anterior mediastinum.

Determining whether the goiter has been present for many years and whether a change has occurred in the recent past is important. Recent or accelerated growth of a discrete nodule or thyroid lobe should raise the suspicion of malignancy. Goiters rarely are painful or grow quickly unless recent hemorrhage into a nodule has occurred.

Tracheal compression is generally asymptomatic until critical narrowing has occurred.

Patients develop dyspnea and stridor, especially with exertion. In patients with intrathoracic goiter, the dyspnea and stridor may be nocturnal or positional (ie, occurring when the patient's arms are raised) when the thoracic outlet is narrowed. Hemorrhage into a nodule or cyst or development of bronchitis may acutely worsen the respiratory symptoms in a patient with tracheal narrowing. The esophagus is more posterior in the neck, and a goiter occasionally extends posteriorly and causes solid food and pill dysphagia. Patients present with dyspnea and cough, especially with exertion, for tracheal obstruction. The patient's voice is assessed for hoarseness.

Hypothyroidism is indicated by a sallow complexion, dysarthric speech, mental slowing, weight gain without change in appetite, cold intolerance, constipation, hypersomnia, and delayed relaxation of deep tendon reflexes.

Hyperthyroidism is indicated by tachycardia, atrial arrhythmia (eg, atrial fibrillation), diaphoresis, weight loss without change in appetite, heat intolerance, hyperdefecation, palmar erythema, lid lag, tremor, and brisk reflexes.

The most common worldwide cause of endemic nontoxic goiter is iodine deficiency. However, in patients with sporadic goiter, the cause is usually unknown. Nontoxic goiters have many etiologies, including the following:

- Iodine deficiency

- Iodine excess
- Goitrogens
    - Drugs - Propylthiouracil, lithium, phenylbutazone, aminoglutethimide, iodine-containing expectorants
    - Environmental agents - Phenolic and phthalate ester derivatives and resorcinol found downstream of coal and shale mines
    - Foods - Vegetables of the genus *Brassica* (eg, cabbage, turnips, brussels sprouts, rutabagas), seaweed, millet, cassava, and goitrin in grass and weeds
- Dyshormonogenesis - A defect in the thyroid hormone biosynthetic pathway is inherited.
- Childhood head and neck radiation - Radiation exposure during childhood results in benign and malignant nodules.

Assess all patients with goiter for thyroid dysfunction with a serum thyrotropin (TSH) assay. Second-generation or better TSH assays can detect clinically inapparent (subclinical) hyperthyroidism and hypothyroidism.

- If the TSH is high, consider chronic autoimmune thyroiditis (Hashimoto thyroiditis) or ingestion of a goitrogen, such as lithium or amiodarone, as well as dyshormonogenesis in a child.
- If the TSH is low, measurement of serum free thyroxine (free T4) or free T4 index and total triiodothyronine (T3) is used to confirm the diagnosis of thyrotoxicosis.

Assessment of size and extent of the goiter is necessary to determine if progressive growth of the thyroid is occurring. Clinical assessment by an experienced clinician is often accurate until the thyroid increases to 4-5 times the normal size.

Ultrasonography is good for estimating the number and size of nodules but is inaccurate in the clinical setting for measuring the volume of large goiters. Computed tomography (CT) scanning and magnetic resonance imaging (MRI), although expensive, are excellent for assessing tracheal compression and intrathoracic extension of the goiter.

Nontoxic goiters usually grow very slowly over decades without causing symptoms. Without evidence of rapid growth, obstructive symptoms (eg, dysphagia, stridor, cough, shortness of breath), or thyrotoxicosis, no treatment is necessary. Therapy is considered if growth of the entire goiter or a specific nodule is present, especially if intrathoracic extension of the goiter, compressive symptoms, or thyrotoxicosis exists. The intrathoracic extension of the goiter cannot be assessed by palpation or biopsy. The goiter, if significant in size, should be removed surgically.[2] The currently

available therapies include thyroidectomy, radioactive iodine therapy, and levothyroxine (L-thyroxine, or T4) therapy.

Radioactive iodine therapy

Recombinant human TSH (rhTSH)[5,6,7] may have a role in radioactive iodine treatment for nontoxic goiter. Pretreatment with rhTSH 24 hours prior to therapy can reduce the amount of radioiodine needed to shrink the goiter (up to a 50% reduction).

Thyroid hormone suppressive therapy

Surgical Care

Thyroidectomy or surgical decompression causes rapid relief for obstructive symptoms.

Diets low in iodine need supplementation, especially in developing countries where government-supported iodine supplementation is not available. Patients taking iodine supplements may need a reduction to avoid iodine-induced thyroid disease in predisposed individuals.

Thyroid hormones (L-thyroxine)

T4 has been used to reduce the size or suppress the further growth of goiters.

Prevention of endemic goiter may be accomplished by iodine supplementation, using iodine supplements in drinking water sources or iodized oil on bread (strategies that can be applied to a whole country).

Prognosis

- Prognosis is good.
- Usually, nontoxic goiters grow very slowly over many years. Any rapid growth behavior must be evaluated for either degeneration or hemorrhage of a nodule or for growth of a neoplasm.

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