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Review article

Pathogenic Mechanisms of Inflammatory Thyroid Disorder: Goiter

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Abstract
A goiter is an enlarged thyroid gland, and it may be diffuse or nodular. A goiter may extend into the retrosternal space, with or without substantial anterior enlargement. Because of the anatomic relationship of the thyroid gland to the trachea, larynx, superior and inferior laryngeal nerves, and esophagus, abnormal growth may cause a variety of compressive syndromes. Thyroid function may be normal (nontoxic goiter), overactive (toxic goiter), or underactive (hypothyroid goiter). Initial screening for goiters should include thyroid-stimulating hormone.

Introduction
Goiter means enlargement of the thyroid gland and is a general term that conveys the information that the volume of the thyroid gland is larger than normal. The presence of goiter can be determined by inspection, palpation, or by an imaging study.Normal thyroid gland measures 4 to 4.8 cm in sagittal, 1 to 1.8 cm in transverse, and 0.8 to 1.6 cm in anteroposterior dimensions. This corresponds to a volume of 7 to 10 mL on ultrasonography calculations and 10-20 grams in weight. Thyroid size increases with age and body size. It is larger in males as opposed to females. The size decreases with higher iodine intake.The thyroid gland can enlarge due to a variety of physiological or pathological stimuli. Goiter during adolescence and pregnancy are two causes of a physiological goiter. Goiter can be associated with euthyroidism, hypothyroidism, or hyperthyroidism.

Etiology
Several pathogenic mechanisms can cause goiter. It can be caused by iodine deficiency, which is often seen in countries that do not have a public health intervention to prevent iodine deficiency. In this instance, the terminology is an endemic goiter.

Inflammatory disorders of the thyroid gland such as autoimmune thyroiditis, postpartum thyroiditis, silent thyroiditis, radiation thyroiditis, subacute thyroiditis, and suppurative thyroiditis can cause thyroid enlargement, hence goiter. As the enlargement of the thyroid is the consequence of the inflammatory process and abates after the inflammation resolves, the term "goiter" is not used to describe the disorder. Goiter is among the symptoms and signs of inflammatory thyroid disorder. Thyroid diseases that cause hyperthyroidism, such as Grave disease, toxic nodular goiter, and toxic multinodular goiter can cause goiter.

Epidemiology
The most common cause of goiters worldwide is iodine deficiency that affects an estimated 2.2 billion people. The prevalence and incidence of goiter are based on the degree of iodine deficiency. With mild iodine deficiency, the incidence of goiter is 5% to 20%. With a moderate deficiency, the prevalence increases to 20% to 30%, and with severe iodine deficiency, the incidence increases to greater than 30%. Even with the use of iodine, there has been an increase in the incidence of thyroid nodules. It is not clear the increase in prevalence represents a true increase or an increased detection. One of the causes of this could be the increased utilization of radiological imaging and more frequent screening with ultrasound.

At this time, ultrasonography can detect even the smallest of nodules, causing the incidence of nodules to be 60% to 70% in adults. However, imaging and screening are not the sole causes of the increase in incidence. It has been thought that obesity, insulin resistance, and metabolic syndrome may be factors that have caused an increased incidence of goiter.

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Dyshormonogenesis

Autosomal recessive disorders (exception DUOX 2 mutation). Enzymatic defect in one of the steps of thyroid hormone synthesis. The most common cause being the deficiency of thyroid peroxidase enzyme. Prevalence is more among females.

Most of the cases present clinically before third decade with as earliest as in neonatal period with goiter and hypothyroidism. In Pendred syndrome dyshormonogenetic goiter is associated with deafness.

Dietary factors

Iodine deficiency

Twenty-nine percent of world population lives in area where soil is deficient in iodine, e.g., the Himalayan region in India. The most common cause of goiter worldwide is iodine deficiency. More than 30% of population having median urine iodine less than 20 mcg/l developed goiter.

Hashimotos thyroiditis

The condition is named after Japanese physician Hakaru Hashimoto. This autoimmune thyroid disorder is characterized by diffuse lymphocytic infiltration of thyroid gland along with follicular destruction. Serologically the patients have high titer of anti-thyroglobulin and anti TPO antibodies.

Multinodular goiter

David Marine and Selwyn Taylor proposed that chronic intermittent stimulus leads to variable thyroid hyperplasia resulting in multinodular goiter. Various factors including genetic heterogeneity of follicular cells, secondary elevation of TSH due to iodine deficiency, goitrogens, and inborn error of thyroid hormone synthesis are considered to be factors involved in pathogenesis of condition.

Pathophysiology of goiter

Thyroid stimulating hormone is the major trophic factor for thyroid gland. TSH acts on TSH receptors present on thyroid cells which are G protein coupled receptors.

Conclusion

A sufficient daily intake of iodine is needed to prevent goiter. The recommended daily intake (RDI) of iodine is 90 µg/day for children aged 2 to 5 years, 120 µg/day for children aged 6 to 9 years, and 150 µg/day for children from 10 years of age, adolescents, and adults. In pregnancy, the RDI is 250 µg/day and for lactating women, an extra 50 µg/day is recommended to provide sufficient iodine in breast milk.

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