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Goiter disease in a nutshell

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Abstract

The disease caused by the enlargement and swelling of the thyroid gland is called goiter disease. Environmental and genetic risk factors affect the formation of the goiter disease. There are factors such as iodine deficiency, selenium deficiency, genetics, anemia, smoking and excessive secretion of TSH hormone in the pituitary gland, factors such as age and gender should also be considered in the formation of goiter. Goiter disease is basically examined in two ways; diffuse goiter and nodular goiter. Diffuse goiter, also called simple goiter, is swelling of the thyroid gland. Nodular goiter is the formation of oval masses in the thyroid gland. In both types of goiter, first of all, increases or decreases in free T3-T4 and TSH levels are taken into account by the biochemical methods. In diffuse goiter, drug therapy or, although very rarely, surgical methods are applied. In nodular goiter, benign and malignant determinations are made about the nodule structure by the radiological methods. Radioactive iodine treatment is applied in addition to the surgical methods in nodular goiter. This short review study has touched focuses on the pathogenesis of goiter disease, treatment approaches and different types of goiter and aims to explain them.

Introduction

The disease caused by the enlargement and swelling of the thyroid gland is called goiter [1]. Although goiter is the most common disease of endochronology, it is called subclinical hypothyroidism [2]. Iodine deficiency affects every age group, starting from the fetus. Goiter disease occurs due to insufficient iodine intake in these age groups [2]. Goiter occurs when serum free thyroid hormone is at a normal level but TSH hormone is high [1]. Iodine deficiency, selenium deficiency, genetics, anemia, smoking, and excessive secretion of TSH hormone by the pituitary gland are the factors affecting goiter. and symptoms such asmanifest itself as pain [2]. Goiter disease is examined in two different ways as diffuse goiter and nodular goiter [1,2].

While diffuse goiter disease causes enlargement of the thyroid gland, nodule formation is also observed. However, nodule formation is not observed in every diffuse goiter disease. If there is no diffuse goiter nodule formation and hyperthyroidism, it is called euthyroid goiter [4]. Euthyroid goiter is examined in two ways as sporadic and endemic. Endemic goiter is bilateral or unilateral enlargement of the thyroid gland. Sporadic goiter, on the other hand, is an enlargement of the thyroid gland that develops outside the endemic region [5]. It is thought that genetic predisposition is more important than other factors in diffuse goiter [4]. Some studies have provided the mapping of the Xp22 chromosome region, which is associated with the dominant form of enlargement of the thyroid gland for clonal reasons. According to this thyroid physiology, they are important candidate genes for thyroglobulin, thyroid peroxidase, Na^+ / I^- symporters, pendrin gene and thyroid stimulating hormone receptor [5,6].

In nodular goiter, round mass formation in different sizes is observed in the thyroid gland. In the first step evaluation of nodules, benign and malignant determinations are made [2,3]. TSH measurement and thyroid ultrasound are observed during primary care evaluation [3]. As a result of these measurements, thyroid fine needle

aspiration biopsy is performed in the second step. In nodular goiter, the forms of cold, hot and warm nodules can be found simultaneously in the same thyroid gland [3,5].

Activation of thyroid stimulating hormone receptors in warm thyroid nodules results in stimulation of adenyl cyclase via the Gsα protein [5,6]. At higher TSH concentration, the phospholipase C cascade has been shown to be active through Gpα [6]. However, some studies focus on the cAMP pathway of TSH signaling. As a result of these studies, it has been proven that the increase in cAMP is effective on goiter [3,5,6].

NIS (Na^+ / I^- symporters) is responsible for the active transport of thyroid follicles [6].

It was observed that the expression levels of Na^+ / I^- symporter decreased in cold thyroid nodules [6].

It is thought that somatic mutations in genes involved in the transport of iodine affect nodular goiter. However, studies have not yet observed mutations in thyroid peroxidase, Na^+ / I^- symporter and thyroid-specific oxidase genes [5,6].

The appearance outcome of goiter due to iodine deficiency in the blood is low T4, normal or high T3 and close to the upper limit of TSH [7]. Depending on these factors levels is result, the examinations are it is followed by Direct X-ray, Thyroid Ultrasonography (USG), Color Doppler USG, Computed Tomography, Magnetic Resonance Imaging and Thyroid Scintigraphy [7].

Levothyroxine suppression therapy can be used in the treatment of diffuse goiter. Levothyroxine is taken by mouth or given by intravenous injection. However, radioactive iodine treatment can be applied to shrink the thyroid gland in patients who require surgery and cannot be operated [7,8].

When diagnosing nodular goiter, biochemical tests are based on serum TSH values. In fine needle aspiration biopsy, benign and malignant detection of the nodule is possibly made.

Treatment methods include antithyroid drug therapy, RAIT and surgical treatment [8,9].

To evaluate the effects of factors such as gender, age, diameter of the nodule, cystic and solid morphological features, use of antithyroid drugs and the dose of radioactive iodine treatment on the treatment results of patients who received radioactive iodine treatment [7,8]. In radioactive iodine treatment, a single capsule and liquid reconstituted capsule is applied to the patients in the form. The capsule is transmitted to the thyroid gland by the blood circulation, and thanks to effective radiation, it causes the thyroid tissue to shrink [8,9].

Biochemical methods are carried out in 2 stages. First, thyroid function tests find the connection between serum thyroid and TSH hormone. Upon the connection between the hormone and the thyroid gland, the TSH hormone is intervened. Any change to the TSH hormone can give positive great results. The most important method in the diagnosis of diffuse goiter or nodular goiter is the measurement of TSH hormone. With this diagnostic method, it is monitored which autoantibody is associated with thyroid disorders. While applying all these treatment methods, conditions such as the age, genetic predispositions and living standards of the patients are also taken into account [9-11].

Discussion

Graves' disease is among the causes of goiter. Graves' disease is in the group of autoimmune diseases. The immune system of the person produces the protein called thyroid stimulating immunoglobulin. The TSI protein stimulates the thyroid gland and causes the thyroid gland to enlarge. As a result, Graves' disease causes goiter formation and hyperthyroidism [7,10].

In a study conducted in the treatment of goiter disease; aAntioxidant vitamins, selenium and erythrocyte glutathione peroxidase levels were determined in the serum of diagnosed patients. As a result of this study, a decrease was observed in the vitamin and selenium levels of the patient with goiter, while no change in erythrocyte glutathione peroxidase was observed [7,8,10].

Conclusion

Goiter is the most common disease of endochronology. The incidence of goitre disease varies according to age, gender and the region of residence. A goiter patient can have one of the different types, such as hyperthyroid, hypothyroid, or euthyroid. The incidence of goiter in women is 30% higher than in men. The frequency of goiter in Turkey varies between 5% and 56%, depending on environmental conditions. Its incidence is higher in endemic regions with iodine deficiency. Studies have also proven that 39% of women live in regions with iodine deficiency due to genetics, and 82% of women live in regions where iodine is taken regularly [9,10].

In some studies, on nodular goiter, it is thought that the decrease in the expression of the Bad protein, which belongs to a subset of the pro-apoptotic Bcl-2 gene in the nodule structure, may contribute to the pathogenesis of nodule formation. Observing the effects of genetic and environmental factors affecting Bbad expression is thought to be a pioneer in the development of new treatment methods for the goiter disease. Future studies should focus on the expression levels of Bad protein, which is one of the proteins that are effective in the formation of goiter disease [10,11].

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