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THE CLINICAL COMPLEX ARISING FROM THYROID DISORDERS: A GENERAL APPROACH

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Summary

The thyroid is an endocrine gland, subject to hormonal stimulation, that is, by the hormone TSH, which is synthesized by the pituitary gland and sent to the thyroid via the bloodstream, thus synthesizing the hormones T4 (thyroxine) and T3 (triiodothyronine), which are responsible for control metabolic functions. Dysfunctions can occur such as hypothyroidism and hyperthyroidism, which are associated with factors such as pregnancy, aging, autoimmune disease and drug interactions with a certain type of drug, all of which can disrupt hormonal levels. Excessive or insufficient production of thyroid hormones can cause pathologies secondary to thyroid dysfunction. Therefore, knowledge about the proper functioning of the thyroid gland is essential, seeking to preserve homeostasis and, subsequently, a better quality of life for the patient. The diagnosis, that in both conditions, serum levels of Thyroid Stimulating Hormone (TSH) and thyroid hormone are essential for this, and treatment are essential for the patient to lead a normal life. The objective was to carry out a literature review on general aspects of the clinical complex arising from thyroid disorders, addressing the respective diagnosis and treatment. **Keywords: Endocrine gland; hormonal disorder; thyroid.**

Abstract

The thyroid is in the endocrine gland, subject to hormonal stimulation, that is, by the hormone TSH, which is synthesized by the pituitary gland and sent to the thyroid via the bloodstream, thus synthesizing the hormones T4 (thyroxine) and T3 (triiodothyronine), which are responsible for controlling metabolic functions. Dysfunctions can occur such as hypothyroidism and hyperthyroidism, which are associated with factors such as pregnancy, aging, autoimmune disease and drug interactions with a certain type of drug, all of which can disrupt hormonal levels. Excessive or insufficient production of thyroid hormones can cause pathologies secondary to thyroid dysfunction. Therefore, knowledge about the proper functioning of the thyroid gland is essential, seeking to preserve homeostasis and, subsequently, a better quality of life for the patient. The diagnosis, that in both conditions, serum levels of Thyroid Stimulating Hormone (TSH) and thyroid hormone are essential for this, and treatment are essential for the patient to lead a normal life. The objective was to carry out a literature review on general aspects of the clinical complex arising from thyroid disorders, addressing the respective diagnosis and treatment.

Keywords: Endocrine gland; hormonal disorder; thyroid.

Introduction

The thyroid gland, similarly known as the butterfly gland due to its shape, is one of the largest glands in the human endocrine system. Located in the anterior portion of the neck. The thyroid is essential for our lives, as it is responsible for regulating our body's metabolism. Acting on various organs and functions of the body, throughout life. It influences organs such as the heart, brain, liver and kidneys; helps regulate the growth and development of the body; menstrual cycles and female fertility; It is important for memory, concentration, mood and emotional control. All of these events occur through the thyroid hormones produced by the gland: triiodothyronine, known as T3, and thyroxine, known as T4. These hormones, when entering the bloodstream, reach any tissue or organ and balance various functions. These hormones cannot become unbalanced, as their increase or decrease causes negative reactions in the body, such as hypothyroidism and hyperthyroidism.

The following article aimed to describe in a narrative way considerations regarding the thyroid, as well as

as disorders that cause pathologies, making early diagnosis and therapy essential.

Methodology

This is a qualitative narrative review study, appropriate to describe the clinical complex arising from thyroid disorders. It consists of a broad analysis of the literature, without establishing a rigorous and replicable methodology at the level of data reproduction and quantitative answers to specific questions).

As it is a bibliographic analysis on the theory of mind and the understanding of this competence in the adult individual, articles indexed in the SciELO, PubMed, Latindex, Google Scholar databases were retrieved, during the month of January 2024, using the reference period as the last 5 years.

The index terms or descriptors thyroid, adrenal gland, hormonal pathologies were used, isolated or in combination, without delimiting a temporal interval. The criteria used for inclusion of publications was to have the expressions used in the searches in the title or keywords, or to have it explicit in the abstract that the text is related to the association of thyroid disorders and linked pathologies. The excluded articles did not meet the established inclusion criteria and/or were duplicated, that is, publications retrieved from more than one of the databases. Dissertations and theses were also excluded.

After the target information was retrieved, the titles and abstracts were initially read, with no publications being excluded at this stage. Subsequently, the complete reading of the 31 texts was carried out. As axes of analysis, we initially sought to classify the studies according to the particularities of the sampling. From there, the analysis of the theoretical foundation of the studies continued, as well as the observation of the general characteristics of the articles, such as year of publication and language, followed by their objectives. Finally, the methodology applied, results obtained and discussion were assessed. Specifically, to analyze the scientific production identified, specific qualitative and/or quantitative data processing techniques were not used, and each text was analyzed.

Results and discussion

The search for articles that made up this study identified 77 references on thyroid disorders in the aforementioned databases, of which 40 publications were included in the review. Among the selected studies, 38 articles present a theoretical approach and 02 are case studies. The prevalence of publications in English was observed, representing 84% of the total, when compared to Spanish (9.6%) and Portuguese (6.4%).

From a careful reading of the selected texts, it was possible to observe that the studies are unanimous regarding the initial contextualization of the thyroid being responsible for the production of the hormones tetraiodothyronine or thyroxine (T4) and tri-iodothyronine (T3). Both originate from the complement of iodine radicals to tyrosine residues contained in a large 660 kDa glycoprotein, called thyroglobulin (Tg), secreted by follicular cells and stored within the thyroid follicle. Tg corresponds to 70 to 80% of the protein content of the thyroid.

The uptake of iodine from the bloodstream is actively carried out by the Na/I Symporter (NIS) protein, present in the basement membrane of the follicular cell. The enzyme responsible for the oxidation of iodine ions and their binding to Tg is thyroperoxidase (TPO), present in the apical membrane of follicular cells. Initially, molecules composed of a tyrosine residue, linked to Tg, and an iodine molecule — monoiodotyrosine (MIT) — or two iodine molecules — diiodotyrosine (DIT) are formed. MIT and DIT are cleaved and released from Tg by the action of TPO, phagocytosed and linked, so that two DITs form T4 and one DIT, added to an MIT, forms T3

T3 and T4 circulate in plasma bound to carrier proteins, such as thyroxine-binding globulin (TBG), and albumin. Only a tiny fraction of these hormones — 0.04% of T4 and 0.4% of T3 — circulates in plasma in free form, not bound to proteins, and is responsible for biological activity of thyroid hormones.

The regulation of thyroid function is mediated mainly by the pituitary hormone thyrotropin, or TSH, which stimulates the synthesis and release of hormones, as well as thyroid growth. TSH, in turn, is positively regulated by hypothalamic thyrotropin-releasing hormone (TRH) and negatively regulated by somatostatin. Secreted T3 binds to receptors in the hypothalamus and pituitary gland, where it inhibits secretion

of TRH and TSH by negative feedback, thus regulating thyroid function

T4 is predominant in thyroid secretions, but T3 is responsible for most of the gland's biological action. Justified by the fact that T3 has an affinity for the thyroid hormone receptor four to 10 times more potent than T4

Intracellular T3 has two main sources: synthesis from T4 and secretion by the thyroid itself. Deiodination: extraction of an iodine atom from T4, making it T3, occurs through the action of enzymes called 5'-deiodinases, present in practically all organs and systems.

In fact, the peripheral conversion of T4 to T3 is responsible for the majority of circulating T3. T3 binds to nuclear receptors belonging to the superfamily of retinoid receptors, present in practically all organs, regulating the transcription of specific gene sequences, which will lead to the most diverse biological actions of the hormone.

The prevalence of hypothyroidism varies in different regions of the world, being more common in places where there is a deficiency or excess of iodine in the diet, despite being common in iodine-replete areas. It is about 10 times more common in females. Congenital hypothyroidism affects 1:4,000 newborns, and central hypothyroidism is found in 1:20,000 individuals.

Hypothyroidism is one of the most common endocrine pathologies, mostly triggered by deficient synthesis of thyroid hormones.

The etiologies of hypothyroidism can be grouped into:

• Primary: conflict located in the peripheral gland — Central thyroid disease

• Secondary: problem located in the pituitary gland — TSH deficiency;

• Tertiary: problem located in the hypothalamus — TRH deficiency.

Primary	Secondary	Tertiary
Iodine deficiency	Pituitary adenoma	Hypothalamic dysfunction
Hashimoto's thyroiditis		Idiopathic
Excess iodine Defects in hormone synthesis		Surgery or irradiation of the hypothalamic region

The abundance of hypothyroidism episodes, representing 95%, is primary. In Brazil, the main cause of this syndrome is chronic autoimmune thyroiditis, or Hashimoto's thyroiditis. Subacute granulomatous, lymphocytic and postpartum thyroiditis can also lead to hypothyroidism, which, in these situations, is usually transient

Cretinism is a mental disability caused by**congenital hypothyroidism**, during the development of the newborn. During this period, the absence of the hormone thyroxine disrupts brain maturation.

The main cause of the disorder is a defect in the**thyroid formation**, but it is also possible that it occurs due to an enzyme deficiency during the development of the hormone. There is one case of cretinism every three thousand births and it is possible to identify the disease through**Foot test**.

Myxedema is usually caused by prolonged hypothyroidism, myxedema is a hard edema with an opaque skin appearance. The disorder occurs five times more frequently in women than in men. People with myxedema experience edema on the face and eyelids, forming “bags” under the eyes. There is also an accumulation of proteins produced in hypothyroidism.

Hashimoto's thyroiditis

Chronic autoimmune thyroiditis — most common autoimmune disease

Goiter is common in around 90% of cases (goiterogenic form). The volume expansion of the Rheoid is generally mild to moderate, symmetrical, slow to develop over decades, and firm in consistency, like rubber, on palpation. Ten percent present with a diminished thyroid, perhaps reflecting the final stage of the gland's chronic destructive process — the atrophic form.

Hashimoto's thyroiditis can, therefore, appear in an incipient form, without hypothyroidism itself and with a normal-sized, enlarged or diminished thyroid. If you have Hashimoto's thyroiditis,

Progression from euthyroidism to subclinical hypothyroidism and then to frank hypothyroidism occurs at a rate of 3 to 15% per year.

The vast majority have high serum titers of antithyroid antibodies, mainly anti-TPO, present in 80 to 99% of cases, and anti-Tg, present in 30 to 60% of cases. However, they are not specific, as they can be elevated in people without thyroid disease — anti-Tg, in up to 10% of the general population, and anti-TPO, in up to 15%.

Hypothyroidism occurs due to immune-mediated destruction of the thyroid parenchyma. Thyroid autoimmunity is of the cytotoxic type, full of cytotoxic or natural killer "T" cells

Diagnosis is based on the abundant identification of autoantibodies (anti-TPO or anti-Tg) in a person with goiter and/or hypothyroidism. Furthermore, the hypoechogenicity of the thyroid parenchyma on ultrasound. The anatomopathological examination may reveal lymphocytic infiltration.

Thyroid nodules

They correspond to benign or malignant tumors in the thyroid gland, mostly proportional to the increase in age. The predominance is benign. The etiology includes hyperplastic colloid goiter, thyroid cyst, thyroiditis, thyroid adenoma

The nodules may be painful or asymptomatic. Pain indicates thyroiditis or hemorrhage into a cyst. The asymptomatic nodule may be malignant, but is usually benign

The clinical picture of hyperthyroidism, usually palpitations, heat intolerance, weight loss, tremors leads to the hypothesis of hyperfunctional adenoma or thyroiditis. Hypothyroidism, which leads to intolerance to cold, causes weight gain, fatigue, suggests Hashimoto's thyroiditis

Predisposing factors for thyroid cancer include a history of thyroid radiation, especially in childhood, male gender, previous family history of thyroid cancer or multiple endocrine neoplasia type 2, solitary nodule, dysphagia, dysphonia. Increase in size (particularly rapid growth or growth while undergoing thyroid hormone suppression treatment)

On physical examination theSymptoms that suggest thyroid cancer include stony stiffness or fixation to adjacent structures, cervical lymphadenopathy, and hoarseness (due to recurrent laryngeal nerve palsy).

The initial evaluation of a thyroid nodule is performed with tests for Thyroid Stimulating Hormone (TSH) and anti-thyroid peroxidase antibodies. If thyroid-stimulating hormone (TSH) is low (consistent with hyperthyroidism), a radioactive iodine test is performed. Nodules with increased iodine uptake (warm) are rarely malignant.

If thyroid function tests do not indicate hyperthyroidism or Hashimoto's thyroiditis, the next step is to examine the nodule with a thyroid ultrasound. Ultrasonography is useful for determining the size of the nodule, but may not be able to distinguish between benign and malignant lesions. Thyroid cancer is suggested in case of hypoechogenicity of the nodules; accentuated internal vascularization; uneven edges; nodule height greater than its width; irregular macrocalcifications; fine dotted psammomatous microcalcifications (papillary-type thyroid carcinoma); or dense homogeneous irregular calcification (medullary thyroid carcinoma).

For nodules with suspicious characteristics, fine needle aspiration biopsy (FNAB) performed under ultrasound guidance is the procedure of choice and the most economical means of distinguishing benign from malignant nodules. In general, FNAC is not recommended for nodules <1 cm or nodules that are entirely cystic.

The cytological examination of cells obtained from FNAC is capable of distinguishing benign from malignant lesions. For cytologically indeterminate lesions, molecular analysis of FNA aspirate looking for mutations and rearrangements or measuring calcitonin looking for possible medullary carcinoma can add clarity sufficient diagnostics to enable an optimally informed treatment recommendation.

Treatment of a thyroid nodule is based on therapy for the underlying disease. Nodules in patients with Hashimoto's thyroiditis or other causes of goiter associated with hypothyroidism may stabilize or regress with thyroxine replacement at doses that make the patient euthyroid. For small benign nodules not associated with hypothyroidism, TSH suppression with thyroxine effectively shrinks the nodule in no more than half of cases, and is rarely done. Thyroxine is not used to treat neoplastic nodules.

The size of large benign nodules that compress adjacent cervical structures may decrease.

nuir after radiofrequency ablation. Partial or complete thyroidectomy is effective in treating nodules that continue to grow or cause compressive symptoms.

Toxic nodules can be treated with radioactive iodine to control hyperthyroidism and reduce their size. Nodules that are not due to thyroid cancer, do not cause compressive symptoms, and are not associated with hypothyroidism or hyperthyroidism can be safely monitored with periodic physical examinations of the neck and, if growth of the nodules is suspected, by repeat ultrasound evaluation.

Goiter

Goiter, also known as puffiness, is an increase in volume in the thyroid. Normally, it is associated with hypothyroidism or hyperthyroidism, but this association is not necessary or mandatory.

Many goiters have nodules and are called nodular goiters, which do not necessarily interfere with thyroid function and therefore do not generate symptoms of hypo or hyperthyroidism.

Diffuse goiter: May occur in thyroid dysfunction or when there is an iodine deficiency.

Nodular goiter: It can be uninodular (just one nodule) or multinodular (more than one goiter).

Nodular goiter is most often diagnosed after age 50. The patient may have had the goiter for several years and only detect it when compressive symptoms such as dysphagia and hoarseness appear; or even just diagnose because it has become hyperfunctioning, causing hyperthyroidism. Another episode that may occur is presenting the diagnosis only when investigating associated hypothyroidism.

In addition to palpation, blood tests and ultrasound must be ordered by the doctor. To assess whether there is a change in thyroid function, TSH measurement is the best initial investigation test.

Further investigation and treatment will depend on the cause, symptoms, changes (or not) in the functioning of the gland and the characteristics of the nodules on ultrasound (in cases of nodular goiters).

Conclusion

It was found that the thyroid plays a very important role in the patient's life. The hormones secreted by it and stimulated by TSH, as long as they are at normal levels, are responsible for controlling various functions of the body. It is worth highlighting the importance of carrying out routine tests to check whether the levels of these hormones are properly regulated, as their dysfunctions can cause harm to the patient's daily life, especially in women over 40 years of age, the elderly and pregnant women.

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