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Morphological Characteristics of the Thyroid Gland

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Abstract: Hypothyroidism is a common endocrine disorder resulting of low levels of thyroid circulating hormones. The prevalence in the general population varies between 0.3% and 3.7%. Presents as clinical or subclinical disease based on presence of symptoms and levels of serum TSH and free thyroxine and T4, respectively. Hypothyroidism has numerous etiologies, some of them are originated on the thyroid itself and some others are of extrathyroid origin, with variable manifestations. Classified as primary, secondary, tertiary and peripheral. Thyroid autoimmune disease is the principal cause. A new class of drugs against cancer, like the anti-CTLA-4 and anti-PD-L1/PD1 therapies have been associated with primary or secondary hypothyroidism. Endocrine disorders can be difficult to diagnose based only on morphological features because endocrine manifestations are caused primarily by a hormonal imbalance. Hypothyroidism may have a higher risk of morbidity and mortality. Finally, myxedematous coma is the main complication of terminal stages hypothyroidism.

Keywords: Endocrine disorders, myxedematous, paratracheal, retropharyngeal, retroesophageal and the internal jugular nodes.

The thyroid gland is a butterfly-shaped organ formed by a right and left lobe connected at the midline by a thin structure called isthmus. Located in the neck, the thyroid covers the anterior side of the trachea underneath the larynx at the vertebral levels of C5 to T1 (Figure 1A). The average size of a thyroid gland is of 5 cm height and 5 cm wide and it weighs between 20 and 30 grams in adults (Figure 1B), being a little more heavy in women. Is a highly vascular organ, receiving blood supply from two main sources, the superior thyroid artery, branch of the external carotid artery irrigates the superior half of the thyroid in more than 95% of the population, the inferior half is irrigated by the inferior thyroid artery that branches from the thyrocervical trunk which is a branch of the subclavian artery. Furthermore, the thyroid gland has extensive lymphatic drainage that involves multiple levels of lymphatic nodes, including the prelaryngeal, pre and paratracheal, retropharyngeal, retroesophageal and the internal jugular nodes.

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Described in 1850, hypothyroidism was the first disorder of endocrine deficiency ever reported . Hypothyroidism is the result of low levels of thyroid circulating hormones. Due to the wide variety of clinical presentations and the lack of specific symptoms, the definition of hypothyroidism is mainly biochemical . Therefore, hormonal levels in overt hypothyroidism are: TSH (Thyroid Stimulating Hormone) >4.8 UI/l, FT4 < 13 pmol/l, and in subclinical hypothyroidism are: TSH >4.8 UI/l, FT4: 13–23 pmol/l [7]. Recent research suggests that the superior reference values for serum TSH varies among different age groups [8]. Nevertheless, up to this present day there is no exact definition of a cut point

for serum TSH values regarding age in our population. According to the moment of clinical presentation, hypothyroidism is divided in congenital or acquired, according to the level of endocrine dysfunction is divided in primary or secondary or central and according to the severity of hypothyroidism is divided in severe or clinic hypothyroidism or in mild or subclinical hypothyroidism.

To understand better hypothyroidism and its consequences it is important to remember the normal physiology of the thyroid gland. The main function of the thyroid follicular cells is the synthesis of thyroid hormones, tetraiodothyronine or (T4; 3,5,3′,5′-L-tetraiodothyronine) and triiodothyronine (T3; 3,5,3′-L-triiodothyronine). Iodine is essential for thyroid hormone synthesis. Food and water are the main sources for iodine intake, with a daily supply that ranges from 50 to 300 μg being absorbed in the small intestine. Both thyroid hormones are synthesized by the iodination and condensation of two tyrosine molecules and differ by an iodine atom. The production and release of thyroid hormones is stimulated by the hypothalamus-pituitary axis. The thyrotropin-releasing hormone (TRH) released from the hypothalamus stimulates the anterior pituitary gland to release thyrotropin, also called TSH . In response to the stimuli of TSH, the thyroid follicular cells produce thyroglobulin, an inactive protein that is then released from the apical surface into the follicle as a colloid. TSH is released into the bloodstream and it then binds to the thyroid stimulating hormone receptor (TSH-R) in the basolateral surface of the follicular cell of the thyroid gland. The TSH-R is a G-protein coupled receptor and its triggering yields to the activation of the Adenylate Cyclase and of increased levels of intracellular cAMP. An increased cAMP activates the protein kinase A (PKA). PKA phosphorylates different proteins in order to change their functions. The thyroid hormone biosynthesis is made by steps, regulated by enzymes that are stimulated by TSH, these steps are: 1) thyroglobulin synthesis (TG): the thyrocites in the thyroid follicles produce a protein called thyroglobulin. Thyroglobulin does not contain iodine and is a precursor protein storaged in the follicle lumen. Thyroglobulin is produced in the rough endoplasmic reticulum, then the Golgi apparatus packs it up in vesicles and then it enters the follicle lumen by exocytosis. 2) Iodine uptake and transport: the phosphorylation of the kinase A protein increases the activity of the sodium/iodide basolateral symporter protein (Na+/I- symporter), driven by the Na + -K + -ATPase to get iodine out of the bloodstream to the thyrocites. Iodine diffuses from the basolateral surface to the apical surface of the cell, where it transports to the colloid through the pendrin transporter; 3) thyroglobulin iodination: the protein kinase A also phosphorylates and activates the thyroid peroxidase enzyme (TPO). The TPO has three main functions: oxidation, organification and coupling reaction. 4) Oxidation: the TPO uses hydrogen peroxide in order to oxidate iodide (I-) to iodine (I2). NADPH oxidase, an apical enzyme generates hydrogen peroxide for the TPO; 5) Organization: the TPO attaches the remainders of tyrosine from the thyroglobulin with the I2. It generates monoiodityrosine (MIT) and diiodotyrosine (DIT) (Figure 4). MIT has only one remaining tyrosine with iodine and DIT has two remaining tyrosine with iodine; 6) mono and diiodotyrosine attachment; the TPO combines the remainers of iodated tyrosine to produce T3 and T4 . MIT and DIT combine to form T3 and two DIT molecules form T4; 5) Storing: thyroid hormones are attached to TG and are storage in the follicular lumen; and 6) secretion: the iodized thyroglobulin returns to the follicular cell, where the degradation of lysosomic proteases releases T3 and T4 in the fenestrated capillaries. Thyroid hormones travel through the bloodstream united to a binding protein called thyroxin . The thyroxine-binding globuline (TBG), transthyretin (TTR) and albumin are proteins capable to bind to the thyroid hormone, thus becoming able to transport it through the bloodstream to their target sites.

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