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Review Article

A Review on Hypothyroidism, its Pathophysiology, Secretion of Thyroid Hormones, Diagnosis and Treatment

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ABSTRACT

One of the most common endocrine illnesses in the world is thyroid problems. Over 42 million individuals in India alone suffer from this category of disorders that impair the thyroid gland's ability to operate. These conditions can show up as either an overactive thyroid (hyperthyroidism) or an underactive thyroid (hypothyroidism). It has a major effect on the human body's development, metabolism, and general health. The primary objective of this review article is to give a thorough overview of the thyroid glands anatomy and physiology, the different kinds of thyroid disorders, such as hypothyroidism, hyperthyroidism, thyroid cancer, goitre, thyroiditis, and thyroid disorders during pregnancy, as well as their causes, risk factors, clinical signs, diagnostic methods, and treatment options. For early discovery and successful treatment, it is essential to comprehend the pathophysiology of these illnesses. The article delves deeper into how environmental and genetic variables contribute to the development of thyroid disorders, as well as the most recent developments in diagnostic methods and treatment approaches. It also emphasizes the significance of early intervention and how thyroid dysfunction affects quality of life, especially in vulnerable groups like the elderly and pregnant women. This review attempts to contribute to a more comprehensive knowledge and improved management of thyroid problems by combining current research and clinical practices.

INTRODUCTION

Hypothyroidism is a disorder in which the thyroid gland is underactive and produces insufficient thyroid hormones. The enlarged thyroid gland (galaganda), today known as goitre, is the most frequent form of this ailment specified in Ayurveda. Iodine deficiency is the most common cause of hypothyroidism, which is largely prevalent exclusively in iodine-deficient areas and is not even avoided by India's National Iodine Deficiency Disease Control Program. In many places of India, iodine deficiency still exists. [1] Hypothyroidism is the most prevalent thyroid

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illness, and it occurs more frequently than previously thought. The majority of people are unaware that they have it. Those who have this illness will experience symptoms due to a slowed metabolism. Subclinical hypothyroidism affects 3 to 8% of people, and it gets worse as they get older. Women are more likely than men to suffer from hypothyroidism. Thyroid hormone insufficiency affects around ten percent of women. Hypothyroidism can have an impact on lipid metabolism, neurological disorders, and other medical issues. [2] Hypothyroidism's effects on peripheral tissues Primary hypothyroidism is caused by a lack of hormone production by the thyroid gland, but secondary hypothyroidism can also be caused by insufficient secretion of either thyrotropin (similar to thyroid-stimulating hormone-TSH) from the pituitary gland or thyrotropin-releasing hormone (TRH) from the hypothalamus (tertiary hypothyroidism). Hypothyroidism is a highly common condition in today's population. Clinical hypothyroidism affects roughly 2% of adult women and 0.1-0.2 percent of males, whereas subclinical hypothyroidism affects up to 9% of the adult population, but this disease worsens with age. This hypothyroidism condition (congenital hypothyroidism) affects about 1 in 3500 infants [3]

TYPES AND CAUSES OF HYPOTHYROIDIS:

Hypothyroidism can be classified as follows: -

I. Primary (due to thyroid hormone deficiency),

II. Secondary (due to TSH deficiency),

III. Tertiary (due to thyrotropin-releasing hormone deficiency), and ➢ Peripheral (extra-thyroidal; panel).

IV. Central hypothyroidism (including both secondary and tertiary) and peripheral hypothyroidism are rare.

I. Primary hypothyroidism**: (**it includes 95% of cases)

- Chronic autoimmune thyroiditis which is also known as Hashimoto's thyroiditis.
- Iodine severe iodine deficiency, severe and mild iodine excess.
- Drugs like amiodarone, lithium, tyrosine kinase inhibitors, interferon-alfa, thalidomide, monoclonal antibodies antiepileptic drugs and the second-line drug used for the treatment of tuberculosis
- Graves' disease or toxic nodular disease radiotherapy (Iatrogenic radioiodine treatment) or surgery in the neck or head region
- Transient thyroiditis viral, post-partum, silent thyroiditis, destructive thyroiditis
- Thyroid gland infiltration infectious like mycoplasma, malignant like thyroid malignancy, lymphoma, metastasis of malignancy, autoimmune like sarcoidosis, inflammatory.
- Genetic autoimmunity-related genes, general and thyroid-specific genes. [4]

II. Secondary hypothyroidism**:** it includes (5% of cases)

- Pituitary or hypothalamic neoplasms
- Congenital hypopituitarism
- Pituitary necrosis (Sheehan's syndrome) [5]

III. Central hypothyroidism

- Pituitary tumours (secreting or non-secreting)
- Pituitary dysfunction
- Hypothalamic dysfunction
- Resistance to thyroid-stimulating hormone (TSH) or thyrotropin-releasing hormone
- Drugs (like dopamine, somatostatins, gluco-corticosteroids.
- Increased TSH concentration due to leptin stimulation

IV. Peripheral (extra-thyroidal) hypothyroidism

- Consumptive hypothyroidism
- Tissue-specific hypothyroidism due to decreased sensitivity to thyroid hormone [6]

Clinical sign and symptoms of hypothyroidism: -

- Constipation
- Depression
- Having trouble concentrating
- Menorrhagia
- Myalgias
- Weakness
- Gaining weight
- Skin that is dry
- Fatigue
- Hair thinning is a problem that affects a lot of people.
- Hair loss is a common problem.
- Impairment of memory
- Puffiness in hand.
- Bradycardia
- Coarse facies
- Cognitive impairment
- Delayed relaxation phase of deep tendon reflexes
- Diastolic hypertension
- Oedema
- Increased creatine kinase
- Increased low-density lipoprotein cholesterol [7]

PATHOPHYSIOLOGY

Hypothyroidism is generally caused by inadequate thyroid gland stimulation or by primary gland failure by the hypothalamus or pituitary gland. Congenital anomalies, autoimmune destruction (Hashimoto disease), iodine shortage, and infiltrative disorders are the most common causes of primary gland failure (primary hypothyroidism). In the United States, autoimmune thyroid disease is the most prevalent cause of hypothyroidism. Symptoms of induced forms of hypothyroidism include thyroid surgery, radioiodine therapy, and neck irradiation. Thyroiditis associated with thyroid-stimulating hormone (TSH) receptor-blocking antibodies, postpartum thyroiditis, subacute thyroiditis, silent thyroiditis, and thyroiditis associated with thyroid-

stimulating hormone (TSH) receptor-blocking antibodies are the most common disorders associated with transient hypothyroidism. The central causes of hypothyroidism are manifested by abnormally normal or low TSH levels in relation to insufficient thyroid hormone, and are associated with various signs of hypothalamic or pituitary dysfunction. Lithium, amiodarone, interferon alfa, interleukin-2, and tyrosine kinase inhibitors are some of the drugs usually associated with thyroid dysfunction. [8]

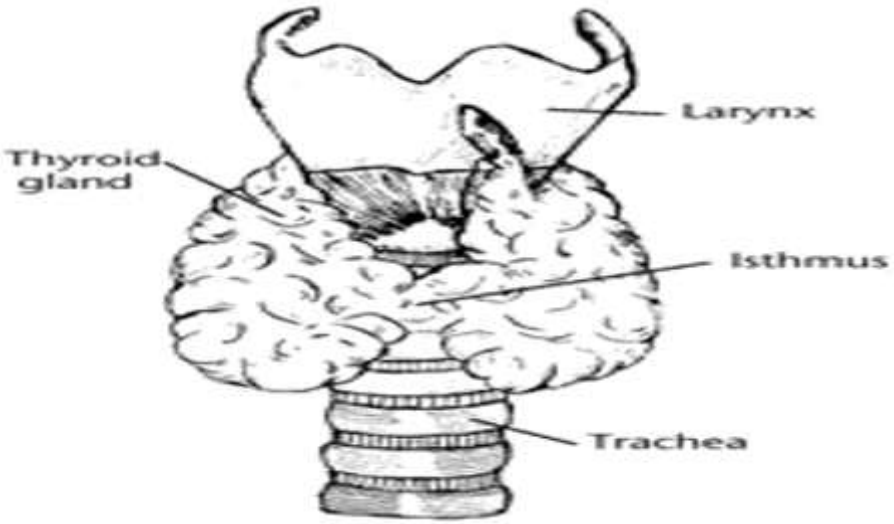
Molecular and biochemical pathophysiology

Thyroid hormone actions are reduced in clinical hypothyroidism, including calorigenesis and oxygen consumption modulation in most tissues, as well as new organ-specific effects. Thyroxine, the thyroid gland's major product and circulating thyroid hormone, is converted to triiodothyronine in the cytoplasm and nucleus of target tissues via outer-ring monodeiodination by three separate tissue-specific deiodinases. Triiodothyronine binding to at least one of the nuclear receptor superfamily's tri-iodothyronine receptor isoforms is expected to mediate most classical thyroid hormone functions gnomically (TR1, TR1, and TR2). Triiodothyronine receptors have regions that allow them to bind triiodothyronine, DNA, and form dimers with other triiodothyronine receptors or nuclear receptors (such as the retinoic acid X receptor). Thyroid hormone-responsive genes have receptors that bind to DNA in the 5' regulatory regions at specific hexametric oligonucleotide sequences and at specific orientations of paired thyroid response elements. Tri-iodothyronine binds to its receptor, causing accessory protein cofactors to bind, either activating or repressing the transcription of a certain gene (as tri-iodothyronine does to the hypothalamus thyroid releasing hormone and thyrotropin component genes). Using this paradigm, several clinical indications of hypothyroidism can be comprehended at the molecular level. Failure to activate the growth hormone gene in pituitary somatotrophs, for example, results in poor stature in prepubescent children. [9]

SYNTHESIS AND SECREATION OF THE THYROID HORMONE:

ANATOMY OF THYROID GLAND

Thyroid gland is one of the largest endocrine glands in the body, weighing about 15 to 20 grams in adults (0.5 to 0.75 ounces). The thyroid gland is found beneath the larynx and in front of the upper section of the trachea. The thyroid gland is made up of two lateral lobes joined by the isthmus, a small strip of thyroid tissue. The region between the second and fourth tracheal cartilages is covered by the isthmus The thyroid gland is made up of several spherical and hollow structures with a significant number of closed follicles (100 to 300 in diameter) filled with colloid, a sticky and viscous substance. Thyroglobulin is the biggest glycoprotein with substantial colloid elements, and it contains thyroid hormones within its molecule.



THYROID HORMONE:

Triiodothyronine and thyroxine, generally known as T3 and T4, are two main hormones secreted by the thyroid gland. Although thyroxine (T4) is more common than triiodothyronine (T3), T3 is the more dominant thyroid hormone, hence T3 is considered the primary thyroid hormone. Both hormones enhance the body's metabolic rate in a satisfactory manner. Thyroid secretion is principally regulated by thyroid stimulating hormone (TSH), a hormone produced by the anterior pituitary gland. The calcitonin hormone, which is vital for calcium metabolism, is also secreted by thyroid hormone. Thyroid hormone synthesis and secretion are meticulously controlled by a system that includes the hypothalamus, pituitary, and thyroid gland (HPT) axis. TRH is a tripeptide that is generated in the paraventricular nucleus of the hypothalamus and then delivered by the portal capillary plexus through axons to the median and then to the anterior pituitary. It attaches to receptors in pituitary thyrotropes, a kind of pituitary cell that aids in thyroid stimulating hormone secretion (TSH). TRH activation causes the release and synthesis of new TSH in thyrotropes, and the hormone receptors are seven-transmembrane spanning receptors. [10]

THYROID HORMONES DISORDERS:

Thyroid disorders are classified into:

- Hypothyroid,
- Hyperthyroid and
- Subclinical state. [11]

DIAGNOSIS OF HYPOTHYROIDISM

Medical History & Physical Check-up:

Doctor asks about your health history and symptoms. Then, they do a physical exam.

- **Blood Tests:**

To confirm if you really have hypothyroidism and to find out the cause, doctors do the following tests:

a. TSH Test (Thyroid Stimulating Hormone):

- This is the most important and first test done.
- If TSH is high, it usually means hypothyroidism.
- If TSH is low, it may mean hyperthyroidism.

- If TSH is slightly high but you have no symptoms, it may be subclinical hypothyroidism.
- Some doctors treat it right away. Others wait and keep checking regularly.

b. T4 Test (Thyroxine):

This checks the level of actual thyroid hormone in your blood. In hypothyroidism, T4 levels are lower than normal.

c. Thyroid Antibody Test:

This test checks if your immune system is attacking your thyroid (as in Hashimoto's disease). People with Hashimoto's usually have these antibodies. If no antibodies are found, the cause may be something else. [12]

TREATMENT

- Patients suffering from primary hypothyroidism should begin thyroid hormone (T4) replacement.
- Healthy adult patients should require about 1.6 mcg/kg per day about and 1 mcg/kg per day in adult patients over age 50 years.
- Healthy and younger patients may be started at the full prescribes dose but patients more than 50 years' age or those patients who have history of coronary artery disease should be started the dose with 25 to 5 mcg per day.
- Patients should recheck their TSH level every 6 weeks after initiation and after any change in levothyroxine dose.
- Make slight changes in levothyroxine dosing to achieve a normal TSH level.
- When the patient's TSH level is within normal range and the patient is symptom free, recheck the TSH level in 6 months.
- Patients with subclinical hypothyroidism should be treated if their TSH levels are 1mU/L or greater. Patients with subclinical hypothyroidism should be treated if TSH levels between 5 and 10 mcIU/mL remains controversial.
- Newly diagnosed pregnant women suffering from hypothyroidism disease should be treated with thyroid hormone (T4) to recover euthyroidism as soon as possible.
- During pregnancy women generally have an increased requirement for T4.
- Women with preexisting hypothyroidism who plan to become pregnant should achieve euthyroidism before conceiving. [13]

CONCLUSION

Hypothyroidism is a condition when the thyroid gland is fail to produce sufficient thyroid hormone to meet the metabolic demand of the body. Hypothyroidism is a common, potentially, serious, often clinically overlooked, readily diagnosed by laboratory testing, and eminently treatable. The available treatments for hypothyroidism are very limited. The understanding of its pathophysiology opened possibilities for rational drug design, motivating innovative approaches in the search for hormone replacement therapy.

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