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Thyroid Disorders: An Updated Review On Definition, Pathophysiology, Risk Factors, Lab Parameters & Quality Of Life.

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ABSTRACT

Thyroid disorders are among the most common endocrine conditions affecting metabolic regulation and overall health. The two major dysfunctions of the thyroid gland are hypothyroidism, characterized by insufficient production of thyroid hormones, and hyperthyroidism, characterized by excessive hormone production. Hypothyroidism results in a reduced metabolic rate and commonly presents with fatigue, weight gain, cold intolerance, constipation, and slowed physiological functions. In contrast, hyperthyroidism accelerates metabolic processes and is associated with symptoms such as weight loss, heat intolerance, tremors, tachycardia, anxiety, and increased bowel activity. Diagnosis is primarily based on

clinical presentation and thyroid function assessment. Management strategies differ according to the disorder. Levothyroxine remains the standard treatment for hypothyroidism, aiming to restore normal hormone levels and relieve symptoms. Hyperthyroidism treatment includes beta-blockers for symptomatic control, antithyroid medications to reduce hormone synthesis, radioiodine therapy, and surgical interventions when required. Early recognition and appropriate treatment are essential to prevent complications and improve patient outcomes.

KEYWORDS

Thyroid disorder

Hypothyroidism

Hyperthyroidism

Levothyroxine

Antithyroid drugs

Thyroid hormone

Metabolism

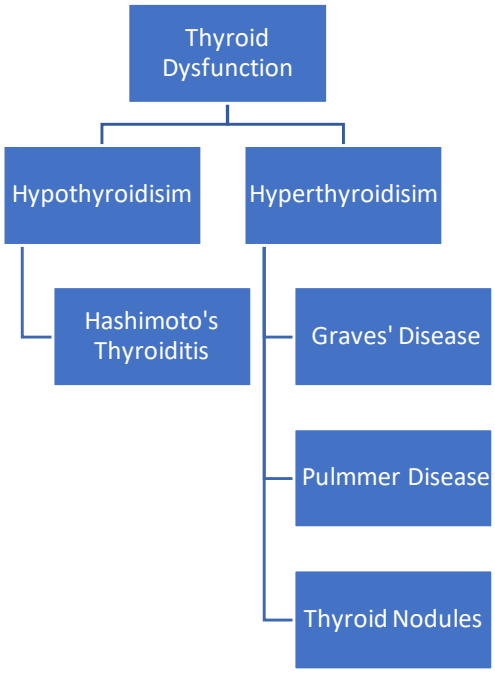
Thyroid treatment

INTRODUCTION

• The thyroid gland is an endocrine gland situated in the neck, which secretes the iodinated amino acid hormones-thyroxine (T4) and triiodothyronine (T3).

• These hormones regulate the basal metabolic rate and influence growth, development and various metabolic processes of the body. [1]

Types Of Thyroid Dysfunction :



Definition:

Hypothyroidism : Hypothyroidism is a common condition where the thyroid doesn't create and release enough thyroid hormone into bloodstream. This makes metabolism slow down. Also called underactive Thyroid.

Hyperthyroidism : Hyperthyroidism is a common condition where the thyroid Gland create and release excess amount of thyroid hormone into bloodstream. This make metabolism speed up. Also called Overactive Thyroid. [1]

Causes :

Hypothyroidism :

Primary hypothyroidism (95% of cases; thyroid gland failure affects the majority of hypothyroid patients): Iodine deficiency, enzyme defects, thyroid surgery, late-stage invasive fibrous thyroiditis, chronic autoimmune thyroiditis (Hashimoto's disease), irradiation of the thyroid after Graves' disease, iodine deficiency, medication (such as lithium, interferon), and infiltrative diseases (e.g., sarcoidosis, amyloidosis, scleroderma, hemochromatosis). Pituitary or hypothalamic neoplasms, congenital hypopituitarism, pituitary tumors, surgery, external pituitary radiation, autoimmune mechanisms, tuberculosis, and pituitary necrosis (Sheehan's syndrome) are some uncommon causes of secondary hypothyroidism (pituitary failure accounts for 5% of cases). Additionally, abnormalities of the hypothalamus and pituitary gland can lead to hypothyroidism. Patients who have undergone cerebral radiation therapy or surgery to remove a pituitary adenoma are more likely to develop certain endocrine problems. TSH blood levels can be simply used to identify hypothyroidism. Subclinical hypothyroidism is indicated by a minor increase in TSH levels together with normal T3 and T4 levels, whereas clinical hypothyroidism is indicated by high TSH levels along with low T3 and T4 levels. More people have subclinical hypothyroidism. [2]

Hyperthyroidism :

Graves' disease

Graves' disease is the most common cause of hyperthyroidism, especially in developed countries. It is an autoimmune disorder in which antibodies stimulate the thyroid-stimulating hormone (TSH) receptor, leading to excessive production of thyroid hormones. It is more common in women and often associated with goiter, ophthalmopathy, and pretibial myxedema.

Plummer Disease

Toxic multinodular goiter or Plummer Disease occurs due to multiple autonomously functioning thyroid nodules that secrete excess thyroid hormones independent of pituitary control. It is more common in elderly individuals and in regions with iodine deficiency.

Thyroid Nodules

Thyroid Nodules is a single autonomously functioning thyroid nodule that produces excess thyroid hormone. It occurs more frequently in women and is commonly seen in iodine-deficient areas [3]

Pathophysiology:

Hypothyroidism

TSH, which is created and secreted in the anterior pituitary under activation of thyrotropin releasing hormone produced in the hypothalamus, directly stimulates thyroid gland hormone synthesis. The thyroid glands metabolism is regulated by a negative feedback regulatory system in people with a healthy hypothalamic-pituitary-thyroid axis. TSH levels are controlled by the pituitary gland in response to feedback from free-thyroxine (FT4) and free-triiodothyronine (FT3) levels, which act as biosensors of thyroid hormone levels. TSH secretion is increased when thyroid hormone synthesis declines. The control system has a rather sluggish response time, and it is possible to detect some discrepancy between the levels of TSH and the plasma thyroid hormone concentrations during non-equilibrium periods, which happen at the beginning of hypothyroidism. For three main reasons, measuring TSH is regarded as the primary test for identifying thyroid illness, specifically overt and subclinical hypothyroidism. First, the concentrations of TSH and FT4 have an inverse log-linear relationship. As a result, minor linear FT4 concentration decreases are accompanied by an exponential rise in TSH levels. Second, the primary illness of the thyroid gland accounts for the majority of hypothyroidism patients in clinical practice. Thirdly, TSH immunometric tests have sensitivity and specificity of better than 99%. Finding the FT4 level is the second stage in the thyroid problem screening process. When compared to previously used measurements of total T4 or triiodothyronine, FT4 analysis is significantly less expensive.[2]

Hyperthyroidism

Graves' Disease

In this disease, the antibody and cell-mediated thyroid antigen specific immune responses are properly defined. • The development of hyperthyroidism in healthy subjects by transferring thyrotropin receptor antibodies in serum from patients with GD (Grave's disease) and the passive transfer of thyrotropin receptor antibodies to the fetus in pregnant women are the direct proof of an autoimmune disorder that is mediated by means of autoantibodies. • By circulating autoantibodies against the thyrotropin receptor, the thyroid gland is under continuous stimulation, and because of the increased production of thyroid hormones pituitary thyrotropin secretion is suppressed. • The release of thyroid hormone and thyroglobulin that is mediated via 3,'5′-cyclic adenosine monophosphate (cyclic AMP) are caused by these thyroid-stimulating SSPC, MEHSANA Page 8 INTRODUCTION antibodies, and they also stimulate iodine uptake, protein synthesis, and thyroid gland growth. • In addition to autoantigens, the cells of thyroid produce specific immune mediators such as cytokines which are involved in various immune process including complement leg isolation and T cell adhesion. Those individuals who are suffering from Graves' Disease have lesser percentage of CD4 lymphocytes in thyroid as compared to their peripheral blood. [4]

Plummer disease

Toxic multinodular goiter emerges from nontoxic multinodular goiter over a long duration. • This disease's hallmark is true functional autonomy with disordered, structural, and functional heterogeneity. The extent to which thyroid tissue has gained functional autonomy governs the transformation from nontoxic multinodular goiter to toxic multinodular goiter. • Somatic point mutations in the TSH receptor (TSHR) gene are substitutions of amino acids that constitutively cause the activation of the TSH receptor in the absence of TSH. TSHR, therefore, appears to be going from an off state to an on-state. Somatic TSHR gene mutations and G protein mutations have been thought to cause this disease, but we see only about 60% TSHR gene mutations in the nodules and even fewer G protein mutations. • Thus, many nodules have an undetermined cause of functional autonomy. Mutations in TSHR genes increase basal (TSH-induced) activation of cAMP and IP cascades, which keeps TSHR in an inactive, unliganded, but thyroid hormone activated-like state. The hyperactive nodules in Plummer disease have these mutations.

According to the 2-state model, the TSH receptor has 3 states.

These are:

• Closed - inactive

• Opened - unliganded

• Opened - hormone activated [5]

Thyroid Nodules

The pathophysiology of a thyroid nodule will vary depending on the lesion. Several disorders may cause thyroid nodules. The most common type is benign macrofollicular nodules, representing either monoclonal adenomas or colloid nodules in multinodular goiter. The latter represents the expansion of relatively monoclonal cells replicating in a nodular fashion. Follicular neoplasms may represent a diagnostic problem as these only differ from follicular carcinomas by lack of vascular or capsular invasion. The association between thyroid irradiation and tumorigenesis is well-known. Radiation may cause a wide range of somatic mutations that increase the risk of cancer, particularly in radiation-sensitive organs such as the thyroid. Compared to adults, children have a higher risk of thyroid cancer after irradiation; this is most likely due to the higher proliferative activity of the thyroid tissue in younger individuals. RET proto-oncogene translocations have been found in thyroid malignancies associated with ionizing irradiation. The presence of RET/PCT translocations has been described in follicular adenomas presenting after irradiation.[6]

Risk Factor:

Hypothyroidism

• Autoimmune disease (Hashimoto's thyroiditis) • Iodine deficiency • Thyroid surgery • Radiation therapy • Medications [2]

Hyperthyroidism

Graves' Disease

• Family history: People who get Graves' disease often have a family history of thyroid conditions or an autoimmune condition. • Sex: Women are much more likely to get Graves' disease than are men. • Age: Graves' disease mostly happens between the ages of 30 and 60. • Another autoimmune condition: People with other conditions of the immune system, such as type 1 diabetes or rheumatoid arthritis, have a higher risk. • Smoking: Cigarette smoking, which can affect the immune system, raises the risk of Graves' disease. People who smoke and have Graves' disease are at higher risk of getting thyroid eye disease.

Plummer disease

Risk Factor	Explanation / Mechanism
Age (older age)	Incidence increases with age; often manifests in people over 50, especially 60-70 yrs, because nodular changes accumulate over time.
Female sex	Women are more often affected; sex is an endogenous risk factor. Possibly due to hormonal influences (TSH fluctuations, estrogen etc.).
Family history / genetic predisposition	Some genetic factors: e.g. mutations in the TSH receptor; hereditary tendency for nodular thyroid disease.
Iodine deficiency	Chronic iodine deficiency → elevated TSH → thyroid hyperplasia → formation of nodules; over long term, some nodules become autonomous.
Elevated or prolonged TSH stimulation	TSH stimulates growth of follicular cells; constant stimulation (from e.g. low hormone levels, iodine deficiency) can encourage nodule formation; over time some nodules may escape regulation.
Smoking	Smoking is cited as a secondary risk factor; may exacerbate thyroid enlargement and contribute to nodular disease.
Other endogenous factors / environmental / drug exposures	Stress; certain drugs; possibly IGF-1 and other growth-factors stimulating thyroid tissue; sometimes radiation exposure or other endocrine disruptors are considered.

Thyroid Nodules • Iron deficiency anemia • Smoking • Obesity • Metabolic syndrome • History of thyroid radiation • Increasing age • Alcohol consumption [3]

Lab Parameter:[7-11]

Thyroid Disorders – Lab Parameters Summary

Condition	TSH (µIU/mL, ref: 0.4–4.0)	Free T4 (ng/dL, ref: 0.8–1.8)	Free T3 (pg/mL, ref: 2.3–4.2)	Antibodies / Markers	Notes
Primary Hypothyroidism	↑ High	↓ Low	Normal or ↓	Anti-TPO ↑ (common), TgAb ↑	Hashimoto's = most common cause
Subclinical Hypothyroidism	↑ Mild	Normal	Normal	Anti-TPO may be ↑	Often early Hashimoto's
Primary Hyperthyroidism	↓ Suppressed	↑ High	↑ High	Cause-specific	Palpitations, weight loss
Graves' Disease	↓ (often <0.01)	↑	↑	TRAb (TSI) ↑, Anti-TPO often ↑	Diffuse ↑ uptake on RAIU scan
Toxic Adenoma (Plummer's)	↓	↑	↑ / sometimes isolated ↑ T3	TRAb negative	Single hot nodule on scan
Toxic Multinodular Goiter	↓	↑	↑	TRAb negative	Patchy uptake on scan
Thyroid Nodules (benign / cold)	Usually, normal	Normal	Normal	No specific marker	Diagnosis via US + FNAC
Thyroid Cancer – Papillary / Follicular	Normal or ↓ if on TSH suppression therapy	Normal (or ↑ if replacement)	Normal	Thyroglobulin (Tg) marker (after surgery/RAI), TgAb interferes	Rising Tg = recurrence
Thyroid Cancer – Medullary	Normal	Normal	↓	Calcitonin ↑, CEA ↑	MTC from parafollicular C-cells

- ↓ = Decrease
- ↑ = Increase

[7-11]

Quality of life in Thyroid dysfunction [12-18]

Domain	Sub-domain	Hypothyroidism	Hyperthyroidism
1.Physical Health	Activities of daily living	Reduced due to fatigue, muscle weakness, and sluggishness.	Reduced due to restlessness, muscle wasting, and excessive activity.
	Dependence on medicinal substance	High dependence on levothyroxine (thyroid hormone replacement).	Dependence on anti-thyroid drugs or radioactive iodine/surgery.
	Energy and fatigue	Severe fatigue, lethargy and low stamina.	Excess energy but quickly exhausted; restlessness
	Mobility	May be reduced due to muscle weakness, cramps, and stiffness	May experiences tremors, muscle wasting, or weakness
	Pain & discomfort	Common (muscle and joint pain, body aches)	Less common; may have muscle tremors or palpitations
	Sleep and rest	Excessive sleepiness, lethargy	Insomnia, restlessness, disturbed sleep
	Work capacity	Decreased due to fatigue and poor concentration	Decreased due to anxiety, irritability, and lack of focus

2.Psychological Health	Bodily image appearance	Weight gain, puffy face, hair loss, dry skin, goiter	Weight loss, protruding eyes (Graves'), fine hair, goiter
	Negative & positive feelings	Depression, sadness, low mood	Anxiety, irritability, nervousness
	Self-esteem	Low due to weight gain, appearance changes, and fatigue	Low due to irritability, emotional instability, and body changes
	Spirituality/personal beliefs	May feel withdrawn or demotivated	May experiences agitation or impatience affecting spiritual well-being
	Thinking/learning /memory & concentration	"Brain fog," poor memory, slow thinking	Difficulty concentrating due to anxiety and hyperactivity
3.Social Relationships	Personal relationships	Strain due to mood changes, fatigue, and sexual dysfunction	Strain due to irritability, anxiety, and emotional Instability
	Social support	Reduced due to withdrawal and low energy	Reduced due to irritability and restlessness; may push others away
4.Environmental Health	Financial resources	Affected due to long-term treatment and decreased productivity	Affected due to medical costs and treatment follow-ups

	Freedom, physical safety, and security	Reduced physical stamina; risk of weight-related issues	Heart palpitations or fainting may slightly affect safety
	Health & social care	Lifelong hormone replacement therapy and follow-up	Frequent monitoring and treatment adjustments
	Home environment	Difficulty maintaining chores due to fatigue	Restlessness and anxiety may disrupt home routine
	Opportunity for acquiring new information & skills	Reduced due to slow cognition and memory issues	Impaired by poor focus and distractibility
	Participation in recreation	Low due to fatigue and low motivation	Low due to anxiety and restlessness
	Physical environment/Transportation	Discomfort in cold environments (cold intolerance)	Discomfort in hot environments (heat intolerance)

[12-18]

Treatment:

Hypothyroidism

First-line treatment

• Levothyroxine (LT4) is the preferred treatment: synthetic thyroxine (T4) replacement because it is stable, has uniform potency, no antigenicity etc.

• The goal is normalization of serum TSH, alleviation of symptoms and prevention of complications (e.g. myxedema).

Dosing guidelines

• For overt hypothyroidism in adults: about 1.7 µg/kg/day of levothyroxine for full replacement.

• In children, higher weight-based doses may be needed (up to ~4 µg/kg/day in some situations.

• For older patients or those with heart disease, the starting dose is lower: ~0.1 µg/kg/day or specific lower daily doses (for example 25-50 µg/d) if risk factors are present.

Other treatments / alternatives

• **Liothyronine (T3 synthetic):** It is discussed as an option, more expensive, more difficult to monitor, higher risk of cardiac side effects. Usually not first choice.

• **Liotrix** (combination T4:T3 in a fixed ratio, e.g. ~4:1) also mentioned, but again with concerns around cost, side effects, stability.[2]

Hyperthyroidism

• **Beta-blockers:** Beta-blockers, like propranolol and atenolol are often the first line of treatment for Graves' disease. These medications regulate your heart rate until other hyperthyroidism treatments take effect.

• **Antithyroid medications:** Antithyroid medications, like methimazole and propylthiouracil, block thyroid hormone production.

• **Radioiodine therapy:** This therapy slowly destroys thyroid gland cells. As your thyroid gland shrinks, hormone levels return to normal.

• **Surgery:** A thyroidectomy involves surgically removing all or part of your thyroid gland.

• Ethanol ablation

• Ultrasonography

• Thyroid lobectomy

• Treatments with iodine-131 therapy [4-6]

In Ahmedabad and Kerala (India), The commonly used medicines for hyperthyroidism are carbimazole and methimazole, while levothyroxine is commonly used for hypothyroidism.[19,20]

In Europe, the commonly medicines for hyperthyroidism are Thyrozol and Propycil, While Euthyrox and L-thyroxine is commonly used for hypothyroidism. [21]

CONCLUSION:

Thyroid dysfunction significantly influences metabolic activity and quality of life, making early diagnosis and effective management essential. Hypothyroidism and hyperthyroidism present with distinct clinical features but both require careful evaluation and individualized treatment. Levothyroxine therapy provides effective hormone replacement in hypothyroidism, while hyperthyroidism can be managed through pharmacological, radioactive, or surgical approaches depending on disease severity and patient condition. Proper monitoring and timely intervention help achieve hormonal balance, reduce symptoms, and prevent long-term complications. Increased awareness and appropriate therapeutic strategies play an important role in improving clinical outcomes in patients with thyroid disorders.

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