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Thyroid disorders: An overview of causes and treatment

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Abstract
Due to a lack of public awareness, thyroid disorders—which are more prevalent, particularly in women—frequently go untreated. In order to evaluate awareness, symptom recognition, and treatment knowledge, 200 individuals (aged 18 to 50) from both urban and rural locations were surveyed using a standardized questionnaire. The findings revealed that only 45% of respondents recognized the fundamentals of thyroid gland function, and less than 30% could recognize important signs like fatigue, weight fluctuations, and hair loss. The significance of early detection and routine screenings was not well understood by many. The study highlights the necessity of public health initiatives to raise thyroid disease knowledge and management.

Keywords: Thyroid disorders, Public Awareness, Symptom Recognition, Health Education, Early Detection

Introduction
Front of neck, two lobes + isthmus, largest endocrine gland [1]. The third week of development begins with the endoderm of the first and second pharyngeal pouches. From the primitive pharynx, the diverticulum develops, moves midline, passes in front of the hyoid bone & laryngeal cartilages, and finally rests at the level of the thyroid cartilage [2]. The thyroid follicle is made up of follicular cells and sporadic parafollicular cells surrounding the colloid [3]. Hormones: calcitonin, T3(triiodothyronine), and (tetraiodothyronine). Functions: Calcitonin → calcium regulation; T3 & T4 → growth, development, metabolism [4]. Disorders: Hyperthyroidism =↑ hormone; Hypothyroidism = ↓ hormone. Prevalence: 2-6% population Effects: Chronic health issues. Treatment: Conventional+ new drug delivery [5]. The CT capsule → trabeculae entering parenchyma → lobules [6]. The follicles range from 20 to 40 (30 toto500 µm) in size. The bulk of the gland, colloid, spherical, cuboidal, or low columnar cells are called follicles [7]. The apices of follicular cells make touch with the colloid on the basal lamina [8].

Anatomy
The thyroid is a thick, highly vascular mass situated anteriorly in the neck beyond the infrahyoid muscles, extending from the 5th cervical to 1st thoracic vertebrae [10,11]. It is brownish-reddish, with two lateral lobes (each with upper and lower poles) connected by a midline isthmus, spanning the 2nd-4th tracheal rings. Each lobe averages 8-10 mm in volume and 12-15 mm in height. The upper poles deviate laterally along the oblique lines of the thyroid cartilage, while the lower poles diverge laterally at the 5th tracheal ring [12]. The lateral side of the thyroid is surrounded by the sternothyroid muscle, which helps to keep the thyroid upper pole from expanding superiorly by attaching to the thyroid cartilage [13,14].

Histology
Trabeculae separate the thyroid parenchyma into lobules, whereas connective tissue envelops it. There are 20-40 follicles (diameter 30-500 µm) in each lobule [15]. The gland's structural and functional unit is the thyroid follicle [16].

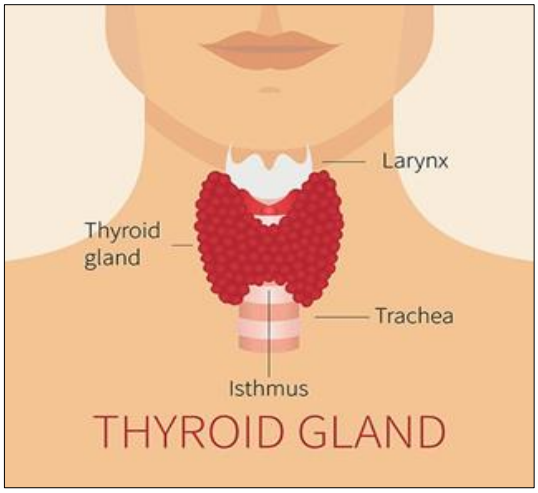

Fig1: Parts of thyroid [9]

Types of Thyroid Disease

1. **Hyperthyroidism**
2. **Hypothyroidism**

1. Hyperthyroidism

A disorder characterized by an overabundance of thyroid hormone production and secretion. Grave's disease (immune mediated glandular hyperplasia, excess hormone) is the most frequent cause [17]. Additional causes include solitary toxic adenoma and toxic multi nodular goiter. Many organs are impacted by excess hormone. **S**igns and symptoms Common: limb tremors, weight loss, and tachycardia other symptoms include increased perspiration, nausea/vomiting, polydipsia, heat sensitivity, palpitations, disrupted sleep, and hyper defecation [18].

Symptoms of Hyperthyroidism: [19]

Nervousness, irritability, palpitation, tachycardia, heat intolerance, excess sweating, tremors, weight loss/gain, appetite changes, frequent stools /diarrhea, leg edema, sudden paralysis, dyspnea, menstrual disturbance, infertility, mental and sleep disturbances, eye changes like photophobia, irritation, diplopia, fatigue, muscle weakness, thyroid enlargement (depends on cause), Pretibial myxedema (Graves' disease)

Causes of Hyoerthyroidism: [20]

Toxic diffuse goiter (Graves' disease), Toxic adenoma, Toxic multinodular goiter (Plummer's disease), Painful subacute thyroiditis, Silent thyroiditis (lymphocytic, postpartum), * Iodine-induced (e.g., amiodarone therapy), Excessive pituitary TSH / trophoblastic disease, Excess thyroid hormone ingestion

2. Hypothyroidism

An inability to produce enough thyroid hormones is known as hypothyroidism. categorized as: Thyroid gland dysfunction, or primary hypothyroidism; ↓T3, ↓T4, ↑TSH [21]. Pituitary failure to produce TSH in secondary hypothyroidism; ↓T3, ↓T4, ↓TSH levels [22]. T3 & T4 and TSH levels are measured to make the diagnosis

Symptoms of Hypothyroidism: [23]

Fatigue, Weight gain (fluid retention), Dry skin & cold intolerance, yellow skin, Coarse or loss of hair, Hoarseness, Goiter, Reflex delay (relaxation phase), Ataxia, Constipation, Memory & mental impairment, Decreased concentration, Depression, Irregular/heavy menses & infertility, Myalgias, Hyperlipidemia, Bradycardia & hypothermia, Myxedema (fluid infiltration of tissues)

Causes of Hypothyroidism

Thyroid surgery, Radiation therapy, Thyroiditis, Pituitary disorder, not enough iodine, auto immune disease

Diagnosis

The following tests are used to diagnose the thyroid disease:

1. Thyroid Antibody Test
2. Thyroglobulin (Tg) Test
3. Thyroid ultra sound Test

Thyroid Antibody Test

A. TSH

Both low FT4I and high TSH are necessary for the diagnosis of hypothyroidism because a high TSH level alone may indicate the nonthyroidal disease's recovery stage [24]. According to community-based assays, the majority of blood thyrotropin abnormalities are transient, and subclinical hypothyroidism has little clinical urgency [25]. TSH25 by itself is insufficient for diagnosis because TSH varies. Nonthyroidal disease, changing thyroid function, central hypothyroidism, hyperthyroidism with inappropriate TSH release, and central thyroid hormone resistance are among the conditions that limit the use of TSH measures [26].

B. T3 (Triiodothyronine)

Adult reference values: 3.8-10 nmol/L (0.25-2.65 ng/dL) Small unbound T3 is measured. used to check for hyperthyroidism or thyroxine over placement during pregnancy or when using medications that alter TBG, such as estrogen. Many non-thyroidal diseases (↓ T4→T3 conversion, ↓ serum TT3) are associated with low FT3. FT4 and FT3 are resistant to thyroid
hormones, and there is no hypermetabolism. Normal/high FT3 and normal/low FT4 are signs of hypothyroidism with severe iodine insufficiency [27].

C. T4 (Thyroxine)

Free Thyroxine (FT4) Adults typically have values between 1.0 and 3.0 ng/dL (13 and 39 pmol/L). A tiny portion of thyroid hormone (T4) is free, circulates in the blood, and is not bound to proteins. This free component can interact with bodily tissues and pass through cell membranes. FT4 and protein-bound T4 are in a reversible equilibrium. In accordance with the
tissue hormone level and its impact, the free hormone level remains constant. Total T4 is impacted by changes in binding proteins, although FT4 stays constant in healthy (euthyroid) individuals. In acute thyroid disease, FT4 may momentarily increase as thyroid-binding protein declines. When there is thyroid disease, FT4 may be low [27].

Thyroglobuline (Tg) TEST

The primary thyroid iodoprotein, Tg, is generated by both healthy and malignant follicular cells and discharged into the bloodstream. For differentiated thyroid carcinoma, serum Tg is a sensitive and specific tumor marker [28]. Serum levels in adults are normally between 1 and 25 ng/mL (1.5 and 38 pmol/L), with a mean of 5 to 10 ng/mL; females have somewhat greater values. Levels are two to

four times greater during the neonatal period and the third trimester of pregnancy, and they gradually decrease from infancy to puberty. Pituitary TSH has a positive connection with Tg secretion. Thyroid stimulation or tissue damage is reflected in elevated Tg, whereas little thyroid tissue or repressed activity is indicated by very low or undetectable levels. Serum Tg increases in non-TSH-mediated diseases (Graves' disease, trophoblastic illness), TBG insufficiency (TSH-mediated), and subacute thyroiditis [29, 30].

Thyroid Ultra Sound Test

ultrasound is the preferred imaging modality for diagnosing thyroid diseases, defining nodules, and determining thyroid size [31].

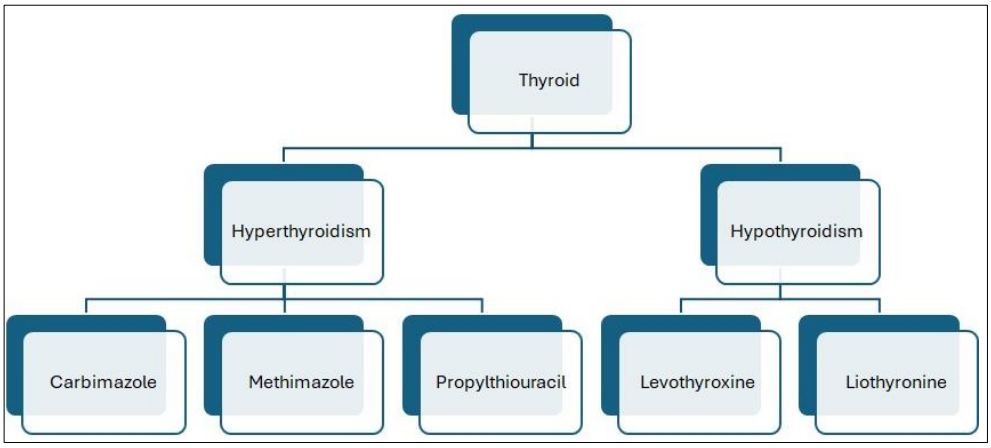

Fig 2: Drugs use to treat thyroid

Hyperthyroidism

A. Carbimazole

Carbimazole is a medicine for hyperthyroidism, a condition caused by excess thyroid hormone production. It is a prodrug converted to the active form, methimazole [32].

B. Methimazole

When surgery or RAI therapy are not appropriate for hyperthyroidism, methimazole is frequently used. MW: 114.17 g/mol, formula $C_4H_6N_2S$. The imidazole ring is composed of five members, with methyl at position one and thione at position two [33].

C. Propylthiouracil

Propylthiouracil (PTU) is used as an antithyroid medication or Graves' disease and hyperthyroidism. The review's objective**:** To lead the medical staff by discussing Action mechanism Negative occurrences the dosage. The pharmacodynamics Pharmacokinetics Observing Interactions between drugs [34].

Mechanism of Action:

The way these medications work is by blocking thyroid peroxidase, which in turn prevents the production of thyroid hormones by:

- Iodotyrosin coupling
- MIT to DIT Conversion
- Formation of MIT
- Propylthiouracil (PTU):
- Additionally inhibits peripheral deiodinase → keeps T_4 from converting to T_3
- $t½ \approx 75$ min
- Carbimazole:
- May encourage remission in Graves' disease (resulting in suppressor T cells and TSH receptor antibodies)
- $t½ = 4\text{-}6$ hours [35].

Hypothyroidism

A. Levothyroxine

Thyroxine, a main endogenous hormone secreted by the thyroid gland, can be synthesized as levothyroxine. Levothyroxine, sometimes referred to as L-thyroxine or the brand-name product Synthroid, is mainly used to treat hypothyroidism, a disorder in which the thyroid gland is unable to produce enough of the thyroid hormones T4 (tetraiodothyronine or thyroxine) and T3 (triiodothyronine or Liothyronine), which results in a reduction in the hormones' downstream effects [36].

B. Liothyronine

To treat hypothyroidism, lower TSH, and help diagnose hyperthyroidism, liothyronine, a thyroid hormone replacement drug, is utilized.

Mechanism of Action

An artificial type of thyroid hormone called liothyronine mimics the actions of native T3 by binding to thyroid receptors on DNA. In addition to supporting tissue growth, maturation, and metabolism, it also speeds up cellular oxidation, boosts energy expenditure, facilitates nervous system functions including myelination and synaptic transmission, and improves the metabolism of proteins and carbohydrates [37].

Conclusion

Thyroid disorders, particularly hypothyroidism and hyperthyroidism, continue to be a significant yet underdiagnosed health concern, especially in women. The lack of awareness about symptoms such as fatigue, weight changes, and hair loss often delays diagnosis, contributing to chronic complications. Proper understanding of thyroid anatomy, physiology, and pathology is essential for early recognition and effective management. Laboratory tests such as TSH, T3, T4, thyroglobulin, and ultrasound imaging remain the cornerstone of diagnosis. Timely interventions with antithyroid drugs, thyroid hormone replacements, and in select cases, surgery or radioiodine therapy, can restore hormonal balance and prevent long-term morbidity. Public

health education, routine screenings, and improved access to treatment are crucial to reduce the burden of thyroid disorders. Future research should emphasize advanced diagnostic tools, novel drug delivery systems, and preventive strategies to improve patient outcomes and enhance overall thyroid health.

In conclusion, thyroid disorders remain a significant yet manageable health problem. By combining advances in medical science with robust public health strategies, early detection, effective treatment, and sustained awareness can significantly reduce disease burden. A holistic approach involving healthcare providers, policymakers, and communities is essential to ensure that individuals at risk are not only diagnosed early but also empowered with the knowledge to manage their condition effectively. Addressing thyroid health is not merely a clinical priority but a vital step toward improving overall public health and quality of life.

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