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THYROID DISORDERS: A COMPREHENSIVE REVIEW OF CAUSES, DIAGNOSIS AND THERAPEUTIC MANAGEMENT

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ABSTRACT

Thyroid disorders are common endocrine conditions that can substantially affect metabolic, cardiovascular and systemic health. The thyroid gland produces thyroxine (T4) and triiodothyronine (T3), which regulate basal metabolic activity, energy expenditure, growth, development and cellular metabolism. Their synthesis is controlled by the hypothalamic-pituitary-thyroid axis through thyrotropin-releasing hormone and thyroid-stimulating hormone. Thyroid dysfunction is broadly classified as hypothyroidism or hyperthyroidism. Autoimmune disease, iodine deficiency or excess, thyroid surgery, radioactive iodine treatment and selected medicines may contribute to thyroid dysfunction. Diagnosis relies mainly on clinical assessment and biochemical testing, particularly serum TSH and free T4, with imaging and pathology used when indicated. Levothyroxine is the standard replacement treatment for primary hypothyroidism, while hyperthyroidism may be treated with antithyroid drugs, radioactive iodine or surgery. Thyroid dysfunction may also influence lipid and glucose metabolism and cardiovascular risk. This review summarizes the causes, pathophysiology, clinical manifestations, diagnosis, pharmacological management and systemic effects of thyroid disorders.

KEYWORDS: thyroid disorders; hypothyroidism; hyperthyroidism; TSH; levothyroxine; antithyroid drugs; iodine; cardiovascular risk.

1. INTRODUCTION

The thyroid gland is an important endocrine organ that produces thyroxine (T4) and triiodothyronine (T3). These hormones influence the metabolic activity of almost every cell and regulate oxygen consumption, energy production, growth and development.[1,3]

T3 is the more biologically active thyroid hormone, while a substantial proportion of circulating T3 is generated by peripheral conversion of T4. Thyroid hormone secretion is regulated by the hypothalamic-pituitary-thyroid axis. Hypothalamic TRH stimulates pituitary TSH release, which acts on the thyroid to promote T4 and T3 synthesis and secretion. Circulating thyroid hormones then provide negative feedback.[1,3]

Thyroid disorders occur when thyroid hormone production, secretion or action becomes abnormal. Hypothyroidism results from inadequate thyroid hormone activity, whereas hyperthyroidism is characterized by excessive thyroid hormone activity.[1,3] Primary hypothyroidism is commonly associated with increased TSH and reduced free T4.

Important causes include autoimmune thyroiditis, iodine deficiency or excess, thyroidectomy, radioactive iodine treatment and medicines such as amiodarone, lithium and interferon.[1,2] Symptoms of hypothyroidism may include fatigue, lethargy, cold intolerance, constipation, dry skin, voice changes and weight changes, although these features are nonspecific.[1]

TSH is a key laboratory parameter for evaluating thyroid function, while free T4 helps characterize thyroid hormone status. Hyperthyroidism may produce increased metabolic activity and systemic and cardiovascular manifestations.[1,3] Thyroid dysfunction can also influence glucose and lipid metabolism, body weight and cardiovascular function.[4]

Treatment depends on the cause and functional state. Levothyroxine is the standard treatment for primary hypothyroidism, whereas hyperthyroidism may be

managed with antithyroid drugs, radioactive iodine or surgery in selected patients.[2,3]

2. Pathophysiology and Thyroid Hormone Synthesis

Iodide is transported into thyroid follicular cells and serves as the substrate for thyroid hormone synthesis. Thyroid peroxidase (TPO) facilitates oxidation of iodide and its incorporation into thyroglobulin, producing monoiodotyrosine (MIT) and diiodotyrosine (DIT). Coupling of MIT and DIT produces T3, whereas coupling of two DIT residues produces T4. Thyroid hormones remain stored within thyroglobulin until proteolysis releases them into the circulation. In peripheral tissues, T4 is converted to the more active T3.[8]

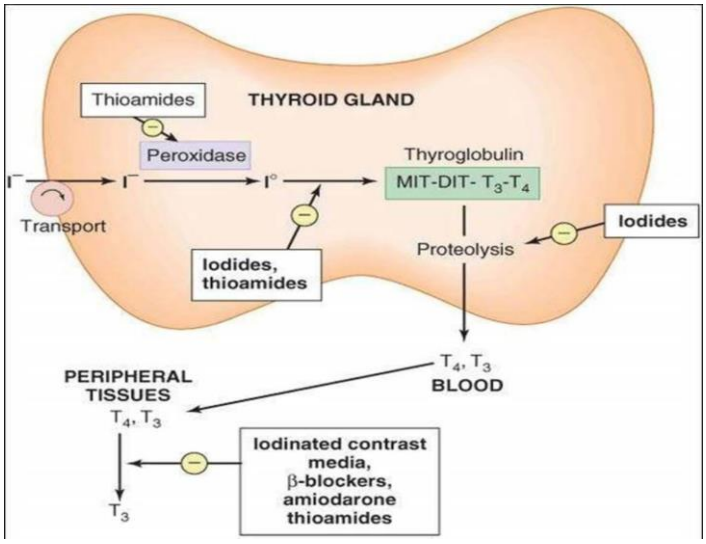

Figure 1: Major steps in thyroid hormone synthesis and peripheral conversion.

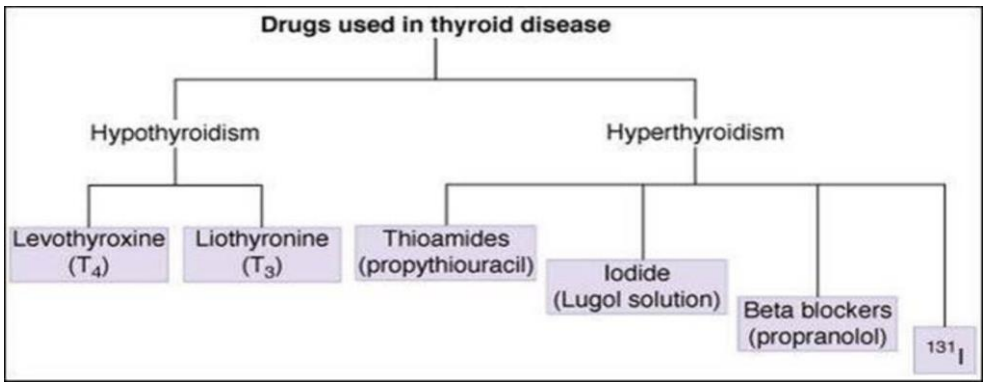

Figure 2: Overview of drug classes used in hypothyroidism and hyperthyroidism.

3. Pharmacological Management

Drug therapy is selected according to the functional disorder. In hypothyroidism, levothyroxine (T4) is the principal replacement therapy. Liothyronine (T3) is another thyroid hormone preparation but is used less commonly. In hyperthyroidism, therapeutic options include thioamides such as propylthiouracil and methimazole, iodide preparations, beta-blockers for symptomatic control and radioactive iodine in appropriate patients.[2,3]

3.1Mechanisms Relevant to Antithyroid Therapy

Thioamides inhibit thyroid peroxidase and thereby reduce iodine organification and coupling reactions required for T3 and T4 synthesis. Propylthiouracil also decreases peripheral conversion of T4 to T3.[3,8]

High-dose iodide can acutely reduce the release of preformed thyroid hormones and inhibit aspects of thyroid hormone synthesis. Because this effect may diminish with continued exposure, iodide is generally used for selected short-term clinical situations.

Beta-blockers such as propranolol reduce adrenergic manifestations of thyrotoxicosis, including palpitations and tremor. At higher doses they can also modestly reduce peripheral T4-to-T3 conversion. Amiodarone and iodinated contrast agents may also influence thyroid hormone metabolism.[3]

4. Adverse Effects of Iodine Therapy

4.1 Acute iodine sensitivity: In susceptible individuals, iodine may cause swelling of the lips or eyelids, fever, joint discomfort and skin manifestations. Severe hypersensitivity-type reactions require prompt discontinuation and clinical assessment.

4.2 Iodism due to chronic excess: Prolonged exposure to excessive iodine may produce iodism, characterized by mucosal irritation, increased salivation, nasal discharge, sneezing and excessive lacrimation.

4.3 Gastro-oral and cutaneous symptoms: Excess iodine may be associated with headache, skin rashes and a burning sensation in the mouth. These manifestations generally improve after withdrawal.

4.4 Hypothyroidism: Long-term high-dose iodine can interfere with thyroid homeostasis and may result in hypothyroidism in susceptible individuals.

4.5 Goiter: Prolonged excessive iodine intake may disturb thyroid homeostasis and contribute to goiter formation.

4.6 Acne exacerbation: Iodine exposure may aggravate acne in susceptible individuals.

4.7 Pregnancy and breastfeeding: Excessive iodine exposure may affect fetal or infant thyroid function and may contribute to goiter or hypothyroidism.

4.8 Thyrotoxicosis: Excess iodine can precipitate or worsen thyrotoxicosis, particularly in patients with multinodular thyroid disease.[9]

5. Thyroid Dysfunction and Metabolic/Cardiovascular Effects

Thyroid dysfunction has systemic effects because thyroid hormones influence lipid metabolism, glucose homeostasis, energy expenditure, vascular function and cardiovascular physiology.[4] Hypothyroidism is particularly associated with an unfavorable lipid profile, including increased total cholesterol and LDL-cholesterol concentrations.[3]

Changes in thyroid hormone status may also affect insulin sensitivity, adipose-tissue metabolism, blood pressure and endothelial function. These interactions contribute to the relationship between thyroid dysfunction, metabolic syndrome and cardiovascular disease.[1,4]

6. Diagnosis and Clinical Assessment

Accurate diagnosis begins with clinical assessment followed by appropriate biochemical testing. Serum TSH and free T4 are central to evaluation of thyroid function. Imaging, cytological examination and histopathological assessment are used when structural thyroid disease or neoplasia is suspected.[1,5,6] Standardized classification systems improve consistency in evaluation of thyroid lesions. In diagnostically difficult cases, immunocytochemical or immunohistochemical techniques may provide supportive information, but they should be interpreted together with morphology and clinical findings.[5,6]

7. Multidisciplinary Management

Optimal management may require coordination among endocrinologists, physicians, pathologists, surgeons and other healthcare professionals. Integration of clinical findings, laboratory results, imaging and pathology can improve diagnostic accuracy and support individualized treatment decisions.[5,6] Regular biochemical monitoring is important during thyroid hormone replacement and antithyroid therapy.

8. FUTURE PERSPECTIVES

Although the relationship between thyroid dysfunction, metabolic syndrome and cardiovascular disease is increasingly recognized, questions remain regarding patient-specific risk, treatment targets and long-term cardiovascular outcomes.[7] Further prospective and interventional studies are needed to clarify these relationships and identify preventive and therapeutic strategies.

9. CONCLUSION

Thyroid disorders are common endocrine conditions with effects extending beyond the thyroid gland. Abnormal thyroid hormone status can influence metabolic processes, lipid and glucose metabolism, cardiovascular function and overall health.[1–4] Accurate diagnosis using clinical assessment and appropriate biochemical testing, supplemented by imaging and pathological techniques when indicated, is essential.[5,6] Levothyroxine remains the standard treatment for primary hypothyroidism, while hyperthyroidism may be managed using antithyroid drugs, iodide, beta-blockers, radioactive iodine or surgery according to the clinical setting.[2,3]

Overall, early recognition, individualized treatment and regular monitoring, supported by multidisciplinary care, can reduce complications and improve long-term outcomes. Continued research is required to refine patient-specific treatment strategies.[4,7]

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