Hyperthyroidism: A Scientific Overview

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Key points:

- Introduction
- Etiology
- Pathophysiology
- Clinical Manifestation
- Diagnosis
- Management and Treatment

Introduction

Hyperthyroidism is a clinical disorder marked by excessive synthesis and secretion of thyroid hormones—triiodothyronine (T_3) and thyroxine (T_4)—from the thyroid gland. These hormones regulate basal metabolic rate and overall metabolic activity, and their overproduction results in a hypermetabolic state affecting multiple organ systems[1]. The condition is relatively common and occurs more frequently in women than in men[2].

Etiology (Causes)

Hyperthyroidism arises from several underlying causes. The most common is Graves' disease, an autoimmune disorder characterized by continuous stimulation of the thyroid gland[3]. Other causes include toxic multinodular goiter and toxic adenoma, which involve autonomously functioning thyroid tissue.

Inflammatory conditions such as thyroiditis may lead to transient hyperthyroidism due to the release of preformed T_3 and T_4. Excess iodine intake can precipitate hyperthyroidism (JodBasedow phenomenon), while iatrogenic causes include excessive intake of thyroid hormone medications[4].

Pathophysiology

The pathophysiology involves dysregulation of the hypothalamic–pituitary–thyroid (HPT) axis. Under normal conditions, thyroid hormone production is controlled by thyroid-stimulating hormone (TSH).

In Graves' disease, thyroid-stimulating immunoglobulins (TSIs) bind to TSH receptors, leading to continuous stimulation of the thyroid gland[5]. This results in increased production of T_3 and T_4, which elevate basal metabolic rate, oxygen consumption, and heat production.

Clinical Manifestations

The symptoms of hyperthyroidism reflect increased metabolic activity and include weight loss despite increased appetite, heat intolerance, and excessive sweating. Cardiovascular manifestations such as palpitations and tachycardia are common. Neuropsychiatric symptoms include nervousness, irritability, and anxiety. Other features include fine tremors, muscle weakness, menstrual irregularities, and goiter. In Graves' disease, specific manifestations such as ophthalmopathy (exophthalmos) and dermopathy may also occur[6].

Diagnosis

Serum TSH levels are typically suppressed, while free T_4 and T_3 levels are elevated. The presence of TSH receptor antibodies supports the diagnosis of Graves' disease. Radioactive iodine uptake (RAIU) testing helps differentiate causes based on uptake patterns, and ultrasound imaging can identify structural abnormalities[7].

Management and Treatment

Management aims to reduce thyroid hormone levels and control symptoms. Antithyroid drugs such as methimazole and propylthiouracil (PTU) inhibit hormone synthesis.

Radioactive iodine therapy is widely used to destroy overactive thyroid tissue. Beta-blockers, such as propranolol, are effective in controlling symptoms like tachycardia and tremors. Surgical intervention (thyroidectomy) may be required in selected cases

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