

# A Compressive Review of Remedies of Hyperthyroidism

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## ABSTRACT

Hyperthyroidism is a condition characterized by an overproduction of thyroid hormones, leading to an accelerated metabolism. This can result in symptoms such as weight loss, rapid heartbeat, anxiety, tremors, and fatigue. Treatment options for hyperthyroidism aim to restore normal thyroid function and alleviate symptoms. Common remedies include antithyroid medications like methimazole and propylthiouracil, which inhibit thyroid hormone production. Beta-blockers are often prescribed to manage symptoms like heart palpitations and tremors. In more severe cases, radioactive iodine therapy is employed to reduce the size of the thyroid gland and decrease hormone production. Surgery, involving the partial or total removal of the thyroid, may be necessary for individuals with large goiters or those who cannot tolerate other treatments. Additionally, lifestyle modifications such as a balanced diet and stress management techniques can help mitigate symptoms. Although these treatments are effective, they often require ongoing monitoring and adjustments to ensure proper thyroid function. This review aims to provide an overview of current treatment options for hyperthyroidism, discussing their effectiveness, side effects, and considerations for long-term management.

**Keywords:** Treatments, Pathophysiology, Epidemiology, Clinical Presentation, Complications, Adverse Effect

## INTRODUCTION

Hyperthyroidism is a metabolic condition caused by the overproduction of thyroid hormones (T3 and T4) by the thyroid gland. This disorder accelerates the body's metabolic processes, leading to symptoms such as fatigue, irritability, excessive sweating, rapid heartbeat, and unexplained weight loss. While nodular thyroid disease or thyroiditis can also trigger hyperthyroidism, the most common cause is Graves' disease, an autoimmune disorder. Managing hyperthyroidism is crucial to prevent complications like heart disease, osteoporosis, and, in severe cases, thyroid storm, a potentially life-threatening condition. Treatment options aim to control symptoms and normalize thyroid hormone levels and include beta-blockers, thyroid surgery, radioactive iodine therapy, and antithyroid medications. Each treatment has its own benefits, risks, and potential side effects, so it is important to tailor the approach based on the patient's age, health, and specific condition. Understanding these treatment options is essential for optimizing patient care and ensuring proper management of hyperthyroidism.

## OBJECTIVE

The following important aims are part of the core objective of controlling Hyperthyroidism (cml): [6]

**Overview of Hyperthyroidism:** - Provide a brief explanation of hyperthyroidism, its symptoms, causes, and complications, to set the context for discussing treatment options.

**Review of Current Treatment Modalities:** - Analyze and compare the various remedies available for hyperthyroidism, such as medication (antithyroid drugs, beta-blockers), radioactive iodine therapy, and surgery. Discuss their mechanisms of action, efficacy, and side effects.

**Alternative and Complementary Approaches:** - Examine any alternative therapies or lifestyle modifications that may help manage symptoms or complement traditional treatments (e.g., diet, herbal remedies, stress management).

**Challenges and Limitations:** - Highlight the challenges of treating hyperthyroidism, such as the

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risk of overtreatment, long-term side effects, or recurrence of symptoms.

**Future Directions:** - Explore the emerging therapies and innovations in the treatment of hyperthyroidism, including personalized medicine, gene therapy, and new drug developments.

**Patient-Centered Care:** - Emphasize the importance of a personalized approach to treatment based on individual patient needs, preferences, and the severity of the condition.

The objective is to provide a comprehensive review of available remedies, critically assessing their effectiveness and potential future advancements in hyperthyroidism treatment.

## TREATMENTS

Hyperthyroidism can be treated through several approaches, each with its advantages and considerations. The first-line treatment often involves antithyroid medications, such as methimazole and propylthiouracil, which work by inhibiting the thyroid gland's ability to produce excessive hormones. These medications can provide long-term control but may require monitoring for side effects, such as liver damage or blood abnormalities. Beta-blockers, like propranolol, are frequently used to control symptoms such as tachycardia, anxiety, and tremors, as they block the effects of excess thyroid hormones on the body's systems, though they do not address the underlying thyroid dysfunction. For patients who do not respond to medications, radioactive iodine therapy is a widely used treatment. This therapy involves the oral intake of radioactive iodine, which is absorbed by the thyroid and selectively destroys overactive thyroid cells, ultimately reducing hormone production. While effective, it often results in hypothyroidism, necessitating lifelong thyroid hormone replacement therapy. In some cases, surgery, such as a thyroidectomy, is recommended, especially when the goiter is large or when other treatments are ineffective. The removal of part or all of the thyroid gland can be a permanent solution but also carries the risk of complications, including damage to the parathyroid glands or vocal cords. Each treatment modality is selected based on factors such as the

severity of the condition, patient age, and overall health, with ongoing monitoring to ensure proper management of thyroid function.

## ANTI-THYROID DRUGS

### Overview: -

antithyroid medications Propylthiouracil, carbimazole, and thiamazole are antithyroid thionamide medications. They are all actively carried into the thyroid, where they prevent the oxidation of iodide. and organization by blocking thyroid peroxidase and the iodotyrosine coupling that produces T4 and T3. 56 Some European and Asian nations have carbimazole, which is transformed into thiamazole, the active form, which shares characteristics with thiamazole. By blocking the outer ring deiodinase of T4, propylthiouracil, but not thiamazole, reduces the conversion of T4 to T3 in peripheral tissues at high dosages. 57 These medications may also have immunosuppressive and anti-inflammatory properties.

### Protocols for ATD therapy and follow-up: -

Titration and block and replace are the two methods used to treat Graves' illness. Titration involves gradually reducing the dosage of ATD to the lowest amount required to maintain a euthyroid condition. 66 Within the block and replace regimen, levothyroxine is administered in conjunction with a greater dose of ATD. Although the block and replace regimen appears to be linked to a higher frequency of side effects than the titration approach, the two regimens are equally effective. 67 Thus, even though some experts believe both techniques are equally safe, the titration regimen ought to be the first-line strategy<sup>44</sup>. The starting dose of thiamazole depends on the severity of the hyperthyroidism and the size of the thyroid gland: mild hyperthyroidism and small glands need 10–15 mg of thiamazole daily, and severe hyperthyroidism and large thyroids need 20–40 mg daily. The equivalent dose of carbimazole is 140% of that of thiamazole. The starting dose of propylthiouracil is usually 50–150 mg administered three times daily. Thyroid function should be checked 4–6 weeks after initiation of therapy and then every 2–3 months once the patient is euthyroid,<sup>44</sup> although

we usually see the patient every 4 months when they are euthyroid. TSH might remain suppressed for several months, which is why serum T4 and T3 should be monitored to assess efficacy of therapy. Once euthyroidism is achieved, a maintenance dose of thiamazole of 5– 10 mg daily, or 50 mg propylthiouracil two or three times daily, or lower, should be continued for 12–18 months,<sup>69</sup> and some suggest an even longer duration of therapy

## EPIDEMIOLOGY

The epidemiology of hyperthyroidism refers to the study of its distribution, patterns, and determinants within populations. Key points for the epidemiology of hyperthyroidism include:

### 1) Prevalence: -

-Hyperthyroidism is a relatively common endocrine disorder, though its prevalence varies across different populations.

-Studies suggest that approximately 1-2% of the general population may have hyperthyroidism.

-The condition is more prevalent in women than men, with a ratio of about 5:1 in adults.

-The incidence increases with age, particularly in individuals over 60 years.

-Graves' disease, the most common cause of hyperthyroidism, is typically seen in younger individuals, while toxic multinodular goiter is more common in older adults.

### 2) Age and Gender: -

-Hyperthyroidism is most prevalent in individuals aged 20 to 40 years (especially Graves' disease).

-It is more common in women (particularly during reproductive years) than in men, but in older age groups, the gender gap tends to narrow.

-The incidence of toxic nodular goiter increases with age and is particularly significant in older adults, especially in regions with iodine deficiency.

### 3) Geographical Variations: -

-The prevalence of hyperthyroidism varies globally, depending on factors like iodine intake, environmental factors, and healthcare access.

-In areas where iodine deficiency is prevalent (e.g., parts of Africa, Asia), the incidence of hyperthyroidism due to goiter is higher.

-In iodine-sufficient regions (e.g., North America, Europe), the most common causes of hyperthyroidism are Graves' disease and toxic multinodular goiter

### 4) Risk Factors: -

-Genetics: A family history of thyroid disease, particularly autoimmune thyroid disorders, increases the risk.

-Autoimmune Diseases: Individuals with other autoimmune disorders (e.g., type 1 diabetes, rheumatoid arthritis) are at higher risk for Graves' disease.

-Iodine Intake: Both iodine deficiency and excessive iodine intake can increase the risk of hyperthyroidism.

-Gender and Hormonal Factors: Women are more susceptible, particularly during periods of hormonal changes (e.g., pregnancy,

-Radiation Exposure: Exposure to radiation, especially in childhood, increases the risk of developing hyperthyroidism later in life.

-Smoking: Smoking is a significant risk factor for developing Graves' disease.

### 5) Morbidity and Mortality: -

-Hyperthyroidism, if left untreated, can lead to serious complications, including thyroid storm, cardiovascular problems (e.g., atrial fibrillation), and osteoporosis.

-Cardiovascular issues, particularly arrhythmias and heart failure, are major contributors to morbidity in hyperthyroid patients, especially in the elderly.

-Mortality rates have decreased with the development of better treatments, but untreated hyperthyroidism still carries a risk of severe complications.

### 6) Trends Over Time: -

-The incidence of hyperthyroidism has remained relatively stable in iodine-sufficient areas, though improvements in diagnostic methods have led to the identification of milder cases.

-The prevalence of Graves' disease appears to have decreased in some regions due to better management and detection, while toxic multinodular goiter is



becoming more prominent, particularly in older populations.

### **Pathophysiology of Hyperthyroidism**

Environmental factors such as dietary iodine, smoking, and emotional stress can precipitate GD in genetically predisposed individuals who harbour multiple susceptibility alleles (in particular at the MHC, CTLA4, and PTPN22 loci). As an organ-specific autoimmune disease, GD is caused by circulating autoantibodies (generally of the IgG1 isotype) directed against the TSH-receptor (TSHR). In the majority of patients TSHR autoantibodies (TRAb) stimulate the thyroid after binding to the TSHR, by increasing the production of intracellular cyclic AMP. TRAbs also interact with IGF1 receptors on the surface of thyroid cells and of orbital fibroblasts. The antibody-receptor complex promotes activation of intracellular cyclic AMP with ensuing thyrocyte hyperplasia (causing gland enlargement), increased vascularity, increased thyroid hormone production and secretion, resulting in hyperthyroidism (14). Expression of TSHR outside the thyroid gland has been observed in retro-orbital and pretibial tissues, causing some of the extrathyroidal manifestations of the disease. In addition to genetic susceptibility, smoking is a noticeable environmental dose-dependent risk factor for GD and especially for Graves' ophthalmopathy (GO). Smoking is associated with recurrence of GD hyperthyroidism as well as a higher risk of worsening eye changes related to GO after radioiodine therapy and a less favourable outcome of GO treatment with steroids or retrobulbar irradiation. Although the exact pathophysiologic basis of this effect is unclear, it might be related to the fact that cigarette smoking enhances the production of Interleukin-1 during the immune response, therefore possibly stimulating growth of orbital fibroblasts. Smoking may also induce hypoxia in Downloaded from [jnm.snmjournals.org](http://jnm.snmjournals.org) by Auckland University of Technology on October 3, 2020. For personal use only. 7 fibroblasts, enhancing hyaluronic acid production and adipogenesis (12,15). Polymorphisms in genes encoding for enzymes involved in the biotransformation of xenobiotics are associated with smoking-related GD susceptibility (16). Anti-thyroglobulin and anti-thyroperoxidase antibodies are

often present in the serum of patients with GD. TMNG causes hyperthyroidism when non-functioning and TMNG causes hyperthyroidism when non-functioning and functioning nodules coexist and with time become autonomous, secreting thyroid hormone independent of circulating levels of TSH (17). Follicles in TMNG have a heterogeneous pattern of iodine uptake, displaying either low ("cold follicle") or high ("hot follicle") iodine uptake on radioiodine scan (18). TA is a monoclonal well-defined encapsulated benign tumor secreting thyroid hormone in the absence of a TSH stimulus, in an otherwise normal gland. It is characterized by increased iodine transport and iodination rate with increased expression of thyroperoxidase (TPO) and sodium/iodide symporter (NIS) mRNAs. In the majority of cases an activating gain-of-function TSHR mutation confined to the adenomatous tissue (somatic mutation) results in stimulation of adenylcyclase and intracellular c-AMP accumulation with consequent increased replication rate of thyroid cells (19,20) Similar to toxic adenoma, gain-of-function TSHR mutations are responsible for the genesis of hyperfunctioning nodules in TMNG (17). Germline mutations of the same gene are responsible for other rare familial forms of non-immune hyperthyroidism (20). The prevalence of somatic TSHR mutations is up to 82% of TAs and to 60% of hyperfunctioning nodules within a TMNG (21). There is also a critical role of iodine deficiency in the development of thyroid autonomy (22)

### **Clinical Presentation and Complications**

#### **Signs and symptoms due to excess thyroid hormones**

Excess thyroid hormone affects many different organ systems. Commonly reported symptoms are palpitations, fatigue, tremor, anxiety, disturbed sleep, weight loss, heat intolerance, sweating, and polydipsia. Frequent physical findings are tachycardia, tremor of the extremities, and weight loss. Signs and symptoms specific to the underlying causes of hyperthyroidism Signs and symptoms include ophthalmopathy, thyroid dermopathy, and thyroid acropachy in Graves' disease; globus sensation, dysphagia, or orthopnoea due to



oesophageal or tracheal compression in nodular goitre; and anterior neck pain in painful subacute thyroiditis. Ophthalmopathy, also known as Graves' orbitopathy, occurs in 25% of patients with Graves' disease. The main signs are proptosis, periorbital oedema, and diplopia. Clinicians who do not have expertise in managing active or moderate-to-severe Graves' orbitopathy should refer patients to a combined thyroid–eye clinic for assessment and management. Thyroid dermopathy is a rare extrathyroidal manifestation of Graves' disease, occurring in 1–4% of patients with thyroid ophthalmopathy. Almost all patients have coexisting ophthalmopathy.

The lesions are characterised by slightly pigmented thickened skin, primarily involving the pretibial area. Acropachy is the rarest extrathyroidal manifestation of Graves' disease and presents with clubbing of the fingers and toes.

### Complications Seen in Hyperthyroidism

Clinical manifestation varies depending on several factors, such as the patient's age and sex, comorbidities, duration of the disease, and cause. Older patients present with fewer and less pronounced symptoms than do younger patients, but are more likely to develop cardiovascular complications. When compared with people older than 60 years with a healthy thyroid, those who are hyperthyroid have three times the risk of atrial fibrillation. Embolic stroke related to atrial fibrillation secondary to hyperthyroidism is significantly more prevalent than embolic stroke related to atrial fibrillation from non-thyroidal causes. However, anticoagulant therapy in patients with atrial fibrillation secondary to hyperthyroidism is still debated. Atrial fibrillation is also thought to be an independent predictor of the development of congestive heart failure in patients with hyperthyroidism.

### Adverse Effect/Side Effects

The remedies for hyperthyroidism (overactive thyroid) include medications, radioactive iodine therapy, and surgery. Each treatment has potential adverse effects, depending on the method used:

### 1. Antithyroid Medications (E.G., Methimazole, Propylthiouracil): -

- ❖ Common Side Effects:-
  - Rash or itching
  - Joint pain
  - Nausea or upset stomach
- ❖ Serious (rare) Side Effects:
  - Agranulocytosis (dangerously low white blood cell count)
  - Liver damage (especially with Propylthiouracil)
  - Vasculitis (inflammation of blood vessels)

### 2. Radioactive Iodine Therapy: -

- ❖ Common Side Effects:
  - Neck tenderness
  - Temporary increase in thyroid hormone levels
- ❖ Long-term Effect:
  - Hypothyroidism (underactive thyroid), which often requires lifelong thyroid hormone replacement therapy

### 3. Surgery (Thyroidectomy): -

- ❖ Risks and Side Effects:
  - Bleeding or infection
  - Damage to the recurrent laryngeal nerve (can affect voice)
  - Hypoparathyroidism (low calcium due to accidental damage/removal of parathyroid glands)
  - Permanent hypothyroidism

## CONCLUSION OF TREATMENTS

Epidemiological studies suggest that hyperthyroidism is a significant public health concern with gender, age, genetic, and environmental risk factors influencing its prevalence. The condition is more common in women, especially those of reproductive age, and its prevalence increases with age. Public health measures, such as iodine supplementation, can influence the distribution of hyperthyroidism in populations



## REFERENCE

1. Carnell NE, Valente WA. Thyroid nodules in Graves' disease: classification, characterization, and response to treatment. *Thyroid* (1998) 8:571–6. doi: 10.1089/thy.1998.8.571
2. Gossage A, Munro D. The pathogenesis of Graves' disease. *Clin Endocrinol Metab.* (1985) 14:299–330. doi: 10.1016/S0300-595X(85)80036-0
3. Burman K, Baker J. Immune Mechanisms in Graves' disease. *Endocr Rev.* (1985) 6:183–223. doi: 10.1210/edrv-6-2-183
3. McKenna TJ. Graves' disease. *Lancet* (2001) 357:1793–6. doi: 10.1016/S0140-6736(00)04906-0
4. Hirota Y, Tamai H, Hayashi Y, Matsubayashi S, Matsuzuka F, Kuma K, et al. Thyroid function and histology in forty-five patients with hyperthyroid Graves' disease in clinical remission more than ten years after thionamide drug treatment. *J Clin Endocrinol Metab.* (1986) 62:165–9. doi: 10.1210/jcem-62-1-165
5. Bitton RN, Sachmechi I, Tabriz MS, Murphy L, Wasserman P. Papillary carcinoma of the thyroid with manifestations resembling Graves' disease. *Endocr Pract.* (2001) 7:106–9. doi: 10.4158/EP.7.2.106
6. Braga M, Graf H, Ogata A, Batista J, Hakim NC. Aggressive behavior of papillary microcarcinoma in a patient with Graves' disease initially presenting as cystic neck mass. *J Endocrinol Invest.* (2002) 25:250–3. doi: 10.1007/BF03343999
7. Thomas FB, Mazzaferri EL, Skillman TG. Apathetic thyrotoxicosis: A distinctive clinical and laboratory entity. *Ann Intern Med.* (1970) 72:679–85. doi: 10.7326/0003-4819-72-5-679
8. Johnson PC, Kahil ME. Apathetic hyperthyroidism. A type of masked thyrotoxicosis. *Tex Med.* (1967) 63:59–62.
22. Wu W, Sun Z, Yu J, Meng Q, Wang M, Miao J, et al. A clinical retrospective analysis of factors associated with apathetic hyperthyroidism. *Pathobiology* (2010) 77:46–51. doi: 10.1159/000272954
9. Siddiqui AR, Karanauskas S. Hurthle cell carcinoma in an autonomous thyroid nodule in an adolescent. *Pediatr Radiol.* (1995) 25:568–9. doi: 10.1007/BF02015798
10. Smith M, McHenry C, Jarosz H, Lawrence AM, Paloyan E. Carcinoma of the thyroid in patients with autonomous nodules. *Am Surg.* (1988) 54:448–9.
11. Tfayli HM, Teot LA, Indyk JA, Witchel SF. Papillary thyroid carcinoma in an autonomous hyperfunctioning thyroid nodule: case report and review of the literature. *Thyroid* (2010) 20:1029–32. doi: 10.1089/thy.2010.0144
12. Siddiqui IN, Friedman J, Barry-Holson KQ, Ma C, Thodima V, Kang I, et al. Characterization of a variant of t(14;18) negative nodal diffuse follicular lymphoma with CD23 expression, 1p36/TNFRSF14 abnormalities, and STAT6 mutations. *Mod Pathol.* (2016) 29:570–81. doi: 10.1038/modpathol.2016.51
13. Vinciguerra GL, Nocchioli N, Bartolazzi A. Diffuse follicular variant of papillary thyroid carcinoma: a case report with a revision of literature. *Rare Tumors* (2016) 8:6536. doi: 10.4081/rt.2016.6536
14. Sobrinho-Simões M, Soares J, Carneiro F, Limbert E. Diffuse follicular variant of papillary carcinoma of the thyroid: report of eight cases of a distinct aggressive type of thyroid tumor. *Surg Pathol.* (1990) 3:189–203.
15. Mori T. [Hashitoxicosis and Hashimoto's disease with the symptoms of thyrotoxicosis]. *Nihon Rinsho* (1980) 38:1677–83.
16. Nabhan ZM, Kreher NC, Eugster EA. Hashitoxicosis in children: clinical features and natural history. *J Pediatr.* (2005) 146:533–6. doi: 10.1016/j.jpeds.2004.10.070
16. Wasniewska M, Corrias A, Salerno M, Lombardo F, Aversa T, Mussa A, et al. Outcomes of children with hashitoxicosis. *Horm Res Paediatr.* (2012) 77:36–40. doi: 10.1159/000334640
17. Waldhausl W, Bratusch-Marrain P, Nowotny P, Buchler M, Forssmann WG, Lujf A, et al. Secondary hyperthyroidism due to thyrotropin hypersecretion: study of pituitary tumor morphology and thyrotropin chemistry and release. *J Clin Endocrinol Metab.* (1979) 49:879–87. doi: 10.1210/jcem-49-6-879

18. Gomez JB, Diaz MA, Jerez ML. [Tertiary hyperthyroidism. Criteria of evaluation]. *Rev Med.* (1973) 17:231–9.
19. Kourides I, Ridgway E, Weintraub B, Bigos S, Gershengorn M, Maloof F. Thyrotropin-induced hyperthyroidism: use of alpha and beta subunit levels to identify patients with pituitary tumors. *J Clin Endocrinol Metab.* (1977) 45:534–43. doi: 10.1210/jcem-45-3-534
20. Dorfman SG. Hyperthyroidism. Usual and unusual causes. *Arch Internal Med.* (1977) 137:995–6. doi: 10.1001/archinte.1977.03630200005005
21. Tolis G, Bird C, Bertrand G, McKenzie J, Ezrin C. Pituitary hyperthyroidism. *Am J Med.* (1978) 64:177–181. doi: 10.1016/0002-9343(78)90202-4.

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