Bülten III
Tirotoksikoz

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1. ETİYOLOJİ


Thyrotoxicosis.

Seigel SC¹, Hodak SP.

Author information:

Abstract

Hyperthyroidism describes the sustained increase in thyroid hormone biosynthesis and secretion by a thyroid gland with increased metabolism. Although the use of radioiodine scanning serves as a useful surrogate that may help characterize the cause of thyrotoxicosis, it only indirectly addresses the underlying physiologic mechanism driving the increase in serum thyroid hormones. In this article, thyrotoxic states are divided into increased or decreased thyroid metabolic function. In addition to the diagnosis, clinical presentation, and treatment of the various causes of hyperthyroidism, a section on functional imaging and appropriate laboratory testing is included.

Hyperthyroidism.

De Leo S¹, Lee SY², Braverman LE³.

Abstract

Hyperthyroidism is characterised by increased thyroid hormone synthesis and secretion from the thyroid gland, whereas thyrotoxicosis refers to the clinical syndrome of excess circulating thyroid hormones, irrespective of the source. The most common cause of hyperthyroidism is Graves' disease, followed by toxic nodular goitre. Other important causes of thyrotoxicosis include thyroiditis, iodine-induced and drug-induced thyroid dysfunction, and factitious ingestion of excess thyroid hormones. Treatment options for Graves' disease include antithyroid drugs, radioactive iodine therapy, and surgery, whereas antithyroid drugs are not generally used long term in toxic nodular goitre, because of the high relapse rate of thyrotoxicosis after discontinuation. β blockers are used in symptomatic thyrotoxicosis, and might be the only treatment needed for thyrotoxicosis not caused by excessive production and release of the thyroid hormones. Thyroid storm and hyperthyroidism in pregnancy and during the post-partum period are special circumstances that need careful assessment and treatment.

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**Pathogenesis of Hyperthyroidism.**

Singh I¹, Hershman JM¹.

Abstract

Hyperthyroidism is a form of thyrotoxicosis in which there is excess thyroid hormone synthesis and secretion. Multiple etiologies can lead to a common clinical state of "thyrotoxicosis," which is a consequence of the high thyroid hormone levels and their action on different tissues of the body. The most common cause of thyrotoxicosis is Graves' disease, an autoimmune disorder in which stimulating thyrotrpin receptor antibodies bind to thyroid stimulating hormone (TSH) receptors on thyroid cells and cause overproduction of thyroid hormones. Other etiologies include: forms of thyroiditis in which inflammation causes release of preformed hormone, following thyroid gland insult that is autoimmune, infectious, mechanical or medication induced; secretion of human chorionic gonadotropin in the setting of transient gestational thyrotoxicosis and trophoblastic tumors; pituitary thyrotropin release, and exposure to extra-thyroidal sources of thyroid hormone that may be endogenous or exogenous. © 2017 American Physiological Society. Compr Physiol 7:67-79, 2017.
2. MEDİKAL VE RADYOAKTİF İYOT TEDAVİSİ


Medical treatment of hyperthyroidism: state of the art.
Fumarola A1, Di Fiore A, Dainelli M, Grani G, Calvanese A.

Abstract
Methimazole (MMI) and propylthiouracil (PTU) are the main antithyroid drugs used for hyperthyroidism. They inhibit the synthesis of thyroid hormone at various levels and are used as the primary treatment for hyperthyroidism or as a preparation before radiiodine therapy or thyroidectomy. MMI is the drug of choice because of its widespread availability, longer half-life and small number of severe side effects. Drugs of second choice are potassium perchlorate, beta blockers, iodine, lithium carbonate and glucocorticoids. Rituximab, a monoclonal antibody directed against human CD20, was recently proposed as a biological therapy for cases of Graves' disease unresponsive to traditional drugs.

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Radioactive iodine therapy.

Lee SL1.

Abstract

PURPOSE OF REVIEW:
Review of the management decisions that must be made by the endocrinologist during the use of radioactive iodine (RAI) therapy of hyperthyroidism and differentiated thyroid cancer.
RECENT FINDINGS:

Since the 1940s radioactive (131)I (RAI) therapy has been a major component of the treatment of hyperthyroidism and differentiated thyroid cancer. RAI is the most common definitive treatment of hyperthyroidism. Pretherapy decisions including use of antithyroid medication and low-iodine diet will be discussed with the relevant supportive literature. The method of semi-quantitative calculation used for RAI treatment of hyperthyroidism will be described. Evidence-based guideline for the management of differentiated thyroid cancer by the American Thyroid Association, new drug development and recent randomized controlled trials have changed current practice of how RAI is used for remnant ablation and adjuvant therapy of differentiated thyroid cancer.

SUMMARY:

RAI is a common tool for the endocrinologist in the management of hyperthyroidism and differentiated thyroid cancer. Review of the management decisions and practice of RAI therapy will educate the endocrinologist of the literature supporting current RAI use in hyperthyroidism and new developments in limiting the radiation exposure to the patients with differentiated thyroid cancer.
Primary hyperthyroidism--diagnosis and treatment. Indications and contraindications for radioiodine therapy.

Gurgul E, Sowinski J.

Abstract

Isotope therapy is one of the methods used in primary hyperthyroidism. The therapy is based on short-range beta radiation emitted from radioactive iodine. Radioiodine administration must always be preceded by pharmacological normalization of thyroid function. Otherwise, post-radiation thyrocyte destruction and thyroid hormones release may lead to hyperthyroidism exacerbation. Indications for radioiodine therapy in Graves-Basedow disease include recurrent hyperthyroidism after thyrostatic treatment or thyroidectomy and side-effects observed during thyrostatic treatment. In toxic nodule, isotope therapy is the first choice therapy. Radioiodine is absorbed only in autonomous nodule. Therefore, it destroys only this area and does not damage the remaining thyroid tissue. In toxic goitre, radioiodine is used mostly in recurrent nodules. Absolute contraindications for radioiodine treatment are pregnancy and lactation. Relative contraindications are thyroid nodules suspected of malignancy and age under 15 years. In patients with thyroid nodules suspected of malignancy, radioiodine treatment may be applied as a preparation for surgery, if thyrostatic drugs are ineffective or contraindicated. In children, radioiodine therapy should be considered in recurrent toxic goitre and when thyrostatic drugs are ineffective. In patients with Graves-Basedow disease and thyroid-associated orbitopathy, radioiodine treatment may increase the inflammatory process and exacerbate the ophthalmological symptoms. However, thyroid-associated orbitopathy cannot be considered as a contraindication for isotope therapy. The potential carcinogenic properties of radioiodine, especially associated with tissues with high iodine uptake (thyroid, salivary glands, stomach, intestine, urinary tract, breast), have not been confirmed.
The Second Antithyroid Drug Treatment Is Effective in Relapsed Graves' Disease Patients: A Median 11-Year Follow-Up Study.

Kim YA, Cho SW, Choi HS, Moon S, Moon JH, Kim KW, Park DJ, Yi KH, Park YJ, Cho BY.

Abstract

BACKGROUND:

Antithyroid drug (ATD) is a widely used treatment for Graves' disease (GD). However, its long-term efficiency remains unclear. This study investigated the long-term disease prognosis and predictive factors for relapse in ATD-treated GD patients.

METHODS:

Newly diagnosed, ATD-treated GD patients with at least four years of follow-up were recruited (n = 187). Remission was defined as maintaining a euthyroid status for more than one year after ATD withdrawal.

RESULTS:

During 11.1 years (range 4.0-23.7 years) of median follow-up, overall, 51.9% of the newly diagnosed ATD-treated GD patients achieved remission, 32.1% continued ATD treatment, and 13.4% underwent other ablation treatments. The 10-year remission rates were higher in the first (34.2%) and second (25.5%) ATD courses than in any of the other subsequent ATD courses, and decreased as ATD treatments were repeated. The 10-year relapse rate was the highest after the third ATD treatment (71.4%) compared with that after the first (60.5%) and second (58.3%) courses. Longer duration of ATD treatment (odds ratio [OR] = 1.4 [confidence interval (CI) 1.2-1.7], \( p < 0.001 \)), higher number of relapses (OR = 4.7 [CI 2.3-9.8], \( p < 0.001 \)), and moderate to severe Graves' ophthalmopathy (OR = 4.1 [CI 1.1-15.2], \( p = 0.032 \)) were associated with persistent disease status.

CONCLUSIONS:

A second course of ATD can be considered for GD patients after the first relapse because the chance of remission and the relapse rate are similar to the one after the first ATD treatment course. For GD patients with more than two relapses, or with an ATD treatment duration of more than four to five years, low-dose maintenance of ATD or ablative treatment needs to be considered.
Advances in the pharmacological treatment of Graves' orbitopathy.
Ruchala M¹, Sawicka-Gutaj N¹.

Abstract

Graves' orbitopathy has a deteriorating effect on patients' appearance and vision, thus significantly decreases their quality of life. A multidisciplinary team of endocrinologists, ophthalmologists, head and neck surgeons, nuclear medicine physicians, radiologists, and psychologists should constitute a standard health care team for those patients. It is vital that the therapy is based on an individual approach, with patients being well informed and involved in the decision-making process. Generally, traditional therapies include immunosuppression with steroids, orbital irradiation and surgical decompression. Novel treatment modalities include: biological agents, somatostatin analogs, antioxidants, methotrexate. Better insight into pathogenesis of Graves' orbitopathy is the only chance for targeted therapy development.

Pathogenesis of thyroid eye disease: review and update on molecular mechanisms.
Khong JJ¹, McNab AA², Ebeling PR³, Craig JE⁴, Selva D⁵.

Abstract

Orbital changes in thyroid orbitopathy (TO) result from de novo adipogenesis, hyaluronan synthesis, interstitial oedema and enlargement of extraocular muscles. Cellular immunity, with predominantly CD4+ T cells expressing Th1 cytokines, and overexpression of macrophage-derived cytokines, perpetuate orbital inflammation. Orbital fibroblasts appear to be the major effector cells. Orbital fibroblasts express both thyrotropin receptor (TSHR) and insulin-like growth factor-1 receptor (IGF-1R) at higher levels than normal fibroblasts. TSHR expression increases in adipogenesis; TSHR agonism enhances hyaluronan production. IGF-1R stimulation leads to adipogenesis, hyaluronan synthesis and production of the chemokines, interleukin (IL)-16 and Regulated on Activation, Normal T Cell Expression and Secreted, which facilitate lymphocyte trafficking into the orbit. Immune activation uses a specific CD40:CD154 molecular bridge to activate orbital fibroblasts, which secrete pro-inflammatory cytokines including IL-1β, IL-1α, IL-6, IL-8, macrophage chemoattractant protein-1 and transforming growth factor-β, to perpetuate orbital
inflammation. Molecular pathways including adenylyl cyclase/cyclic adenosine monophosphate, phosphoinositide 3 kinase/AKT/mammalian target of rapamycin, mitogen-activated protein kinase are involved in TO. The emergence of a TO animal model and a new generation of TSHR antibody assays increasingly point towards TSHR as the primary autoantigen for extrathyroidal orbital involvement. Oxidative stress in TO resulting from imbalances of the oxidation-reduction state provides a framework of understanding for smoking prevention, achieving euthyroidism and the use of antioxidants such as selenium. Progress has been made in the understanding of the pathogenesis of TO, which should advance development of novel therapies targeting cellular immunity, specifically the CD40:CD40 ligand interaction, antibody-producing B cells, cytokines, TSHR and IGF-1R and its signalling pathways. Further studies in signalling networks and molecular triggers leading to burnout of TO will further our understanding of TO.


**Total thyroid ablation in Graves' orbitopathy.**

**Menconi F**, **Leo M**, **Vitti P**, **Marcocci C**, **Marinò M**.

**Abstract**

Graves' orbitopathy (GO) is an autoimmune condition almost always associated with autoimmune thyroid disease, especially Graves' disease (GD). According to the most widely accepted model, the autoantigens responsible for GO would include molecules expressed by thyroid epithelial cells that are present also in orbital tissues. The high likelihood that the etiologies of GO and of the underlying autoimmune thyroid diseases are somehow linked is confirmed by the very close relationship between GO, the onset and the course of Graves' diseases, the size of the thyroid gland, and most importantly, thyroid function and thyroid treatment. Based on these considerations, it has been proposed that complete removal of thyroid antigens and of thyroid infiltrating lymphocytes, the so-called total thyroid ablation (TTA), may be followed by an attenuation of the immune reaction against orbital antigens, and ultimately by an amelioration of GO. The possibility that TTA, achieved by near total thyroidectomy followed by radioiodine, may be beneficial for GO was initially suggested by two retrospective studies and more recently by two prospective, randomized clinical trials conducted in patients with moderate GO treated with intravenous glucocorticoids. Although there seemed to be no difference in the long term, compared with near total thyroidectomy alone TTA was associated with a shorter time required for GO to improve, or anyway to reach its best possible outcome, and with a lesser requirement for additional treatments for GO to improve. Whether this is sufficient to offer ablation to patients remains a matter of discussion. At present, this procedure could be offered only to patients scheduled to thyroidectomy and glucocorticoid treatment.
Medical Treatment of Graves' Orbitopathy.
Salvi M1, Campi I1.

Abstract

The medical treatment of Graves' orbitopathy (GO) is usually reserved to moderate to severe disease. Steroids have been widely employed and possess anti-inflammatory activity, but about 20-30% of patients are not responsive and about 20% present with disease recurrence. Immunosuppressive therapy alternative to corticosteroids may target the different antigens involved in pathogenic mechanisms of GO. Some have already been employed in clinical studies and showed interesting results, although the lack of randomized and controlled trials suggests caution for their use in clinical practice. Potential targets for therapy in GO are the TSH receptor and the IGF-1 receptor on the fibroblasts, inflammatory cytokines, B and T cells. Most promising results are obtained by interacting with the PIK3/mTORC1 signaling cascades for adipogenesis and the anti-IGF-1R with the monoclonal antibody teprotumumab. A recent open study has shown that tocilizumab, an anti-sIL-6R antibody, inactivates GO. Consistent reports on the efficacy of rituximab have recently been challenged by randomized controlled trials. Clinical practice will greatly benefit from the use of disease modifying agents in GO, as compared to steroids, currently standard treatment for GO. Among these, rituximab may be useful, especially in patients resistant to steroid or with contraindications to steroids. However, larger randomized controlled trials are needed for definitive data on the potential disease-modifying role of rituximab in GO. Direct targeting of the orbital fibroblast via immunosuppression or nonimmunosuppressive drugs is emerging as a promising alternative.

4. CERRAHİ TEDAVİ STRATEJİLERİ


Surgery of the thyroid: recent developments and perspective.

Fortuny JV1, Guigard S, Karenovics W, Triponez F2.

Abstract

In the past century, thyroid surgery has benefited from physiological and technical revolutions. In the early 1900s, the most important aspect of thyroidectomy was the volume resected, without knowledge of exactly what was removed and if there were important structures around the thyroid gland. The main indications were respiratory problems for tracheal compression and the death rate was greater than 36% due to
bleeding, infections, unrecognised bilateral recurrent laryngeal nerve lesions and unrecognised severe hypocalcaemia leading to tetany. At some point this surgery was, therefore, banned in some countries such as France and the United States. Today, thyroid surgery is a common surgery: about 45,000 thyroidecomies are performed per year in France, 60,000 in Germany and 4,000 in Switzerland. Thyroid surgery has become very safe with a mortality of almost 0% and a very low complication rate. In our centre, the number of thyroidecomies has more than tripled in the last decade. There are many indications leading to thyroid surgery, but the three main indications covering 90% of the interventions are cancer (or suspected cancer), hyperthyroidism and size / volume / intrathoracic goitres. In this paper, we highlight some historical points, describe important knowledge and technical improvements made during the last century and give our opinion on expected evolution in this field for the near future.


**Surgical management of hyperthyroidism.**
Quérat C¹, Germain N², Dumollard JM³, Estour B², Peoc'h M³, Prades JM⁴.

**Abstract**

**AIMS:**

Hyperthyroidism includes several clinical and histopathological situations. Surgery is commonly indicated after failure of medical treatment. The aim of this study was to analyze the indications and complications of surgery as well as endocrine results.

**MATERIALS AND METHODS:**

Patients operated on for hyperthyroidism between 2004 and 2012 were included in a retrospective study. Total thyroidecomy was performed for Graves' disease, toxic multinodular goiter and amiodarone-associated thyrotoxicosis; patients with toxic nodule underwent hemithyroidectomy. Pathologic analysis assessed surgical specimens; postoperative complications and resolution of hyperthyroidism were noted.

**RESULTS:**

Two hundred patients from 15 to 83 years old were included. One hundred and eighty-eight underwent primary surgery and 12 were re-operated for recurrent goiter (6 with subtotal thyroidecomy for multinodular goiter 25 years previously; 6 with hemithyroidectomy for solitary nodule 15 years previously). Eighty-two patients suffered from toxic multinodular goiter, 78 from Graves' disease, 35 from solitary toxic nodules and 5 from amiodarone-associated thyrotoxicosis. Fourteen papillary carcinomas (including 11 papillary microcarcinomas) and 34 healthy parathyroid glands (17%) were
identified in the pathological specimens. Postoperative complications comprised 4% permanent recurrent laryngeal nerve palsy (1 year follow-up), 9% hematoma requiring surgical revision, and 3% definitive hypocalcemia. Normalization of thyroid hormone levels was observed in 198 patients. Two recurrences occurred due to incomplete resection (1 case of Graves' disease and 1 intrathoracic toxic goiter that occurred respectively 18 and 5 months after resection). Postoperative complications were more frequent in multinodular goiter (23%) than in Graves' disease (13%) (ns: P>0.05).

CONCLUSION:

Surgical management of hyperthyroidism enables good endocrinal control if surgery is complete. Patients need to be fully informed of all possible postoperative complications that could occur, especially vocal ones. Long-term follow-up is necessary to detect recurrence, which can occur more than 20 years after partial thyroidectomy surgery. Surgery allows early diagnosis of 12.5% of papillary carcinomas.

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5. TAKİP ORGANİZASYONU VE REPLASMAN TEDAVİSİNİN MONİTORİZASYONU


2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis.

Ross DS¹, Burch HB², Cooper DS³, Greenlee MC⁴, Laurberg P⁵, Maia AL⁶, Rivkees SA⁷, Samuels M⁸, Sosa JA⁹, Stan MN¹⁰, Walter MA¹¹.

Abstract

BACKGROUND:

Thyrotoxicosis has multiple etiologies, manifestations, and potential therapies. Appropriate treatment requires an accurate diagnosis and is influenced by coexisting medical conditions and patient preference. This document describes evidence-based clinical guidelines for the management of thyrotoxicosis that would be useful to generalist and subspecialty physicians and others providing care for patients with this condition.

METHODS:

The American Thyroid Association (ATA) previously cosponsored guidelines for the management of thyrotoxicosis that were published in 2011. Considerable new literature has been published since then, and the ATA felt updated evidence-based guidelines were needed. The association assembled a task force of expert clinicians who authored
this report. They examined relevant literature using a systematic PubMed search supplemented with additional published materials. An evidence-based medicine approach that incorporated the knowledge and experience of the panel was used to update the 2011 text and recommendations. The strength of the recommendations and the quality of evidence supporting them were rated according to the approach recommended by the Grading of Recommendations, Assessment, Development, and Evaluation Group.

RESULTS:

Clinical topics addressed include the initial evaluation and management of thyrotoxicosis; management of Graves’ hyperthyroidism using radioactive iodine, antithyroid drugs, or surgery; management of toxic multinodular goiter or toxic adenoma using radioactive iodine or surgery; Graves' disease in children, adolescents, or pregnant patients; subclinical hyperthyroidism; hyperthyroidism in patients with Graves' orbitopathy; and management of other miscellaneous causes of thyrotoxicosis. New paradigms since publication of the 2011 guidelines are presented for the evaluation of the etiology of thyrotoxicosis, the management of Graves' hyperthyroidism with antithyroid drugs, the management of pregnant hyperthyroid patients, and the preparation of patients for thyroid surgery. The sections on less common causes of thyrotoxicosis have been expanded.

CONCLUSIONS:

One hundred twenty-four evidence-based recommendations were developed to aid in the care of patients with thyrotoxicosis and to share what the task force believes is current, rational, and optimal medical practice.

6. GEBELİKTE TİROTOKSİKOZ


Managing hyperthyroidism in pregnancy: current perspectives.

Andersen SL¹, Laurberg P².

Abstract

Hyperthyroidism in women who are of childbearing age is predominantly of autoimmune origin and caused by Graves' disease. The physiological changes in the maternal immune
system during a pregnancy may influence the development of this and other autoimmune diseases. Furthermore, pregnancy-associated physiological changes influence the synthesis and metabolism of thyroid hormones and challenge the interpretation of thyroid function tests in pregnancy. Thyroid hormones are crucial regulators of early development and play an important role in the maintenance of a normal pregnancy and in the development of the fetus, particularly the fetal brain. Untreated or inadequately treated hyperthyroidism is associated with pregnancy complications and may even program the fetus to long-term development of disease. Thus, hyperthyroidism in pregnant women should be carefully managed and controlled, and proper management involves different medical specialties. The treatment of choice in pregnancy is antithyroid drugs (ATDs). These drugs are effective in the control of maternal hyperthyroidism, but they all cross the placenta, and so need careful management and control during the second half of pregnancy considering the risk of fetal hyper- or hypothyroidism. An important aspect in the early pregnancy is that the predominant side effect to the use of ATDs in weeks 6-10 of pregnancy is birth defects that may develop after exposure to available types of ATDs and may be severe. This review focuses on four current perspectives in the management of overt hyperthyroidism in pregnancy, including the etiology and incidence of the disease, how the diagnosis is made, the consequences of untreated or inadequately treated disease, and finally how to treat overt hyperthyroidism in pregnancy.

7. TİROİD FIRTINASI YÖNETİMİ


2016 Guidelines for the management of thyroid storm from The Japan Thyroid Association and Japan Endocrine Society (First edition).

Abstract

Thyroid storm is an endocrine emergency which is characterized by multiple organ failure due to severe thyrotoxicosis, often associated with triggering illnesses. Early suspicion, prompt diagnosis and intensive treatment will improve survival in thyroid storm patients. Because of its rarity and high mortality, prospective intervention studies for the treatment of thyroid storm are difficult to carry out. We, the Japan Thyroid Association and Japan Endocrine Society taskforce committee, previously developed new diagnostic criteria and conducted nationwide surveys for thyroid storm in Japan. Detailed analyses of clinical data from 356 patients revealed that the mortality in Japan was still high (~11%) and that multiple organ failure and acute heart failure were common causes of death. In addition, multimodal treatment with antithyroid drugs, inorganic iodide, corticosteroids and beta-adrenergic antagonists has been suggested to improve mortality of these patients. Based on the evidence obtained by nationwide surveys and additional literature searches, we herein established clinical guidelines for the management of thyroid storm. The present guideline includes 15 recommendations for the treatment of thyrotoxicosis and organ failure in the central nervous system, cardiovascular system, and hepato-gastrointestinal tract, admission criteria for the intensive care unit, and prognostic evaluation. We also proposed preventive
approaches to thyroid storm, roles of definitive therapy, and future prospective trial plans for the treatment of thyroid storm. We hope that this guideline will be useful for many physicians all over the world as well as in Japan in the management of thyroid storm and the improvement of its outcome.


**Treatment and management of thyroid storm: analysis of the nationwide surveys: The taskforce committee of the Japan Thyroid Association and Japan Endocrine Society for the establishment of diagnostic criteria and nationwide surveys for thyroid storm.**

Isozaki O1, Satoh T2, Wakino S1, Suzuki A4, Iburi T5, Tsuboi K6, Kanamoto N7,8, Otani H9, Furukawa Y10, Teramukai S11, Akamizu T10.

**Abstract**

**OBJECTIVE:** Thyroid storm (TS) is a life-threatening endocrine emergency. This study aimed to achieve a better understanding of the management of TS by analyzing therapeutic modalities and prognoses reported by nationwide surveys performed in Japan.

**DESIGN, PATIENTS AND MEASUREMENTS:**

Retrospective analyses were performed on clinical parameters, outcomes, and treatments in 356 TS patients.

**RESULTS:**

Patient disease severities assessed via Acute Physiology and Chronic Health Evaluation (APACHE) II and Sequential Organ Failure Assessment (SOFA) scores significantly correlated with mortality. Free triiodothyronine (FT3) and the FT3/free thyroxine (FT4) ratio inversely correlated with disease severity. Methimazole (MMI) was used in the majority of patients (78.1%), and there were no significant differences in mortality or disease severity between those treated with MMI and those receiving propylthiouracil (PTU). Patients who received inorganic iodide (KI) demonstrated higher disease severity but no change in mortality compared to those who did not. Patients treated with corticosteroids (CSs) demonstrated significantly higher disease severity and mortality than those who were not. Disease severity in patients treated with intravenous administration of beta-adrenergic antagonists (AAs) was significantly higher than those treated with oral preparations, although no significant difference in mortality was observed between these groups. In addition, mortality was significantly higher in patients treated with non-selective beta-AAs as compared with other types of beta-AAs.
CONCLUSION:

In Japan, MMI was preferentially used in TS and showed no disadvantages compared to PTU. In severe TS, multimodal treatment, including administration of antithyroid drugs, KI, CSs and selective beta1 -AAs may be preferable to improve outcomes.

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Thyroid storm: an updated review.

Chiha M1, Samarasinghe S1, Kabaker AS2.

Abstract

Thyroid storm, an endocrine emergency first described in 1926, remains a diagnostic and therapeutic challenge. No laboratory abnormalities are specific to thyroid storm, and the available scoring system is based on the clinical criteria. The exact mechanisms underlying the development of thyroid storm from uncomplicated hyperthyroidism are not well understood. A heightened response to thyroid hormone is often incriminated along with increased or abrupt availability of free hormones. Patients exhibit exaggerated signs and symptoms of hyperthyroidism and varying degrees of organ decompensation. Treatment should be initiated promptly targeting all steps of thyroid hormone formation, release, and action. Patients who fail medical therapy should be treated with therapeutic plasma exchange or thyroidectomy. The mortality of thyroid storm is currently reported at 10%. Patients who have survived thyroid storm should receive definite therapy for their underlying hyperthyroidism to avoid any recurrence of this potentially fatal condition.

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PMID: 23920160 [Indexed for MEDLINE]
Rituximab (RTX) use in open-label series has been associated with very encouraging responses in patients with active and moderate-to-severe Graves' orbitopathy (GO). Recently, randomized controlled trials of RTX have been performed in such patients to answer the question of clinical efficacy and the safety profile of this agent. That data, reported separately, focused on Clinical Activity Score (CAS) and indicated in one trial a strong benefit of RTX in comparison with IV glucocorticoids, whereas the other trial noted the absence of a benefit by comparison with placebo. The outcome was reanalyzed post hoc here, using EUGOGO criteria, and the results were not significantly different. The authors comment further on the differences between the two trials regarding populations treated, methodology, analysis of outcomes and the adverse effect profile of RTX. The populations treated appear different with younger patients, lower TRAb and shorter duration of disease prevalent in the Italian trial, all elements favoring a better response. Smoking, usually diminishing a...' convey the intended meaning., usually diminishing a response, was also more prevalent in some patients. The combined outcome proposed by EUGOGO revealed similar results with CAS regarding RTX efficacy; yet, it might be a more comprehensive outcome. The adverse events of concern relate mainly to the risk of DON, which seems to be increased by the use of RTX in a certain subset of patients. Based on available data, a multicenter trial using the EUGOGO-proposed outcomes might be the next best step to define the role of RTX in GO therapy.