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The MERCK Manual

Of Diagnosis and Therapy

NINETEENTH EDITION

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Table 93–2. TREATMENT OF THYROID STORM

Propylthiouracil: 600 mg po given before iodine, then 400 mg q 6 h
Iodine: 5 drops saturated solution of K iodide po tid *or* 10 drops Lugol's solution po tid *or* 1 g Na iodide slowly by IV drip over 24 h
Propranolol: 40 mg po qid *or* 1 mg slowly IV q 4 h (not to exceed 1 mg/min) under close monitoring A repeat 1-mg dose given after 2 min, if needed, or esmolol
IV dextrose solutions
Correction of dehydration and electrolyte imbalance
Cooling blanket for hyperthermia
Antiarrhythmic drugs (eg, Ca channel blockers, adenosine, β-blockers) if necessary for atrial fibrillation
Treatment of underlying disorder, such as infection
Corticosteroids: Hydrocortisone 100 mg IV q 8 h *or* Dexamethasone 8 mg IV once/day
Definitive therapy after control of the crisis via ablation of the thyroid with ^{131}I or surgical treatment

Graves' disease and toxic nodular goiter in all patients, including children. Dosage of ^{131}I is difficult to adjust because the response of the gland cannot be predicted; some physicians give a standard dose of 8 to 10 mCi. Others adjust the dose based on estimated thyroid size and the 24-h uptake to provide a dose of 80 to 120 μCi/g thyroid tissue.

When sufficient ^{131}I is given to cause euthyroidism, about 25% of patients become hypothyroid 1 yr later, and the incidence continues to increase yearly. Thus, most patients eventually become hypothyroid. However, if smaller doses are used, incidence of recurrence is higher. Larger doses, such as 10 to 15 mCi, often cause hypothyroidism within 6 mo.

Radioactive iodine is not used during pregnancy. There is no proof that radioiodine increases the incidence of tumors, leukemia, thyroid cancer, or birth defects in children born to women who become pregnant later in life.

Surgery: Surgery is indicated for patients with Graves' disease whose hyperthyroidism has recurred after courses of antithyroid drugs and who refuse ^{131}I therapy, patients who cannot tolerate antithyroid drugs, patients with very large goiters, and in some younger patients with toxic adenoma and multinodular goiter. Surgery may be done in elderly patients with giant nodular goiters.

Surgery usually restores normal function. Postoperative recurrences vary between 2 and 16%; risk of hypothyroidism is directly related to the extent of surgery and occurs in about one half of patients. Vocal cord paralysis and hypoparathyroidism are uncommon complications. Saturated solution of K iodide 3 drops (about 100 to 150 mg) po tid should be given for 10 days before surgery to reduce the vascularity of the gland. Propylthiouracil or methimazole must also be given, because the patient should be euthyroid before iodide is given. Dexamethasone can be added to rapidly restore euthyroidism. Surgical procedures are more difficult in patients who previously underwent thyroidectomy or radioiodine therapy.

Treatment of thyroid storm: A treatment regimen for thyroid storm is shown in Table 93–2.

Treatment of infiltrative dermopathy and ophthalmopathy: In infiltrative dermopathy (in Graves' disease), topical corticosteroids sometimes relieve the pruritus. Dermopathy usually remits spontaneously after months or years. Ophthalmopathy should be treated jointly by the endocrinologist and ophthalmologist and may require corticosteroids, orbital radiation, and surgery.

Subclinical Hyperthyroidism

Subclinical hyperthyroidism is low serum TSH in patients with normal serum free T_4 and T_3 and absent or minimal symptoms of hyperthyroidism.

Subclinical hyperthyroidism is far less common than subclinical hypothyroidism (see p. 787). Patients with serum TSH < 0.1 mU/L have an increased incidence of atrial fibrillation (particularly elderly patients), reduced bone mineral density, increased fractures, and increased mortality. Patients with serum TSH that is only slightly below normal are less likely to have these features. Many patients with subclinical hyperthyroidism are taking L-thyroxine; in these patients, reduction of the dose is the most appropriate management unless therapy is aimed at maintaining a suppressed TSH in patients with thyroid cancer or nodules. The other causes of subclinical hyperthyroidism are the same as those for clinically apparent hyperthyroidism.

Therapy is indicated for patients with endogenous subclinical hyperthyroidism (serum TSH < 0.1 mU/L), especially those with atrial fibrillation or reduced bone mineral density. The usual treatment is ^{131}I. In patients with milder symptoms (eg, nervousness), a trial of antithyroid drug therapy is worthwhile.

HYPOTHYROIDISM

(Myxedema)

Hypothyroidism is thyroid hormone deficiency. It is diagnosed by clinical features such as a typical facies, hoarse slow speech, and dry skin and by low levels of thyroid hormones. Management includes treatment of the cause and administration of thyroxine.

Hypothyroidism occurs at any age but is particularly common among the elderly. It occurs in close to 10% of women and 6% of men > 65. Although typically easy to diagnose in younger adults, it may be subtle and manifest atypically in the elderly.

Primary hypothyroidism: Primary hypothyroidism is due to disease in the thyroid; thyroid-stimulating hormone (TSH) is increased. The most common cause is probably autoimmune. It usually results from Hashimoto's thyroiditis and is often associated with a firm goiter or, later in the disease process, with a shrunken fibrotic thyroid with little or no function. The 2nd most common cause is post-therapeutic hypothyroidism, especially after radioactive iodine therapy or surgery for hyperthyroidism or goiter. Hypothyroidism during overtreatment with propylthiouracil, methimazole, and iodide abates after therapy is stopped.

Most patients with non-Hashimoto's goiters are euthyroid or have hyperthyroidism, but goitrous hypothyroidism may occur in endemic goiter. Iodine deficiency decreases thyroid hormonogenesis. In response, TSH is released, which causes the thyroid to enlarge and trap iodine avidly; thus, goiter results. If iodine deficiency is severe, the patient becomes hypothyroid, a rare occurrence in the US since the advent of iodized salt.

Iodine deficiency can cause endemic cretinism in children; endemic cretinism is the most common cause of congenital hypothyroidism in severely iodine-deficient regions and a major cause of mental deficiency worldwide.

Rare inherited enzymatic defects can alter the synthesis of thyroid hormone and cause goitrous hypothyroidism (see p. 2887).

Hypothyroidism may occur in patients taking lithium, perhaps because lithium inhibits hormone release by the thyroid. Hypothyroidism may also occur in patients taking amiodarone or other iodine-containing drugs, and in patients taking interferon alfa. Hypothyroidism can result from radiation therapy for cancer of the larynx or Hodgkin lymphoma (Hodgkin's disease). The incidence of permanent hypothyroidism after radiation therapy is high, and thyroid function (through measurement of serum TSH) should be evaluated at 6- to 12-mo intervals.

Secondary hypothyroidism: Secondary hypothyroidism occurs when the hypothalamus produces insufficient thyrotropin-releasing hormone (TRH) or the pituitary produces insufficient TSH. Sometimes, deficient TSH secretion due to deficient TRH secretion is termed tertiary hypothyroidism.

Symptoms and Signs

Symptoms and signs of primary hypothyroidism are often subtle and insidious. Symptoms may include cold intolerance, constipation, forgetfulness, and personality changes. Modest weight gain is largely the result of fluid retention and decreased metabolism. Paresthesias of the hands and feet are common, often due to carpal-tarsal tunnel syndrome caused by deposition of proteinaceous ground substance in the ligaments around the wrist and ankle. Women with hypothyroidism may develop menorrhagia or secondary amenorrhea.

The facial expression is dull; the voice is hoarse and speech is slow; facial puffiness and periorbital swelling occur due to infiltration with the mucopolysaccharides hyaluronic acid and chondroitin sulfate; eyelids droop because of decreased adrenergic drive; hair is sparse, coarse, and dry; and the skin is coarse, dry, scaly, and thick. The relaxation phase of deep tendon reflexes is slowed. Hypothermia is common. Dementia or frank psychosis (myxedema madness) may occur.

Carotenemia is common, particularly notable on the palms and soles, caused by deposition of carotene in the lipid-rich epidermal layers. Deposition of proteinaceous ground substance in the tongue may cause macroglossia. A decrease in both thyroid hormone and adrenergic stimulation causes bradycardia. The heart may be enlarged, partly because of dilation but chiefly because of pericardial effusion. Pleural or abdominal effusions also may be noted. The pericardial and pleural effusions