Thyrotoxicosis:
Pathophysiology, assessment and management

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Thyrotoxicosis, which results from the biochemical and physiologic effects of excess thyroid hormone regardless of cause, is one of the more common endocrine disorders presenting to the family physician. By definition, hyperthyroidism is a term restricted to situations in which the thyroid gland is responsible for overproducing thyroid hormone. Arbitrarily the causes of thyrotoxicosis can be differentiated into those associated with a high uptake on radioactive iodine or technetium scanning (most commonly Graves disease) and those with a low uptake (Table 1).

The normal adult thyroid is one of the largest endocrine organs weighing about 15–20 grams. It consists of two lobes joined by an isthmus, with arterial blood supply from both the superior and the inferior thyroid arteries. The normally high blood flow of 4–6mL/minute/g (compared to the renal blood flow of 3mL/min/g) may increase to over 1L/minute in the diffuse toxic goitre of Graves disease, resulting in an audible bruit or palpable thrill. At a microscopic level the gland is composed of follicles filled with thyroglobulin-containing colloid in which thyroid hormone is stored. In addition the gland contains parafollicular (C cells) cells, which secrete calcitonin. The thyroid gland synthesises and secretes thyroid hormones sufficient to meet bodily needs. This involves active uptake of iodide by the thyroid transmembrane sodium-iodide symporter, subsequent oxidation of the iodide by the enzyme thyroid peroxidase (TPO) and incorporation into the thyroglobulin molecule in the form of monoiodotyrosine (MIT) and diiodotyrosine (DIT). These precursors are joined or coupled to form thyroxine (T_4) and triiodothyronine (T_3) in the thyroglobulin molecule. The thyroid stores large amounts of thyroid hormones in the colloid, of which only about 1% are secreted daily. This excess storage protects against hypothyroidism should thyroid hormone synthesis be temporarily inhibited, for example by a dietary goitrogen. This excess storage of thyroglobulin helps explain why anti-thyroid medications may not reduce the T_4 levels for at least two weeks. In addition, the thyrotoxicosis occurring after an inflammatory thyroiditis in which the thyroid cells are disrupted results from the release of this preformed thyroid hormone storage. All of the steps in thyroid hormone synthesis and release are stimulated by Thyroid Stimulating Hormone (TSH), secreted from the anterior pituitary. The thyroid gland releases both T_4 and T_3, however in the euthyroid state about 80% of the T_3 is produced peripherally from T_4. Being relatively lipid-soluble both thyroid hormones circulate bound to plasma proteins – Thyroxine-Binding Globulin (TBG) and transthyretin and albumin and to a lesser extent as the free hormone. Thyroid hormone readily crosses the plasma membrane and in the form of T_3 binds to spe-

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cific intra-cellular thyroid hormone receptors, which as a complex act as transcription factors to up- or down-regulate many specific genes. The circulatory levels of T_3 are controlled by negative feedback loops involving the thyroid, pituitary and hypothalamus. Stability of thyroid hormone levels are assisted by the large intra-thyroidal hormone pool.

Although thyrotoxicosis is not always obvious clinically, (commonly presenting as tiredness), the biochemical profile of an elevated Free T_3 (FT_3), and/or Free T_4 (FT_4), with a suppressed TSH make the diagnosis relatively straightforward. In T_3-toxicosis the FT_3 is elevated and TSH is suppressed with a normal FT_4.

Occasionally a family physician receives laboratory results in which the T_3 and/or T_4 levels are above the upper limit of the reference range and the TSH is not suppressed. The most common cause of elevated free thyroid hormone levels with a measurable TSH is interference with the assay by heterophile antibodies in a clinically euthyroid patient. Specific tests for heterophile antibodies can be requested from the laboratory. Thyroid hormone resistance is another cause of elevated thyroid hormone levels with a normal or elevated TSH. Recent intake of excess thyroxine in a previously noncompliant patient may also cause the same pattern, as free thyroid hormone levels will elevate before the TSH falls.

Secondary thyrotoxicosis (elevated free thyroid hormone levels, with a measurable levels of TSH in a clinically thyrotoxic patient) due to a TSH-secreting pituitary adenoma is rare, (estimated incidence approximately one per million per annum). Although extremely rare, TSHomas are important not to miss as treatment is primarily directed towards the pituitary.

Given the complexity, patients with elevated free thyroid hormone levels and a nonsuppressed TSH should be referred to an endocrinologist for more detailed investigation to ensure correct diagnosis and treatment.

Table 1. Causes of thyrotoxicosis

Thyrotoxicosis with high radioiodine uptake	Thyrotoxicosis with low radioiodine uptake
Autoimmune – Graves disease Autonomous thyroid tissue – Toxic multinodular goitre – Solitary toxic adenoma TSH-mediated – TSHoma (rare) HCG-mediated – Hyperemesis gravidarum – Hydatidiform mole/ choriocarcinoma (rare) – Testicular tumours (rare)	Thyroiditis – Subacute – Postpartum – Drug-induced e.g. interferon, amiodarone – Radiation Autonomous thyroid tissue with iodine load e.g. amiodarone/x-ray contrast Excessive exogenous thyroid hormone intake Ectopic thyrotoxicosis – Struma ovarii (ectopic thyroid tissue) (rare) – Metastatic follicular cancer with with functioning metastases (rare)

Clinical features of thyrotoxicosis

Presenting symptoms resulting from excess metabolic activity include tiredness, heat intolerance, unexplained weight loss, excess sweating, palpitations, tremor and irritability (Table 2). Older patients with 'apathetic thyrotoxicosis' may present predominantly with weight loss, anorexia, muscle weakness, depression and lethargy. Occasional patients may present with sudden onset profound muscle weakness (which may progress to a flaccid tetraparesis), associated with severe hypokalaemia, which resolves completely on restoration of the serum potassium (thyrotoxic hypokalaemic periodic paralysis – THPP). THPP, which may be precipitated by exercise or a large carbohydrate meal, is more common in males, especially Asian, Maori, and Polynesian, and results from increased intracellular uptake of potassium.

The severity of thyrotoxicosis can vary from the patient who has very few symptoms to those who present with florid congestive cardiac heart failure, life-threatening arrhythmias, and even severe psychotic illness. Thyroid storm, although extremely rare, is usually of sudden onset and may be precipitated by infection, surgery or major illness. Fever is almost always present, with profound tachycardia and possibly arrhythmias. Restlessness and delirium or even frank psychosis may occur and progress to coma. Nausea, vomiting

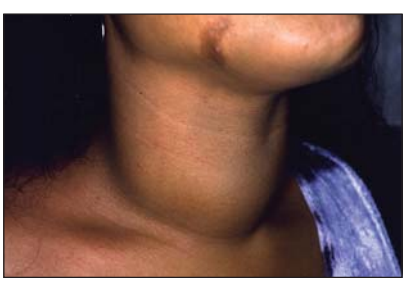


Figure 1. Diffuse goitre

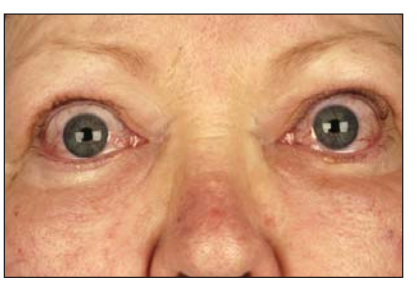
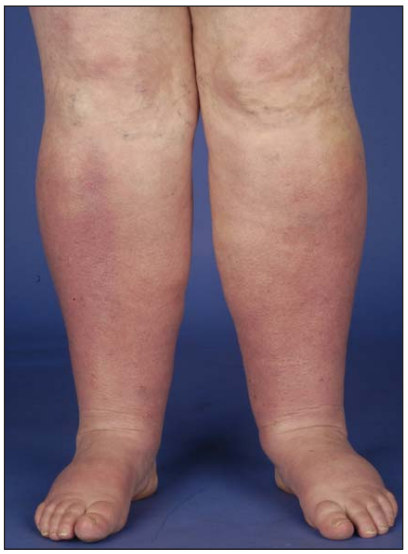


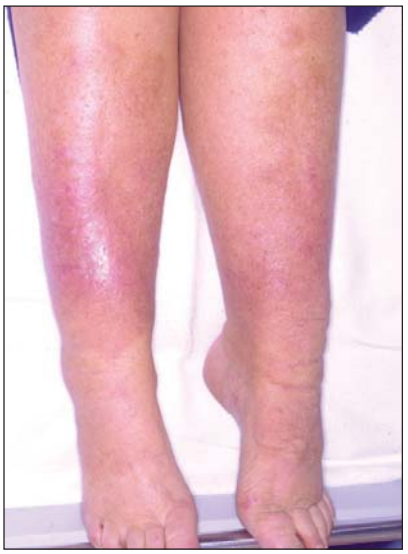
Figure 2. Graves ophthalmopathy

and abdominal pain frequently occurs early. Although this condition is often exaggerated by the inexperienced clinician, unrecognised thyroid storm is usually fatal.

Graves disease

Graves disease is an autoimmune condition with the thyrotoxicosis caused by autoantibodies stimulating the TSH receptor (TSHRAb or Thyroid Stimulating Immunoglobulins, TSI). As in other autoimmune conditions, Graves disease is more common in females (7–10:1) and a family history of thyroid or other autoimmune disease is often present. The classical manifestations of Graves disease include a diffuse toxic goitre (Figure 1), congestive ophthalmopathy (Figure 2) and dermopathy (pretibial-myxoedema) (Figures 3 and 4). These three features of Graves disease may occur simultaneously or independently in a given individual. In practice, patients commonly present with thyrotoxicosis and a diffuse goitre usually with a soft bruit. Although congestive ophthalmopathy is often present, it is usually mild and may not be recognised.





Figures 3 and 4. Graves dermopathy

Dermopathy (pretibial myxoedema) is relatively uncommon. It is important to distinguish the congestive ophthalmopathy of Graves disease from the eye signs secondary to sympathetic overactivity, which may occur in thyrotoxicosis from any cause. Congestive ophthalmopathy is due to swelling of the extraocular muscles and orbital fat by accumulation of excess water and glycosaminoglycans secreted from fibroblasts. This results in bulgy eyes (exophthalmos/proptosis) and conjunctival injection, chemosis, periorbital oedema, restriction in eye movements and, in severe cases, visual loss. Thyrotoxicosis from any cause may cause sympathetic eye signs such as lid lag and lid retraction and the stare ('startled rabbit' look), which are not specific to Graves disease.

Table 2. Symptoms and signs of thyrotoxicosis

Symptoms	Signs
Fatigue	Fine tremor
Heat intolerance	Tachycardia
Weight loss (rarely weight gain)	Sweating
Hyperphagia (rarely anorexia)	Goitre
Palpitations	Lid lag, retraction
Sweating	Heart failure
Tremor	Atrial fibrillation
Reduced exercise tolerance	Ophthalmopathy
Increased frequency of defaecation	Dermopathy
Loose bowel motions	Proximal myopathy
Anxiety	Gynaecomastia
Irritability	Systolic hypertension
Poor sleep	Hyperdynamic circulation
Irregular periods	Hyperreflexia
Lighter periods	Palmar erythema
Difficulty concentrating	
Urinary frequency	**Other**
Weakness	Hypercalcaemia, raised alkaline phosphatase, deterioration in glycaemic control
Shortness of breath	

Toxic multinodular goitre

Toxic multinodular goitre (MNG) is due to the development of autonomy in a (usually) long-standing pre-existing goitre. The thyroid feels nodular and there is no bruit. The thyrotoxicosis may be precipitated by recent iodine exposure, such as iodine containing contrast from radiological procedures, iodine from medications (e.g. amiodarone) or from health shop preparations. Patients with toxic multinodular goitre are often older and more severely affected by the hyperthyroidism. Radioactive iodine is an effective treatment for toxic MNG provided there hasn't been recent iodine exposure.

Solitary toxic nodule

Solitary toxic nodule refers to the development of a (usually) solitary nodule in a thyroid that is otherwise normal. Most are due to a somatic

mutation in the *TSH Receptor* gene resulting in constitutive activation of the receptor without TSH binding. With increased growth and activity of the nodule the remainder of the gland becomes suppressed and eventually atrophic. It is more common in young-middle-aged patients with mild thyrotoxicosis and presents with a single smooth palpable nodule (with no bruit). T_3-toxicosis is not uncommon in this setting. Treatment is either radioactive iodine (with a low risk of subsequent hypothyroidism, as the rest of the suppressed gland usually recovers function) or surgical removal of the nodule.

Thyroiditis

Subacute viral thyroiditis is another relatively common cause of thyrotoxicosis. The patient presents acutely with hyperthyroidism, with or without neck pain, and general features of malaise, fatigue and myalgia. The thyroid gland may be impalpable or mildly enlarged, and may or may not be tender. The erythrocyte sedimentation rate and other inflammatory markers are usually raised, associated with a mild normochromic anaemia. If the clinician is uncertain of the diagnosis a Technetium 99m thyroid scan will reveal reduced uptake of the radioisotope (in contrast to Graves disease in which the uptake is increased). This condition is self-limiting with an initial thyrotoxic phase due to release of preformed thyroid hormone from disrupted thyroid cells, followed by a hypothyroid phase, which may persist for several months before the return to euthyroidism. Treatment is symptomatic with analgesia for any thyroid discomfort (paracetamol being first-line, followed by NSAIDs. Glucocorticoids such as prednisone are only rarely required). It should be noted that none of these treatments alter the natural course of the disease. Beta-blockers can reduce the symptoms of thyrotoxicosis such as palpitations and can be weaned as the thyrotoxicosis resolves. The hypothyroid phase typically doesn't need treatment but occasionally a short course of thyroxine is required if the patient is severely symptomatic. The whole process may take six to nine months. In about 5% of patients with subacute thyroiditis the hypothyroidism may be permanent.

Table 3. Comparison of radioactive iodine and surgery for thyrotoxicosis

Radioactive Iodine	Surgery
Advantages	**Advantages**
Noninvasive	Rapid control of thyrotoxicosis
Well-tolerated even in the elderly	Rapid relief from pressure symptoms
Lower risk of hypothyroidism especially if toxic nodular goitre	No risk of recurrence (if total thyroidectomy performed)
Can reduce size of goitre	
No need for anaesthetic	
Disadvantages	**Disadvantages**
Doesn't work immediately	Invasive
Risk of late hypothyroidism - may be insidious so ongoing monitoring required	Risk of complications including hypoparathyroidism, recurrent laryngeal nerve injury and other surgical complications Anaesthetic risk
Precautions required	May not be suitable for patients with significant underlying cardiac disease
Absolutely contraindicated in pregnancy	Scarring (keloid in some patients)
Risk of flare secondary to radiation thyroiditis (uncommon)	Need for lifelong thyroxine replacement therapy
May need a second or third dose (especially if Maori)	Increased anaesthetic and surgical risk if patient severely thyrotoxic preoperatively
Need for lifelong thyroxine replacement therapy	Recurrent disease following incomplete surgery

Investigations

The diagnosis usually just requires thyroid function tests – FT_4, FT_3, and TSH followed by a subsequent good clinical assessment. Measurement of thyroid antibodies may assist in determining the autoimmune nature of the condition. Often the cause of thyrotoxicosis is obvious from the history and examination findings (e.g. a diffuse goitre with bruit, with signs of congestive ophthalmopathy is clearly Graves disease and no further imaging is required). When the diagnosis is not obvious, e.g. the thyroid is not palpable, or when there is the suspicion of silent thyroiditis, radionuclide (Technetium 99m) imaging can be very helpful. Patients with Graves disease and a small thyroid gland will have increased uptake of the isotope and in those with thyroiditis, recent iodine or thyroxine exposure, the uptake of Tc99m will be reduced. In the majority of thyrotoxic patients, ultrasound is not useful as a first-line test (in contrast to euthyroid patients with a solitary nodule).

Treatment

Treatment needs to be directed according to the underlying cause. In patients with the common causes of primary hyperthyroidism (Graves, toxic MNG and solitary toxic nodule) there are generally three options for management. The first is the temporary use of oral medications including Carbimazole (CBZ), or propylthiouracil (PTU). More permanent

options include radioactive iodine I^{131}, or surgery (Table 3). All these options have their place in the management of primary hyperthyroidism and, as with all good medical practice, treatment should be tailored to the patient's circumstances.

1. **Anti-thyroid drugs** are commonly used as first-line treatment to achieve a euthyroid state and allow time for the more definitive options to be discussed and considered by the patient. CBZ can usually be safely taken once per day, whereas PTU needs to be taken 2–4x/d depending on dose. PTU is preferred in pregnant women as increasing reports suggest an association between methimazole (which is the metabolite of CBZ) and a rare embryopathy. In non-pregnant patients, CBZ is usually preferred as it is effective when taken once daily and, in New Zealand, PTU currently requires an exceptional circumstances form. Both drugs can result in agranulocytosis and the potential for death. Agranulocytosis is reported to occur in <1% of patients, usually in the first few weeks to months of treatment. Because, severe neutropaenia can develop suddenly, warning the patient of the risk is more important than routine monitoring of the neutrophil count. It is critical that any patient commenced on anti-thyroid medication should be fully educated about the risk of agranulocytosis. In addition, the patient can be provided with a laboratory form for a full blood count with the suggestion that should they develop any sign of infection such as sore throat and fever, they must stop the medication, have a blood test and phone their family doctor for the result the same day. After confirming the neutrophil count is normal the anti-thyroid medication can be recommenced. If the neutrophils are low, the test must be immediately confirmed, and urgent referral is required as the patient may require hospital admission for antibiotics and granulocyte colony-stimulating factor. Anti-thyroid medication should never be used in such patients again. One clinical difficulty is that some patients with thyrotoxicosis have a low neutrophil count prior to the use of anti-thyroid medications. In this circumstance consultation with colleagues in endocrinology and/or haematology and close monitoring is recommended. Other side-effects from anti-thyroid medications include liver dysfunction, arthralgia and rash. Approximately 40% of patients with Graves disease are reported to remain in remission after ceasing a 12–18 month period of medical therapy. This figure of 40% is clearly an overstatement as many patients on anti-thyroid drugs will be requiring such high doses of CBZ or PTU at 12 months that they certainly require definitive therapy, and many other patients just choose to have either radioactive iodine or surgery or they have reacted adversely to the drugs.
2. **Radioactive iodine I^{131}:** This is an effective treatment administered as an outpatient drink which carries a risk of permanent hypothyroidism, especially in Graves disease. Hypothyroidism is generally preferable to hyperthyroidism since thyroxine replacement is usually simpler for the patient and physician and requires less monitoring than suppression with CBZ, or PTU. I^{131} has been in use for more than 50 years, appears to be safe and is the recommended option for the majority of patients requiring definitive treatment, especially the elderly. It cannot be given to pregnant women, especially after the first trimester when the fetal thyroid can concentrate iodine. I^{131} can also worsen the congestive ophthalmopathy in a small number of patients although this is usually only mild and temporary. Precautions such as avoiding close contact for several days or two weeks off work for those working in the food industry are required following therapeutic I^{131} administration. One disadvantage of radioactive iodine is that it may take weeks to months to have an effect. In addition, the development of hypothyroidism may be insidious and occur years after therapy. For this reason it is important to continue monitoring thyroid function tests in euthyroid patient following I^{131} treatment, even if it was administered decades earlier. Rarely patients may develop a radiation thyroiditis, which results in a flare of the thyrotoxicosis 10–14 days post-treatment. Administration of radioactive iodine to children remains controversial, especially after the reports of thyroid cancer in young people after the Chernobyl disaster.
3. **Surgery:** Prior to offering surgery it is best to normalise the thyroid function tests with anti-thyroid drugs. Moderate to severe thyrotoxicosis increases the anaesthetic and surgical risk. Generally surgery is considered for patients with large goitres causing compressive symptoms, amiodarone-induced thyrotoxicosis refractory to treatment or in young patients with a solitary toxic nodule. However, some patients just prefer surgery to the concept of taking a drink of radioactive iodine. In addition, surgery has the advantage of rapid control of the thyrotoxicosis when compared to I^{131} and many patients just want to get on with their lives. With the exception of the solitary toxic nodule in an otherwise normal gland, near total thyroidectomy is the preferred operation because of the risk of recurrence of thyrotoxicosis in both Graves disease and toxic MNG. Surgery does

carry a mortality risk (as does every operation) as well as the risk of permanent hypoparathyroidism and recurrent laryngeal nerve injury. As the complication rate is lower if thyroidectomy is performed by an experienced thyroid surgeon, it is important that the referring physician be aware of their specific surgeon's skills.

Subclinical hyperthyroidism

Low or undetectable TSH levels with levels of FT_4 and FT_3 within the reference range are found in subclinical hyperthyroidism. This state may result from excessive exogenous thyroid hormone administration or due to endogenous overproduction of thyroid hormone, e.g. multinodular goitre with some autonomy. Excess exogenous thyroid hormone is common and may be intentional, as in the treatment of metastatic or localised thyroid cancer with high risk of recurrence, or unintentional, as in hypothyroid patients replaced with thyroxine. It is important not to confuse patients with hypopituitarism who will have a similar biochemical profile and low TSH levels as a result of pituitary gland hypofunction.

Overt thyrotoxicosis is a risk factor for osteoporosis and fractures although this is more controversial in subclinical disease.[1] Patients with subclinical hyperthyroidism receiving thyroxine replacement for benign thyroid disease should have the dose reduced to ensure the TSH is in the reference range. The treatment of subclinical hyperthyroidism remains controversial, although many studies report increased rates of atrial fibrillation and increased left ventricular mass in these patients.[1] A recent consensus panel supported the treatment of patients with undetectable TSH (<0.01mU/L) especially for patients older than 60 years, and those at increased risk of heart disease, osteoporosis, or those with symptoms suggestive of hyperthyroidism.[2] Patients with endogenous subclinical hyperthyroidism should be followed and referral to an endocrinology service considered if the condition persists, particularly in those with atrial fibrillation or in whom thyrotoxicosis would be poorly tolerated because of comorbid disease.

Other causes of a low TSH and normal free thyroid hormone levels include central hypothyroidism (usually the free thyroid hormone levels are at the lower end of the reference range), recovery from hyperthyroidism and as a transient effect in patients with nonthyroidal illness (sick euthyroid syndrome).

Pregnancy

Pregnancy is a special situation and pregnant women with thyrotoxicosis should be referred to an endocrinology service. Normal pregnancy with thyroid enlargement and hyperdynamic circulation can mimic some features of hyperthyroidism. In addition, TSH levels can be suppressed in pregnancy especially during the 8th–14th weeks because of HCG stimulation of the TSH receptor. Hyperemesis gravidarum may be associated with frankly elevated thyroid hormones, which then settle around 14–16 weeks as the HCG levels settle. Distinguishing this condition from Graves disease can be difficult with pre-existing thyroid disease, sleeping tachycardia, family history of autoimmune disease and positive TSH-receptor stimulating antibodies being more indicative of Graves disease. Thyrotoxicosis in pregnancy is associated with an increased rate of spontaneous miscarriage, stillbirth, preterm labour, low birth weight, and preeclampsia. Treatment with PTU, rather than CBZ, is increasingly recommended because of rare reports of methimazole embryopathy. While this has not been reported with carbimazole as carbimazole is metabolised to methimazole it is probably reasonable to opt for PTU. Both medications cross the placenta and can cause fetal hypothyroidism and goitre so the lowest possible dose should be given and the maternal free thyroid hormone levels should be maintained at the upper end of the reference range and measured monthly over the gestation. Obstetric involvement is indicated given the complexity and the small risk of fetal thyrotoxicosis. Women with only very mild thyrotoxicosis can be monitored carefully without treatment. Graves disease improves throughout pregnancy and often women can have the anti-thyroid medication stopped by the third tri-

Radioactive iodine is absolutely contraindicated during pregnancy, but surgery occasionally is required in special situations with the safest time being during the second trimester

Key Points

- Consider the underlying cause of the thyrotoxicosis – making a specific diagnosis will allow the practitioner to proceed directly to appropriate therapy. Uncertainty about the diagnosis will require early referral to endocrinology or further investigation such as technetium scanning.
- Early referral should be made for patients with severe thyrotoxicosis, those on amiodarone and those with reduced physiological reserve, e.g. the elderly and patients with cardiac disease.
- Anti-thyroid-induced agranulocytosis is rare, but can be life-threatening. Any patient started on anti-thyroid medication needs to be warned about this risk and instructed what to do in the event of illness.

mester. Radioactive iodine is absolutely contraindicated during pregnancy, but surgery occasionally is required in special situations with the safest time being during the second trimester. Post-partum flare of Graves disease in the mother is common and should be anticipated based on the pre- or early-pregnancy thyroid status of the mother. Women with a history of Graves disease who are now euthyroid (e.g. post thyroid ablation with radioactive iodine or surgery) may still have TSH-R antibodies which can cross the placenta and cause hyper- or hypo-thyroidism in the fetus so TSH-R Abs should be measured at the end of the second trimester and obstetric involvement is required to monitor for evidence of fetal thyrotoxicosis, although this is rare. Overzealous treatment with antithyroid medications in the third trimester can result in a fetal goitre, which may lead to difficulties with delivery as the fetal neck may be unable to fully flex or with airway compromise.

Elderly

Thyrotoxicosis in the elderly may present more subtly so a high index of suspicion is required. The older patient often does not tolerate thyrotoxicosis well and has an increased risk of atrial fibrillation, exacerbation of preexisting ischaemic heart disease and congestive cardiac failure. Any elderly patient with newly diagnosed atrial fibrillation should have their thyroid function measured, as atrial fibrillation may be the only manifestation of thyrotoxicosis.

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Amiodarone-induced thyrotoxicosis

Amiodarone is an iodine-rich medication (approximately 37% iodine by weight) and, while most patients on amiodarone remain euthyroid, it may cause either hypo- or hyperthyroidism. Patients on amiodarone should have regular monitoring of their thyroid function every three months. Amiodarone-induced thyrotoxicosis (AIT) may even occur months after the amiodarone is stopped and can be very resistant to treatment. Urgent referral to an endocrinology service should occur as the typical patient has underlying cardiac disease so the thyrotoxicosis may be poorly tolerated. As the amiodarone takes months to clear (half-life is 50–60 days) there is no need to stop it immediately on diagnosis of amiodarone-induced thyrotoxicosis, and indeed it may provide some cardioprotection from the thyrotoxicosis initially. The decision to continue or stop the amiodarone should be made by a cardiologist familiar with the patient's condition so that an alternative therapy, e.g. beta-blockade, can be considered. As amiodarone blocks the uptake of iodide by the thyroid gland, the only effective therapies for AIT include high dose antithyroid medications or surgery. Beta-blockers or glucocorticoids may have a role in treatment.

Iodine

Excess iodine from any source may cause hyperthyroidism (iodine-induced hyperthyroidism or the Jod-Basedow effect). For this reason, for patients with known nodular thyroid disease, clinicians need to consider whether it is necessary to administer iodine-containing materials (e.g. radiological contrast agents, or drugs such as amiodarone) as the extra iodine load may result in subsequent thyrotoxicosis.

Thyrotoxicosis factitia

This is thyrotoxicosis that occurs from excessive exogenous intake of thyroid hormone. Patients are usually aware that they are taking the medication although may deny it. It may also be taken inadvertently as part of weight loss preparations. Typically there are typical biochemical and clinical features of thyrotoxicosis along with suppressed serum thyroglobulin levels and reduced uptake on technetium scanning.

Patient information

American Thyroid Association website: www.thyroid.org/patients/brochures.html

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'There are in fact two things, science and opinion; the former begets knowledge, the latter ignorance. '

Hippocrates, Law