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Thyroiditis and Thyroid Storm

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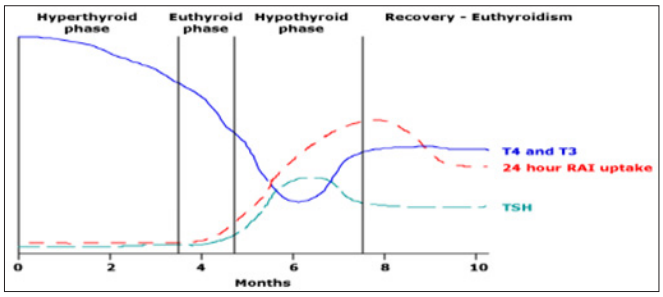
Autoimmune Thyroiditis

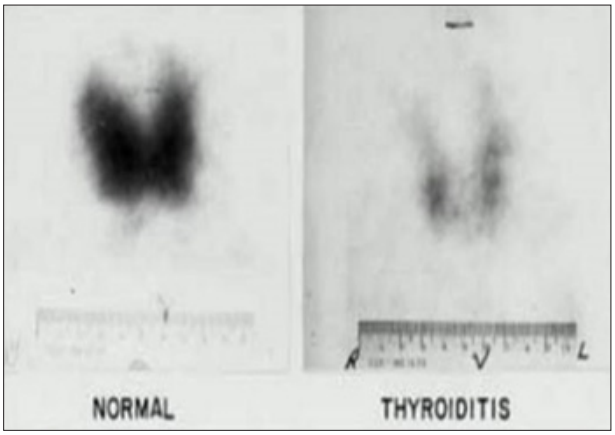
- Principal goal of management is to monitor for progression to a permanent overtly hypothyroid state warranting ongoing treatment with a replacement dose of levothyroxine
- TSH and free or total T4 levels can be checked at 3-6 week intervals to track status

Diagnosis[1,2,3,4,5]

Labs/Imaging

- Low TSH and high T4 and T3 in the hyperthyroid phase
- Elevated TSH and low T4 in the hypothyroid phase
- Presence of TPO antibodies and TG antibodies indicates autoimmunity, as in Hashimoto and postpartum/silent thyroiditis, but it is not 100% sensitive or specific.
- Elevated ESR and/or CRP levels indicate the presence of inflammation, as in subacute thyroiditis
- Low radioiodine uptake differentiates the hyperthyroid phase of thyroiditis from Graves' disease, which has increased uptake





Checkpoint Inhibitors

- Immunomodulatory antibodies that inhibit programmed cell death receptor 1 and receptor ligand 1 (PD-1, PD-L1) and cytotoxic T-lymphocyte-associated antigen 4 (CTLA-4)
- Immunologic enhancement can trigger new- onset autoimmune thyroiditis
- May present with severe thyrotoxicosis followed by rapid progression to overt hypothyroidism

Subacute Thyroiditis

- Also known as granulomatous thyroiditis or DeQuervain's thyroiditis
- Inflammation leads to the formation of granulomas consisting of giant cells clustered about foci of degenerating thyroid follicles
- Onset is often preceded by a nonspecific viral illness
- Patients present with pain localized to the thyroid gland which may radiate upwards to the neck and jaw
- Thyroid gland may be slightly enlarged and exquisitely tender to palpation
- Inflammation may spread from one lobe to the other
- Low thyroid uptake

- Lab tests may reveal an elevated erythrocyte sedimentation rate (ESR)
- Thyroid function test profiles typically reveal a thyrotoxic phase lasting 1-4 weeks caused by leakage of thyroid hormone, followed by a resolving hypothyroid phase lasting 1-3 months caused by impaired production of thyroid hormone
- Treatment is directed at controlling pain and minimizing thyrotoxic symptoms
- Pain may respond to treatment with high doses of aspirin or NSAIDS, but doses that are effective frequently cause gastrointestinal side effects
- Prednisone is the mainstay of therapy
- Low doses of beta-blockers (atenolol, metoprolol, long-acting propranolol) can be prescribed to relieve thyrotoxic symptoms
- TSH and free or total T4 levels should be tracked at 2-4 week intervals to identify transitions
- Less likely to require supportive treatment with levothyroxine

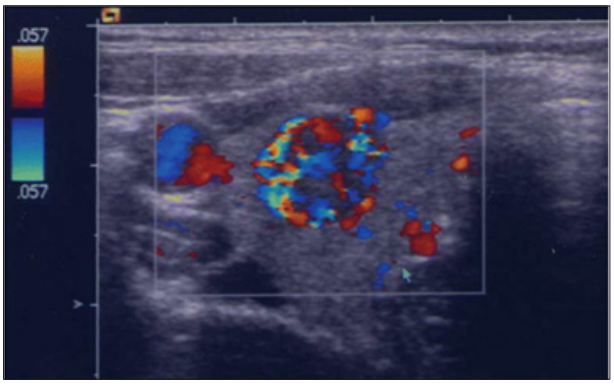
Prednisone

- Usually started at a dose of 20-40 mg daily - Relief occurs within 1-3 days of starting treatment
- Once pain has been controlled, the dose can be tapered over days to weeks
- Pain may flare up again once treatment is discontinued
- Can start back on treatment with the last effective dose and taper at a slower rate

Amiodarone-Induced Thyrotoxicosis

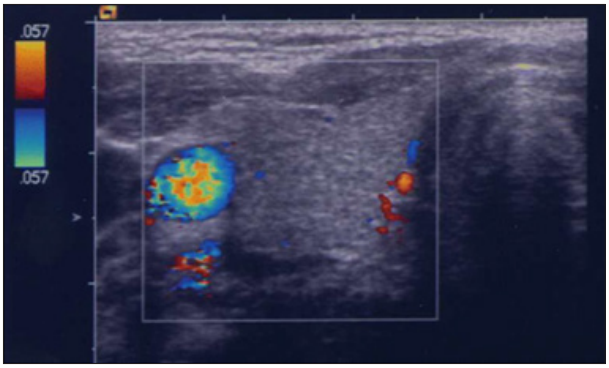
Type 1 AIT

- Caused by unregulated production and secretion of thyroid hormone
- Usually occurs in patients with underlying nodular or autoimmune thyroid disease
- Responds to treatment with antithyroid drugs



Type 2 AIT

- Caused by inflammation with resultant leakage of thyroid hormone
- Distinction between the two types of AIT can be very difficult
- Thyroid uptake tends to be low in both cases
- Interleukin-6 may be high in type 2 AIT, but it isn't a useful clinical test
- Thyroid doppler may reveal high blood flow in type 1 AIT and low blood flow in type 2 AIT



Amiodarone-Induced Thyrotoxicosis

- The majority of cases in iodine-sufficient regions represent type 2 AIT
- Prednisone is the mainstay of therapy
- Concurrent treatment with methimazole or propylthiouracil may be considered if there is uncertainty
- A quick response to treatment with prednisone favors type 2 AIT

Prednisone

- Usually started at a dose of 40 mg daily
- Thyroid hormone levels usually begin to decrease within 3-7 days of starting treatment
- Once thyrotoxicois has been controlled, the dose can be tapered over weeks to months
- Thyrotoxicosis may flare up again once treatment is discontinued
- Can start back on treatment with the last effective dose and taper at a slower rate

Thyroiditis: Key Points

- Serologic diagnosis of autoimmune thyroiditis is only warranted in specific circumstances
- Thyrotoxicosis presenting with concurrent focal thyroid pain and tenderness is likely to be a manifestation of subacute thyroiditis
- The majority of cases of amiodarone-induced thyrotoxicosis in iodine-sufficient regions represent type 2 AIT
- Subacute thyroiditis and type 2 AIT respond well to treatment with prednisone, but associated pain and thyrotoxicosis may flare up when doses are tapered or discontinue

Thyroid Storm[6,7]

- Acute exacerbation of manifestations of hyperthyroidism, usually secondary to stressors (severe trauma, surgery, illness, childbirth)
- Fever, tachycardia, agitation or psychosis, confusion, GI symptoms
- 20% death rate

Diagnostic criteria for thyroid storm*

Thermoregulatory dysfunction	
Temperature (°F | °C)	
99 to 99.9 | 37.2 to 37.7	5
100 to 100.9 | 37.8 to 38.2	10
101 to 101.9 | 38.3 to 38.8	15
102 to 102.9 | 38.9 to 39.4	20
103 to 103.9 | 39.4 to 39.9	25
≥104.0 | >40.0	30
Central nervous system effects	
Mild	10
Agitation	
Moderate	20
Delirium	
Psychosis	
Extreme lethargy	
Severe	30
Seizure	
Coma	
Gastrointestinal-hepatic dysfunction	
Moderate	10
Diarrhea	
Nausea/vomiting	
Abdominal pain	
Severe	20
Unexplained jaundice	

Cardiovascular dysfunction	
Tachycardia	
99 to 109	5
110 to 119	10
120 to 129	15
130 to 139	20
≥140	25
Atrial fibrillation	10
Heart failure	
Mild	5
Pedal edema	
Moderate	10
Bibasilar rales	
Severe	15
Pulmonary edema	
Precipitant history	
Negative	0
Positive	10

* A score of 45 or more is highly suggestive of thyroid storm; a score of 25 to 44 supports the diagnosis; and a score below 25 makes thyroid storm unlikely.

TREATMENT of Thyroid Storm[8,9,10]

- Supportive therapy – IVF, cooling blankets, glucose
- Antithyroid agents (PTU or methimazole)
- Propylthiouracil (PTU) – preferred over methimazole
- Both drugs interfere with the production of thyroid hormones, but PTU also decreases the peripheral conversion of T4 to T3, and T3 is more metabolically active than T4 (3 to 4 times).
- Both agents can be administered by NG tube or rectally if needed.
- Note: the FDA has recently released an advisory on PTU for its liver toxicity potential.
- Iodine administration
- Used to temporarily inhibit thyroid hormone production and release (never give before PTU or Methimazole).
- Iodine administration causes temporary suppression of thyroid peroxidase – Wolff-Chaikoff effect – which decreases:
- Organification of iodide
- production and release of thyroid hormones.
- The effect lasts for up to 2 weeks, after which the effect wears off. Therefore, iodine administration is unsuitable as a long-term therapeutic option.
- Iodine is administered in various formulations, including saturated solution of potassium iodide (SSKI) or Lugol's solution orally.
- Beta blockers for heart rate control and decreased conversion of T4 to T3
- Promptly administer antiadrenergic drugs (eg, propranolol) to minimize sympathomimetic symptoms (i.e, tachycardia; cardiac dysrhythmias).
- Important to avoid propranolol in conditions such as asthma, chronic obstructive pulmonary disease or decompensated heart failure.
- Cardioselective beta blockers such as atenolol or metoprolol may be administered in patients with reactive airway disease, and non-dihydropiridine calcium channel blockers may be used when beta blockers are contraindicated. The use of intravenous beta blockers (as esmolol) allows quick dose titration with minimization of side effects.
- Dexamethasone to prevent peripheral conversion of T4 to T3-Administer glucocorticoids to decrease peripheral conversion of T4 to T3. This may also be useful in preventing relative adrenal insufficiency due to hyperthyroidism and improving vasomotor symptoms.
- Cholestyramine 4g orally two to four times each day has been used in the management of thyrotoxicosis due to reduced reabsorption of metabolized thyroid hormone from the enterohepatic circulation.
- Plasma exchange
- Thyroidectomy is occasionally employed in the management of thyroid storm refractory to medication, but is associated with a risk of storm exacerbation if preoperative thyroid hormone levels are high.

Management of thyroid storm should not disregard the search for and treatment of precipitating factors. An active search should be made for infection, and antibiotics should be chosen on the basis of likely pathogens or microbial cultures. Other likely precipitants, such as trauma, MI, DKA, and other underlying processes, should be managed as per standard care.

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