Update: Thyroid Emergencies

•

Introduction

Often referred to as the "great imitator," the thyroid gland exerts its influence on virtually every organ system. Thyroid emergencies are rare but life-threatening conditions. They occur most commonly in patients with a known underlying thyroid disorder. Diagnosis relies primarily on clinical and historical findings. Treatment must be initiated before thyroid hormone assay results are available. A precipitating factor should always be sought. The clinical manifestations of thyroid dysfunction can range from isolated symptoms to cardiogenic shock with immediate risk to life.

The aim of this review is twofold: first, to describe the principal symptoms and organ dysfunction associated with thyroid disease; and second, to present thyroid emergencies which, though rare, constitute genuine life-threatening conditions.

Diagnostic Approach

History

The patient typically has a known thyroid disorder in whom a precipitating factor is identified: infection, acute coronary event, surgical procedure, or modification of thyroid treatment.

Clinical Examination (1, 2, 3)

Clinical Feature	Thyrotoxic Crisis	Myxedema Coma
Cardiac	Tachycardia Atrial fibrillation (AF) Heart failure Exacerbation of coronary insufficiency	Bradycardia Hypotension Hemodynamic failure
General Signs	Asthenia Muscle weakness Tremors Hyperthermia Diaphoresis	Edema Cold extremities Hypothermia Central hypoventilation
Gastrointestinal	Abdominal pain Diarrhea Nausea, vomiting Jaundice	Ileus (bowel obstruction)
Neurological	Altered higher cognitive functions Seizures	Impaired consciousness Coma

Laboratory Investigations (1, 2, 3)

Laboratory Investigation	Thyrotoxic Crisis	Myxedema Coma
Thyroid Panel (TSH, T3, T4)	*Do not delay treatment pending results*	
Complete Blood Count	Leuko-neutropenia	Anemia
Electrolytes	Hyperglycemia	Hyponatremia (SIADH)
Serum Calcium	Elevated	—
Troponin	Myocardial injury	—
Liver Function Tests	Elevated transaminases	—
Blood cultures / Urinalysis	*Workup for infectious precipitating factor*	

Imaging

- Electrocardiogram (ECG): to assess for arrhythmias or signs of myocardial ischemia.
- Chest X-ray: to identify an infectious focus or pleural effusion.
- CT scan and MRI of the neck and chest: indicated in cases of substernal (plunging) goiter.
- Echocardiography: indicated in myxedema coma to assess for pericardial effusion.

Thyroid Emergencies

Thyrotoxic Crisis (Thyroid Storm) (1, 7, 8, 9, 10)

Thyrotoxic crisis (TC), also known as thyroid storm, is defined as a sudden, severe exacerbation of hyperthyroidism. It is a life-threatening complication of hyperthyroidism, with a mortality rate of 10–30% that is virtually universal in the absence of treatment. Prognosis depends on the speed of management and the degree of resulting multi-organ failure. It is a diagnostic and therapeutic emergency that is becoming increasingly rare.

Its pathogenesis is incompletely understood; several mechanisms appear to contribute:

- Rapid and abrupt rise in circulating thyroid hormone levels
- Sympathetic nervous system hyperactivity
- Amplification of cellular response to thyroid hormones

A precipitating factor is frequently identified (infection, surgical procedure, hypo- or hyperglycemia, ischemic event, or significant emotional stress). The clinical presentation involves multiple organ system failure. The patient presents with hyperthermia, diaphoresis, and dehydration. This is associated with cardiac thyrotoxicosis (atrial fibrillation, ventricular arrhythmias, pulmonary arterial hypertension [PAH], orthostatic hypotension, predominantly right-sided heart failure, coronary insufficiency) and neuropsychiatric disturbances (agitation, obtundation, seizures, coma). Gastrointestinal symptoms may also be present (vomiting, diarrhea, intestinal ileus, jaundice, or hepatic failure).

Myxedema Coma (2, 6, 7, 8, 9)

Myxedema coma represents profound and prolonged thyroid hormone deficiency. This complication is currently rare. A precipitating factor is often identified (infection, surgery, sedative or antidepressant medications). Clinically, it presents as a quiet coma with bradycardia, bradypnea, hypothermia, hypotension, slow and diminished deep tendon reflexes, no focal neurological signs, and no obvious etiology on initial workup. Seizure episodes have been reported. Cerebrospinal fluid (CSF) analysis via lumbar puncture (LP) may occasionally reveal elevated protein (hyperproteinorachia). Hyponatremia is a consistent finding. The prognosis of myxedema coma is severe, with a mortality rate of 15–60%.

Hashimoto Encephalopathy (8, 11)

Hashimoto encephalopathy is an emerging and underrecognized cause of neurological emergencies. Its clinical presentation is highly variable. The course may be acute, subacute, or chronic. Manifestations may include cognitive deterioration, seizures, stroke-like episodes, or coma.

Graves' Orbitopathy (Thyroid Eye Disease) (1, 5, 8)

Graves' orbitopathy is an emergency characterized by eyelid retraction, exophthalmos (proptosis), local inflammatory signs, eyelid edema, and restriction of extraocular movement due to involvement of one or more muscles — potentially resulting in diplopia and loss of visual acuity. In selected cases, combined medical and surgical management may be required to prevent permanent visual impairment.

Thyrotoxic Hypokalemic Periodic Paralysis (8, 12)

This is an increasingly rare complication of hyperthyroidism. It is characterized by episodic hypokalemia accompanied by muscle weakness. It occurs in the context of hyperthyroidism and results from transcellular potassium shift into cells, driven by thyroid hormone-mediated upregulation of Na-K-ATPase activity in skeletal muscle.

Compressive Emergencies: Goiter (7, 8, 9)

A compressive goiter constitutes a surgical emergency due to compression of the trachea and/or esophagus, manifesting as dysphonia, dysphagia, and dyspnea. The clinical presentation may mimic an acute asthma attack or acute pulmonary edema (APE).

Management

Supportive Treatment (1, 2, 3)

Thyrotoxic Crisis

- Fluid resuscitation with isotonic saline (0.9% NaCl).

- Management of hyperthermia with antipyretics and physical cooling (ice packs).
- Correction of electrolyte disturbances, particularly hypercalcemia.
- In cases of agitation and/or seizures: sedation with diazepam.

Myxedema Coma

- Fluid restriction: isotonic saline 50–100 mL if Na^+ < 120 mmol/L.
- Gradual external rewarming.
- Ventilatory support to correct hypoxia and desaturation in cases of respiratory failure related to pulmonary edema.
- Corticosteroid therapy with hydrocortisone hemisuccinate (HSHC) 50–100 mg every 6–8 hours, to compensate for functional hypocortisolism associated with hypometabolism.

Specific Treatment

Thyrotoxic Crisis (3)

- Antithyroid drugs (ATDs): Propylthiouracil (PTU) 50 mg tablets — loading dose of 1 g, then 200 mg every 4–6 hours; or Carbimazole (Neo-Mercazole®) 20 mg every 4–6 hours. There is a rare but serious risk of agranulocytosis, requiring close monitoring of blood counts.
- Beta-blockers: Counteract adrenergic hyperactivity and inhibit peripheral conversion of T4 to T3. Propranolol is generally used at 60–80 mg every 4 hours orally, or as an IV bolus of 0.5–1 mg given slowly, followed by 4–8 mg/hour via syringe pump. Alternatively, Esmolol — bolus of 250–500 µg/kg followed by 50–100 µg/kg/min via syringe pump — may be preferred due to its short half-life, particularly when cardiac function is compromised.
- Corticosteroids: Prevent functional adrenal insufficiency and may inhibit peripheral T4-to-T3 conversion. Hydrocortisone hemisuccinate (HSHC) — bolus 300 mg IV then 100 mg every 8 hours — or Dexamethasone 2 mg IV or orally every 6 hours.

Myxedema Coma

Treated with thyroid hormone replacement therapy:

- Levothyroxine (T4) injection 200 µg/mL: loading bolus of 200–500 µg, then 25–100 µg/day.
- Liothyronine (T3), Cynomel® 25 µg tablets: rapid onset of action and short half-life.
- Combination T3 + T4 (Euthyral®) tablets: 20 µg T3 + 100 µg T4.

Conclusion

The clinical presentations of thyroid dysfunction are often misleading, including in severe forms. These conditions, fortunately rare, constitute therapeutic emergencies, and treatment should not be delayed pending biochemical confirmation of thyroid dysfunction.

Despite appropriate management, mortality from severe thyroid emergencies remains considerable, and therapeutic protocols have changed relatively little in recent years.

References

[1] Société Française d'Endocrinologie. Hyperthyroïdie. http://www.sfendocrino.org/article/398/item-246-ndash-hyperthyroidie

[2] Société Française d'Endocrinologie. Hypothyroïdie. http://www.sfendocrino.org/article/399/item-248-ndash-hypothyroidie

[3] Pateron D, Raphaël M, Trinh-Duc A. Urgences thyroïdiennes. In: Méga-guide pratique des urgences. Fiche 73:478–481.

[4] Ae Lee K, Taek Park K, Min Yu H, et al. Subacute thyroiditis presenting as acute psychosis: a case report and literature review. Korean J Intern Med. 2013;28:242–6.

[5] Soeters MR, Van Zeijl CJ, Boelen A, et al. Optimal management of Graves orbitopathy: a multidisciplinary approach. Neth J Med. 2011;69 Suppl(7):302–8.

[6] Boileau E. L'urgence thyroïdienne: la grande imitatrice! Le Médecin du Québec. 2012;47 Suppl(2):49–56.

[7] Andronikof M. Urgences thyroïdiennes. Encycl Méd Chir (Elsevier, Paris). Urgences. 2014;24-132-A10.

[8] Papi G, Corsello SM, Pontecorvi A. Clinical concepts on thyroid emergency. Front Endocrinol. 2014;5(102):1–11.

[9] Wanczuk W. Myocardial infarction and shock associated with thyrotoxicosis. Anesteziol Intens Ter. 2010;42 Suppl(3):142–6.

[10] Tzakas P, Sit SW. Progressive impairment of cognition and motor function: Hashimoto encephalopathy. CMAJ. 2011;183 Suppl(8):495–7.

[11] Lin YC, Wu CW, Chen HC, et al. Surgical treatment for thyrotoxic hypokalemic periodic paralysis: case report. World J Surg Oncol. 2012;10(21):1–3.

[12] Lionet A, Barsumau J, Azar R. Paralysie périodique hypokaliémique thyréotoxique chez un Caucasien. Néphrology. 2004;25 Suppl(1):29–32.

[13] Bula G, Paliga M, Koziolek H, et al. Acute respiratory failure in goitre patients qualified for operative treatment. Endokrynol Pol. 2013;63 Suppl(3):215–9.