Myxedema Coma and Thyroid Storm: Diagnosis and Management

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Myxedema Coma and Thyroid Storm: Diagnosis and Management

Maybelline V. Lezama, MD, Nnenna E. Oluigbo, MD, and Jason R. Ouellette, MD

INTRODUCTION

Thyroid storm and myxedema coma are disorders that fall within the spectrum of endocrine emergencies. Considered true medical emergencies, these entities should be suspected in patients with extreme manifestations of hypothyroidism or hyperthyroidism. Although they have a low incidence, they must be promptly recognized and adequately treated in order to reduce associated morbidity and mortality. Aggressive treatment should be initiated in the medical intensive care unit and should not be delayed until the results of thyroid function tests are available. With prompt recognition, morbidity and mortality related to myxedema coma and thyroid storm may be diminished.

MYXEDEMA COMA

CASE PRESENTATION

A 65-year-old woman with no known past medical history is brought to the emergency department with altered mental status. On arrival, the patient is drowsy but arousable. Vital signs include: oral temperature, 95°F; heart rate, 52 bpm; blood pressure, 110/50 mm Hg; respiratory rate, 15 breaths/min; and oxygen saturation, 92% on 4 L of oxygen. The physical examination in the emergency department is unremarkable. Notable laboratory values include: white blood cell count (WBC), 12,000 cells/µL; sodium, 128 mEq/L; potassium, 5 mEq/L; blood urea nitrogen, 8 mg/dL; and creatinine, 1.5 mg/dL. The arterial blood gas reveals acute respiratory acidosis with pH of 7.23; PaCO₂ of 63.7 mm Hg; and PaO₂ of 71.2 mm Hg on room air. Chest radiograph and head computed tomography are unremarkable. Diagnosis of hypercapnic respiratory failure is made, and the patient is admitted to the medical intensive care unit. The patient becomes more lethargic and her respiratory acidosis worsens, requiring mechanical ventilation. A more exhaustive examination reveals generalized puffiness, periorbital edema, ptosis, and macroglossia, and her extremities are dry and cool with nonpitting edema. Myxedema coma is highly suspected, and the patient is started on intravenous levothyroxine. Her thyroxine (T₄) level is below 0.35 ng/dL and her thyroid-stimulating hormone level is above 200 µIU/mL.
Myxedema Coma and Thyroid Storm

Table 1. Common Precipitating Factors of Myxedema Coma

<table>
<thead>
<tr>
<th>Stroke</th>
<th>Congestive heart failure</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>Gastrointestinal bleeding</td>
<td>Infections</td>
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<td>Drugs</td>
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<td>Anesthetics</td>
<td>Amiodarone</td>
<td>Lithium</td>
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<tr>
<td>Sedatives</td>
<td>Metabolic disturbances</td>
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<td>Hypoglycemia</td>
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<td>Hypoxemia</td>
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<td>Acidosis</td>
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<td>Hypercapnia</td>
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OVERVIEW

Myxedema coma is an extreme manifestation of hypothyroidism. It presents as central nervous system dysfunction, defective thermoregulation, and cardiopulmonary decompensation. While most believe that patients must be comatose for myxedema coma to be considered, deterioration in the patient’s mental status in the appropriate clinical setting is enough to consider this entity.1

Epidemiology

Myxedema coma was first reported by Ord in 1879 in London. It is a rare disorder, with only approximately 300 cases described in the literature. Patients are typically elderly females and often have longstanding, undiagnosed hypothyroidism.2 More than 90% of cases occur during winter months, suggesting that low temperatures are a potential contributing factor. Age-related loss of temperature regulation, in addition to a decrease in heat production secondary to hypothyroidism, may also contribute.1

Mortality had once been estimated to range from 50% to 60%.3 However, a high level of clinical suspicion with earlier recognition, as well as advances in intensive care and overall management, have decreased mortality to approximately 20% to 25%.4

ETIOLOGY

Myxedema most commonly develops in patients with neglected, inadequately treated, or undiagnosed hypothyroidism. Multiple factors appear to precipitate myxedema coma, including the following: gastrointestinal bleeding; infection; metabolic disturbances such as acidosis, hypoxemia, and hypercapnia; stroke; and cardiovascular compromise (Table 1).5

Many patients who develop myxedema coma are initially hospitalized with an unrelated medical condition. During hospitalization, the patient slowly develops a change in mental status. The diagnosis may not be suspected initially, especially when narcotics or sedatives are being used.3

Drugs such as amiodarone and lithium are also reported as precipitants of myxedema coma, especially in patients with a history of lithium- or amiodarone-induced hypothyroidism. This is due in part to the effects of these drugs on thyroid hormone metabolism.6,7 Lithium increases intrathyroidal iodine content, inhibits the coupling of iodotyrosine residues to form T4 and triiodothyronine (T3), and inhibits their secretion.8 Amiodarone has a high iodine content, and hypothyroidism can occur due to the antithyroid effects of iodine. It also has an effect on thyroid hormone synthesis and release, especially in patients with underlying thyroid disease.6

CLINICAL PRESENTATIONS

The diagnosis of myxedema coma is a clinical one, and virtually every organ system can be af-
fected. Patients often present with symptoms of hypothyroidism, including excessive fatigue, weight gain (despite a decrease in appetite), constipation, and cold intolerance. Atypical presentations can occur in elderly patients, who may simply exhibit decreased mobility.9

Central Nervous System
Altered mental status may range from mild confusion, apathy, and lethargy to obtundation and coma. While all patients with myxedema coma present with some degree of altered mental status, only a few present with true coma.10 Cognitive impairment (eg, decreased attention, motor speed, memory, and visual/spatial organization) and psychiatric disorders (eg, depression, dementia) are other manifestations. The mechanism for this change in mental status is largely unknown; however, it has been reported that there is decreased cerebral blood flow and cerebral glucose metabolism in hypothyroid patients.11 Focal and generalized seizures may also occur, possibly related to concomitant hyponatremia, and hypoglycemia is seen in up to 25% of patients.12 There is also a delay in deep tendon reflexes.13

Respiratory
Alveolar hypoventilation also occurs in myxedema as a result of depressed hypoxic ventilatory drive and hypercapnic ventilatory response.14,15 The respiratory depression seen in these patients is related to both a reduced central nervous system drive to breathe and decreased respiratory muscle activity.14,16 Increased alveolar-arterial oxygen gradient and ventilation-perfusion mismatch are common and contribute to hypercapnia seen in these patients. Pleural effusions as a result of fluid accumulation may also occur. Myxedematous infiltration of the pharynx and tongue may significantly contribute to the need for mechanical ventilation.17 Respiratory dysfunction may also lead to obstructive sleep apnea,17 and this may be associated with weight gain and obesity.

Cardiovascular
Cardiovascular manifestations include bradycardia and low cardiac output due to decreased cardiac contractility; however, frank congestive heart failure is rare.3 Narrow pulse pressure and diastolic hypertension is often seen in the early stages. Hypotension may be seen in the later stages, and it is frequently exacerbated by bradycardia, low cardiac output, and decreased blood volume. Increased systemic peripheral resistance occurs and is thought to be due to decreased T3 levels. Pericardial effusions may occur as a result of increased capillary permeability, leading to fluid accumulation. Cardiac tamponade, however, is rare.16

Renal
Renal impairment may be severe in patients with myxedema and is attributed to decreased renal blood flow and increased vascular resistance in the afferent and efferent arterioles, which leads to a low glomerular filtration rate.18,19 There is also decreased free water clearance and reduced levels of Na+/K+ ATPase with concomitant reduction in sodium re-absorption; these changes lead to hyponatremia, which is commonly seen in these patients.18

Gastrointestinal
The most common gastrointestinal presentation is constipation, which occurs as a result of decreased intestinal motility.18 This may progress to gastric atony, megacolon, and paralytic ileus, mimicking an acute abdomen, as these patients may present with anorexia, nausea, abdominal pain, and distension.20 Ascites may be present but is uncommon.
Skin and Facial Appearance

Patients may present with classic myxedematous facies, characterized by generalized puffiness, ptosis, macroglossia, coarse and sparse hair, and periorbital edema. The skin is dry, pale, and thickened with nonpitting edema (myxedema); edema occurs due to increased dermal glycosaminoglycan content, which traps water. The hair is dry, brittle, and falls out easily, and the voice may be hoarse.¹³

Metabolic

Hypothermia is usually present, with a body temperature that may fall as low as 24°C (75°F); increased severity is associated with worse prognosis.³ Hypothermia is frequently not recognized as many thermometers do not register extremely low temperatures. It is important to be aware of the presence of complicating factors, such as underlying infection, as fever may be masked by coexistent hypothermia.⁵

LABORATORY TESTING AND OTHER STUDIES

Thyroid function tests in patients with myxedema coma always demonstrate reduced free T₄ and T₃. The thyroid-stimulating hormone level may be elevated, suggesting a primary thyroid disorder, but it also may be low or normal in central/secondary hypothyroidism. Arterial blood gases may reveal hypoxia and hypercapnia with respiratory acidosis. Electrolyte abnormalities include hyponatremia, due to impairment in free water secretion and increased vasopressin secretion. Hypoglycemia may be present as a result of down-regulation of metabolism,³ or it may indicate coexisting adrenal insufficiency. Elevated creatinine may occur due to decreased renal perfusion and glomerular filtration rate. Complete blood count may show normocytic anemia and mild leukopenia. Creatine kinase and serum transaminases are often elevated, and the lipid profile often reveals hyperlipidemia. Serum cortisol level should be checked to evaluate for coexisting adrenal insufficiency. Electrocardiogram abnormalities include bradycardia, decreased voltage, prolonged QT interval, nonspecific ST-T changes, and Osborn waves in cases of hypothermia. Chest radiograph may reveal pleural effusions and cardiomegaly.

DIAGNOSIS

A thorough past medical history should be elicited in all patients, especially if there is a prior history of thyroid disease, thyroidectomy, radioiodine therapy, or noncompliance with thyroid therapy. Given a reasonable index of suspicion in a patient presenting as previously described, initiation of treatment should not be delayed until the results of thyroid function tests are available. Furthermore, because of concurrent illness, thyroid-stimulating hormone values may not be elevated in proportion to the severity of hypothyroidism. Thyroid-stimulating hormone is elevated in most patients. An exception is patients in whom hypothyroidism is attributed to hypothalamic or pituitary disease (< 5% cases of myxedema coma), in which case the thyroid-stimulating hormone is low or normal. Independent of whether the underlying cause of hypothyroidism is primary or secondary, all patients with myxedema coma have low total and free T₄ and T₃ concentrations.³

TREATMENT

Myxedema coma is an acute medical emergency and should be treated in the intensive care unit. Continuous monitoring of the patient’s cardiovascular and pulmonary status is required, and ventilatory support is often needed (Table 2).

Thyroid Hormone

The optimal mode of thyroid hormone therapy in patients with myxedema coma is controversial.
As the incidence of the condition is low, no adequate trials have been conducted to compare different regimens. Some of the arguments for T₄ (levothyroxine) over T₃ (liothyronine) therapy are based on the fact that levothyroxine therapy, despite its slow onset of action, maintains steadier hormone levels with less risk of adverse events. Because T₄ → T₃ conversion is impaired in sick, hypothyroid patients, some experts prefer the faster-acting liothyronine. Liothyronine has the disadvantage of higher cost and low availability. In addition, its serum concentration fluctuates between doses.²²

Patients with myxedema coma may absorb drugs poorly, and for this reason, it is important that levothyroxine be administered intravenously. An initial loading dose of 300 to 400 µg of levothyroxine intravenously is indicated (due to marked total body depletion) followed by 80% of the calculated full replacement dose intravenously daily. If after a few days the clinical response has been suboptimal, some experts recommend adding intravenous liothyronine to the regimen at a dose of 5 µg IV every 8 hours. A rise in body temperature and the return of normal cerebral and respiratory function often indicate that therapy is adequate.²

In older patients, especially those with underlying cardiovascular disease, treatment should be started slowly as there could be a risk of precipitating angina, heart failure, and arrhythmia. Dosage should be adjusted accordingly and thyroid-stimulating hormone should be measured frequently.²,³

**Glucocorticoid Therapy**

Due to the possibility of concomitant adrenal insufficiency, it is prudent to initiate hydrocortisone 100 mg intravenously, followed by 50 mg intravenously every 6 hours. In addition, thyroid hormone therapy may increase cortisol clearance and precipitate adrenal insufficiency.⁵ Glucocorticoid therapy can be withdrawn if the pretreatment plasma cortisol is 25 µg/dL or greater, or if results of a corticotropin (ACTH)-stimulation test are within normal limits.

**Supportive Measures**

Associated illnesses, such as infection or heart failure, must be evaluated and appropriately treated. Hypotonic fluids should be avoided as they may worsen hyponatremia. Passive rewarming is preferred. Active rewarming is not recommended as it increases the risk of vasodilatation, which may lead to hypotension and cardiovascular collapse.²³

**THYROID STORM**

**CASE PRESENTATION**

A 45-year-old woman with no significant past medical history and who has not seen a physician in
several years is admitted with a 2-day history of flu-like symptoms, worsening shortness of breath, fever, nausea/vomiting, and diarrhea. In the month prior to admission, she developed increased shortness of breath (especially on exertion), palpitations, fatigue, and 8-lb weight loss despite an increased appetite. She is not on any medications at home. Her family history is significant for Graves’ disease in her mother and sister. She is a nonsmoker and denies any history of alcohol or illicit drug use. Her vital signs on admission include: temperature, 103°F (39.4°C); heart rate 145 to 160 bpm; blood pressure, 120/60 mm Hg; respiratory rate, 22 breaths/min; and oxygen saturation, 96% on room air.

On physical exam, she is alert and oriented to person but not to place or time. She appears restless and anxious. She has no exophthalmos, lid lag, or stare. Her thyroid gland is diffusely enlarged with a bruit. Her cardiovascular exam reveals a laterally displaced apical beat with irregular tachycardia. She has a fine resting tremor of her upper extremities and a nonpitting pretibial edema in both lower extremities. Her deep tendon reflexes are hyperactive. The rest of her exam is unremarkable.

Her electrocardiogram shows atrial fibrillation with rapid ventricular response. Her chest radiograph reveals cardiomegaly. Results of her laboratory studies include: WBC, 15,000 cells/µL; hemoglobin, 9 g/dL; platelet count, 250,000 cells/µL; sodium, 136 mEq/L; potassium, 3.2 mEq/L; and creatinine, 1.0 mg/dL. Urinalysis is negative. Blood cultures are taken.

A diagnosis of a viral illness is made and underlying hyperthyroidism is suspected. Intravenous fluid is initiated and a stat dose of oral metoprolol and potassium chloride is given. Six hours after admission, she has worsening dyspnea and is hypotensive as well as increasingly irritable and agitated. She is transferred to the intensive care unit for close monitoring and treatment with vasopressors. A presumed diagnosis of thyroid storm is made and propylthiouracil and propranolol are started. She is also given a stat dose of hydrocortisone. Her thyroid-stimulating hormone level and free T₄ level are reported at < 0.01 µU/mL and 8 ng/dL, respectively.

OVERVIEW

Thyroid storm is a syndrome characterized by excess thyroid concentration and is considered the most extreme manifestation of thyrotoxicosis. The diagnosis is clinical, bearing no direct relation to the absolute levels of thyroid hormones in the serum. Although some authors have proposed a point system for determining whether a patient’s condition represents true storm or severe thyrotoxicosis, the distinction between these 2 entities is not useful clinically.

EPIDEMIOLOGY

Thyroid storm is relatively rare, with a frequency of less than 10% in hospitalized patients with thyrotoxicosis. Mortality associated with this disorder ranges from 20% to 30%. Thyroid storm typically occurs in untreated or partially treated thyrotoxic patients.

ETIOLOGY

Graves’ disease is the most common underlying cause of thyrotoxicosis and is a common cause of hyperthyroidism in middle-aged patients. It is an autoimmune disease in which the presence of anti–thyroid-stimulating-hormone antibodies cause hyperstimulation of thyroid hormone along with uncontrolled thyroidal synthesis and secretion of T₄ and T₃.

Thyroid storm may also occur in patients with a solitary toxic adenoma and toxic multinodular goiter. Rare causes of thyrotoxicosis leading to thyroid storm include hypersecretory thyroid carcinoma, thyrotropin-secreting pituitary adenoma,
struma ovarii/teratoma, and human chorionic gonadotropin–secreting hydatidiform mole.\textsuperscript{28,29}

Historically, thyroid surgery in patients with uncontrolled hyperthyroidism was the most common precipitant of thyroid storm. Currently, infections appear to be the most common culprit.\textsuperscript{24} Other precipitating factors associated with thyrotoxic crisis include stress, trauma, thyroidal or nonthyroidal surgery, diabetic ketoacidosis, labor, heart disease, discontinuation of antithyroid drugs, and rarely, radioactive iodine treatment (Table 3).\textsuperscript{30}

### Table 3. Precipitating Factors in Thyroid Storm

<table>
<thead>
<tr>
<th>Category</th>
<th>Factors</th>
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<tbody>
<tr>
<td>Infection</td>
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<td>Stress</td>
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<tr>
<td>Trauma</td>
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<td>Non-thyroidal surgery</td>
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<tr>
<td>Diabetic ketoacidosis</td>
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<tr>
<td>Psychosis</td>
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<tr>
<td>Labor</td>
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<td>Myocardial infarction/heart diseases</td>
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<tr>
<td>Pulmonary thromboembolism</td>
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<td>Drugs</td>
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<td>Salicylates</td>
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<tr>
<td>Pseudoephedrine</td>
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<tr>
<td>Thyroid-related factors</td>
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<tr>
<td>Excessive ingestion or intravenous administration of iodine (radio-contrast dye, amiodarone)</td>
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<tr>
<td>Discontinuation of antithyroid drugs</td>
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<tr>
<td>Thyroid surgery</td>
<td></td>
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<tr>
<td>Thyroid injury (palpation, infarction of adenoma)</td>
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<tr>
<td>Radioiodine treatment</td>
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</tbody>
</table>

### PATHOPHYSIOLOGY

The effects of thyroid storm are due to an increased concentration of free thyroid hormone.\textsuperscript{31} Another suggested mechanism of thyroid storm is alteration of tissue responsiveness to catecholamines by thyroid hormone via modulation of adrenergic receptor expression or post-receptor modifications in the submembranetic signaling pathways. This mechanism may explain why features of thyroid storm are similar to those seen in catecholamine excess.\textsuperscript{32,33}

### CLINICAL PRESENTATION

Thyroid storm is characterized by an exaggeration of the clinical manifestations of hyperthyroidism; however, fever, tachycardia, central nervous system dysfunction, and gastrointestinal symptoms predominate.\textsuperscript{33}

#### General

Patients may present with weight loss (despite unchanged caloric intake), generalized weakness, and fatigue. These patients also have a hypermetabolic state, which leads to increased heat production and loss, resulting in increased sweating and heat intolerance. Fever associated with inappropriately excessive diaphoresis is common and can easily progress to frank hyperpyrexia.\textsuperscript{33}

#### Central Nervous System

Neuropsychiatric symptoms, including anxiety, emotional lability, agitation, confusion, psychosis, and even coma may be seen in patients with thyroid storm.\textsuperscript{33} Neurologic findings may include muscle wasting, hyperreflexia, fine tremors, and periodic paralysis.\textsuperscript{28}

#### Gastrointestinal

Gastrointestinal symptoms typically manifest as diarrhea or hyperdefecation due to increased motor contraction of the small bowel.\textsuperscript{28} Patients may also present with nausea/vomiting and rarely jaundice.\textsuperscript{34}

#### Cardiovascular

Cardiorespiratory symptoms include palpitations and dyspnea on exertion due to decreased lung compliance or left ventricular failure.\textsuperscript{28} Congestive heart failure may occur, especially in the elderly,
and most patients have systolic hypertension. Cardiovascular findings include a hyperdynamic precordium with tachycardia, increased pulse pressure, and a strong apical impulse. Sinus tachycardia and atrial fibrillation occur in approximately 40% and 10% to 20% of patients with thyrotoxicosis, respectively.

Genitourinary
Oligomenorrhea and anovulation can be seen in females, while symptoms in males may include decreased libido and gynecomastia.

Eye and Skin/Thyroid
Typical eye findings include exophthalmos, ophthalmoplegia, diplopia, and conjunctival injection/chemosis. Thyroid gland findings will depend on the etiology of thyrotoxicosis and can vary. Myxedema associated with Graves’ disease typically occurs in pretibial areas as asymmetric, raised, firm, and pink to purple plaques of nonpitting edema. Hair loss, palmar erythema, and warm moist skin may also be seen in these patients.

In the Elderly
Elderly patients typically display few of the sympathomimetic features characteristic of the thyrotoxic state. The term apathetic thyrotoxicosis has been applied to these patients. They often present atypically with depression, lethargy, weakness, and cachexia. Agitation and confusion can mimic dementia, and unexplained weight loss occurs in up to 80%. Elderly patients may also present with atrial fibrillation, with a slow ventricular response in up to 20% of patients.

LABORATORY TESTING AND OTHER STUDIES
Serum total and free $T_4$ and $T_3$ levels are elevated and thyroid-stimulating hormone is undetectable. Mild hyperglycemia can be seen due to catecholamine-mediated inhibition of insulin release and increased glycogenolysis. Serum electrolytes are usually normal, although mild hypercalcemia can be observed secondary to both hemoconcentration and thyroid hormone–stimulated acute bone resorption. Nonspecific elevations in liver enzymes as well as lactate dehydrogenase and creatine kinase may be observed. A leukocytosis with a left shift may be seen, even in the absence of infection. Conversely, leukopenia can occur in cases of Graves’ disease.

Radioactive iodine uptake, although not necessary for diagnosis, may reveal increased uptake of radioiodine as early as 1 to 2 hours after isotope administration, indicating rapid intraglandular turnover of iodine.

DIAGNOSIS
The diagnosis of thyroid storm remains a clinical one. The results of laboratory tests can be indistinguishable from those seen in uncomplicated thyrotoxicosis. A very small study by Brooks and colleagues comparing the hormone levels in patients with thyroid storm and mild thyrotoxicosis found that total $T_4$ was similar in both groups but free $T_4$ was higher in the thyroid storm group. If the clinical suspicion is strong, initiation of treatment should not be delayed until laboratory confirmation becomes available.

TREATMENT
Initial treatment of thyroid storm involves stabilization, airway protection, oxygenation, intravenous fluids, and continuous monitoring in an ICU setting (Table 4).

Inhibition of New Hormone Synthesis and Prevention of $T_4 \rightarrow T_3$ Conversion
Antithyroid medications are the first-line therapy in the management of thyroid storm. They in-
include propylthiouracil, methimazole, and carbimazole (available in Europe only). These medications act by interfering with the thyroperoxidase-catalyzed coupling process by which iodotyrosine residues are combined to form $T_4$ and $T_3$. Propylthiouracil is a thiouracil bound to albumin (80%–90%), and it remains the drug of choice. Its mechanism of action includes inhibiting conversion of $T_4$ to $T_3$ outside the thyroid gland. The recommended dose is 800 to 1200 mg orally daily in divided doses of 200 to 300 mg every 6 hours. Methimazole is an imidazole and has a longer half-life than propylthiouracil. It does not have any effect on $T_4$ conversion to $T_3$. The recommended dose is 80 to 100 mg orally daily, divided in doses of 20 to 25 mg every 6 hours, which can be decreased to once or twice daily. Both medications are available in rectal preparations. Side effects of both antithyroid drugs include pruritus, urticaria, and fever. Dose-related agranulocytosis occurs with methimazole (rare at doses < 40 mg daily) but not with propylthiouracil.38

### Halt the Release of Stored Thyroid Hormone

Iodine therapy blocks the release of pre-stored hormone and decreases iodine transport and oxidation in follicular cells. This decrease in organification due to increasing doses of inorganic iodine is known as the Wolff–Chaikoff effect. Large amounts of exogenous iodine actually inhibit hormone formation. It is important to consider that despite maintenance of high doses of iodine, the thyroid gland eventually escapes inhibition after approximately 48 hours.39,40

Iodine therapy should be given 2 hours after antithyroid medications to allow organification blockade. The use of iodine is limited, and it is used...
only in severe thyrotoxicosis or thyroid storm in combination with thionamide therapy.\textsuperscript{23,39,40}

**Reverse Systemic Disturbances**

Hyperpyrexia should be managed aggressively, preferably with acetaminophen. Passive measures can also be used, including ice packs and cooling blankets. Volume depletion should be managed with fluid replacement.\textsuperscript{41}

Beta-blockade is essential in controlling the peripheral action of thyroid hormone, having beneficial effects on the associated fever, diaphoresis, agitation, psychosis, and gastrointestinal symptoms. In thyroid storm, propranolol is dosed at 60 to 80 mg every 4 hours, or 80 to 120 mg every 6 hours. The onset of action after oral dosing is within 1 hour. If oral propranolol is ineffective, intravenous propranolol and esmolol are reasonable options.\textsuperscript{42}

Glucocorticoids may be used as an adjunctive therapy and are effective in reducing T\textsubscript{3} levels as well as inhibiting the conversion of T\textsubscript{4} to T\textsubscript{3}.\textsuperscript{29,43} It also may be used to treat possible relative adrenal insufficiency.\textsuperscript{26}

**Assess the Precipitating Factor**

The precipitating event should be recognized promptly and managed appropriately, since treatment of the underlying pathology greatly increases chances of a successful outcome. Once the patient has been rendered euthyroid, definitive treatment should be considered with radiiodine therapy or surgery.\textsuperscript{26}

**SUMMARY POINTS**

- Myxedema coma and thyroid storm are considered true medical emergencies.
- These entities should be suspected in patients with extreme manifestations of hypothyroidism or hyperthyroidism.
- Aggressive treatment should be initiated in the medical intensive unit, even in the absence of known thyroid hormones levels at the time of treatment initiation.
- The principles in management of these emergencies include: rapid initiation of therapy with either thyroid hormone replacement or antithyroid medications; treatment of any precipitating cause; and supportive measures, including ventilation, if needed.
Myxedema Coma and Thyroid Storm


