Thyroid Gland Disorder Emergencies

Thyroid Storm and Myxedema Coma

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ABSTRACT

Although thyroid dysfunction will develop in more than 12% of the US population during their lifetimes, true thyroid emergencies are rare. Thyroid storm and myxedema coma are endocrine emergencies resulting from thyroid hormone dysregulation, usually coupled with an acute illness as the precipitant. Careful assessment of risk and rapid action, once danger is identified, are essential for limiting morbidity and mortality related to thyroid storm and myxedema coma. This article reviews which patients are at risk, explains thyroid storm and myxedema coma, and describes pharmacological treatment and supportive cares.

Keywords: hyperthyroidism, hypothyroidism, myxedema coma, thyroid, thyroid storm

Thyroid disorders most commonly are caused by autoimmune processes, leading to hormone excess or deficiency. The American Thyroid Association estimates that thyroid dysfunction will develop in more than 12% of the US population, making it relatively common. Crisis related to thyroid disorder, however, is rare. Thyroid storm and myxedema coma are endocrine emergencies resulting from thyroid hormone dysregulation, usually coupled with an acute illness as the precipitant. For those who experience thyrotoxicosis and myxedema coma, mortality rates are high. This article reviews which patients are at risk of these endocrine emergencies, describes thyroid storm and myxedema coma, and describes pharmacological treatment and supportive cares.

Thyroid Gland

The thyroid gland produces 2 related hormones, thyroxine (T4) and triiodothyronine (T3). Thyroxine is converted to T3 by the removal of 1 of T4’s 4 iodine atoms. Thyroxine and T3 play critical roles in cell differentiation during development and regulate thermogenic and metabolic homeostasis in adults. Thyroid hormones affect the function of virtually every organ system. The hypothalamic-pituitary-thyroid axis works to maintain peripheral free thyroid hormone levels in a narrow range through a negative feedback system (see Figure 1 and Table 1).

The hypothalamus directly secretes thyrotropin-releasing hormone (TRH) into the blood supply of the anterior pituitary via a capillary system. In response to TRH release, the anterior pituitary produces and releases thyroid-stimulating hormone (TSH). In response to TSH, the thyroid increases hormone (T3 and T4) synthesis and secretion. The thyroid hormones provide negative feedback to inhibit TRH and TSH. Thyroid-stimulating hormone plays a pivotal role in control of the hypothalamic-pituitary-thyroid axis, making it the key physiological marker of thyroid hormone action.

Thyrotoxicosis

Thyrotoxicosis is a state of thyroid hormone excess, which may be the result of hyperthyroidism or other causes. Primary hyperthyroidism is most often due to Graves disease (60%–80% of cases), but also may
be due to toxic multinodular goiter or toxic adenoma. Thyrotoxicosis without hyperthyroidism is rarer, as in the cases of subacute thyroiditis, silent thyroiditis, or infarction of adenoma. Iatrogenic causes of thyrotoxicosis include ingestion of excess thyroid hormone or thyroid destruction as a result of amiodarone use or radiation. Secondary hyperthyroidism resulting from pituitary adenoma is another cause of thyrotoxicosis. 1,2,5

Symptoms of thyrotoxicosis include hyperactivity, irritability, dysphoria, heat intolerance and sweating, palpitations, fatigue and weakness, weight loss in spite of increased appetite, diarrhea, polyuria, oligomenorrhea, and loss of libido. 1,2 Signs of thyrotoxicosis include tachycardia, atrial fibrillation (more common in older adults), tremor, goiter, warm and moist skin, muscle weakness, lid lag, and rarely gynecomastia. 1,5,7,8 Thyrotoxicosis may be treated with thioamides, iodides, and/or adrenergic blockers (see Table 2). 8

**Thyroid Storm**

Thyrotoxic crisis or thyroid storm is a rare but life-threatening exacerbation of thyrotoxicosis. The incidence of thyroid storm has been noted to be less than 10% of patients hospitalized for thyrotoxicosis. 9 Thyroid storm is more common in women than in men (10% vs 2%) and occurs most commonly between the ages of 20 and 49 years. 5 Anyone with untreated hyperthyroidism is at risk of thyrotoxic crisis, but a precipitating event such as stroke, infection, trauma, or diabetic ketoacidosis usually occurs (see Table 3). Thyrotoxic crisis may also be precipitated by surgery, particularly on the thyroid; radioiodine treatment in a patient with unmanaged hyperthyroidism; noncompliance with antithyroid medications; or alcohol abuse. 1,2 Individuals being treated with certain medications are also at higher risk of thyroid storm; these medications include nonsteroidal anti-inflammatory

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**Figure 1.** Hypothalamic-pituitary-thyroid gland axis feedback loops. T3 indicates triiodothyronine; T4, thyroxine; TRH, thyrotropin-releasing hormone; TSH, thyroid-stimulating hormone.

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**Table 1: Normal Thyroid Hormone Levels**

<table>
<thead>
<tr>
<th>Thyroid Function Test</th>
<th>Conventional Units</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSH</td>
<td>0.5–5.0 μIU/mL</td>
<td>0.5–5.0 mIU/L</td>
</tr>
<tr>
<td>T3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total serum</td>
<td>70–195 ng/dL</td>
<td>1.1–3.0 nmol/L</td>
</tr>
<tr>
<td>T4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free serum</td>
<td>0.9–2.4 ng/dL</td>
<td>12–31 pmol/L</td>
</tr>
<tr>
<td>Total serum</td>
<td>5–12 mcg/dL</td>
<td>64–155 nmol/L</td>
</tr>
</tbody>
</table>

Abbreviations: IU, international units; T3, triiodothyronine; T4, thyroxine; TSH, thyroid-stimulating hormone.
drugs, salicylates, tricyclic antidepressants, insulin, thiazide diuretics, amiodarone, chronic steroids, and fludrocortisone.

**Signs and Symptoms**
The critical exacerbation of hyperthyroidism, thyrotoxic crisis, may demonstrate the same symptoms and signs noted earlier plus tachycardia, fever, delirium, seizures, coma, and jaundice. Diagnosis relies primarily on clinical presentation; therefore, astute clinical assessments are vital. Patients may have sinus or supraventricular tachycardias or atrial fibrillation and also may exhibit signs of a high cardiac output state. High output can be related to preload increase as a result of renin-angiotensin-aldosterone system activation and a decreased afterload, which can be a result of vascular relaxation from thyroid hormone influence. The patient can exhibit both tachypnea and dyspnea, with an increased oxygen demand.

Alterations in the gastrointestinal (GI), renal, and hepatic systems are demonstrated through diarrhea and vomiting, increased glomerular filtration rate and excess proteinuria, and elevated lactate dehydrogenase, aspartate aminotransferase, and bilirubin levels.

Patients in thyroid storm may demonstrate hyperglycemia, even in the absence of diabetes, from increased glycogenolysis and inhibition of insulin release. In addition, these patients may have sinus or supraventricular tachycardias or atrial fibrillation and also may exhibit signs of a high cardiac output state. High output can be related to preload increase as a result of renin-angiotensin-aldosterone system activation and a decreased afterload, which can be a result of vascular relaxation from thyroid hormone influence. The patient can exhibit both tachypnea and dyspnea, with an increased oxygen demand.

### Table 2: Pharmacological Treatment for Thyroid Storm

<table>
<thead>
<tr>
<th>Medication</th>
<th>Class/Action</th>
<th>Function</th>
<th>Dose, Delivery</th>
<th>Adverse Effects, Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTU—first choice in life-threatening thyroid storm</td>
<td>Thioamide</td>
<td>Inhibits thyroperoxidase-mediated iodination of thyroglobulin to form T4 and T3 within thyroid gland, also blocks peripheral T4 to T3 conversion</td>
<td>600-mg loading, then 200–300 mg every 6 h PO/NG tube/PR</td>
<td>Rashes, arthralgias, fevers, benign transient leukopenia, hepatotoxicity</td>
</tr>
<tr>
<td>Potassium iodide saturated solution</td>
<td>Stable iodide</td>
<td>Blocks thyroid hormone release, decreases size and vascularity of gland</td>
<td>5 drops PO every 6 h (38-mg iodide per drop)</td>
<td>Hypersensitivity reactions, salivary gland swelling, metallic taste</td>
</tr>
<tr>
<td>Methimazole—could be used first, instead of PTU, if severe, but not life-threatening thyrotoxicosis</td>
<td>Thioamide</td>
<td>Inhibits thyroperoxidase-mediated iodination of thyroglobulin to form T4 and T3 within thyroid gland</td>
<td>20 mg PO every 6 h</td>
<td>Rashes, arthralgias, fevers, benign transient leukopenia, less hepatotoxicity than PTU</td>
</tr>
<tr>
<td>Propranolol</td>
<td>β-adrenergic blocker</td>
<td>Reduce symptoms and signs, such as tachycardia induced by increased adrenergic tone, inhibits conversion of T4 to T3</td>
<td>40-80 mg PO every 4 h, or 2 mg IV every 4 h</td>
<td>Nausea, vomiting, bradycardia</td>
</tr>
<tr>
<td>Dexamethasone or hydrocortisone</td>
<td>Glucocorticoid</td>
<td>Decrease peripheral conversion of T4 to T3, treat relative adrenal insufficiency, promote vasomotor stability, antipyretic</td>
<td>Dexamethasone: 2 mg every 6 h × 48 h, then taper</td>
<td>Hyperglycemia</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td></td>
<td></td>
<td>Hydrocortisone: 100 mg every 8 h</td>
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</tbody>
</table>

*Abbreviations: IV, intravenous; NG, nasogastric; PO, by mouth; PR, by rectum; PTU, propylthiouracil; T3, triiodothyronine; T4, thyroxine.*
have a higher incidence of increased insulin clearance and resistance, thereby exacerbating hyperglycemia. Lipolysis and ketogenesis can result in ketoacidosis and lactic acidosis.  

Patients can exhibit significant hematologic changes, such as moderate leukocytosis, even when an infection is not present, and increased red blood cell mass related to erythropoietin upregulation.  Hypercoagulation is a concern; in fact, 18% of thyroid deaths are attributed to thromboembolic complications. 

The TSH will be suppressed and thyroid hormones elevated to similar levels as in none-mergergent thyrotoxicosis. Although no difference exists in the hormone levels’ degree of deviation from normal, the patient’s response is magnified in the setting of an acute illness. One plausible explanation is that individuals with thyrotoxicosis have more adrenergic binding sites at baseline, so the catecholamine surge resulting from the precipitating event elicits an exaggerated response.

Because early recognition and appropriate management of thyroid storm are vital to limiting morbidity and mortality rates, attempts have been made to systematically analyze risk. Burch and Wartofsky created a point scale for the diagnosis of thyroid storm that considers level of thermoregulatory dysfunction, level of cardiovascular disturbance (degree of tachycardia, presence of atrial fibrillation, and/or congestive heart failure), level of GI-hepatic dysfunction, level of central nervous system disturbance, and presence of precipitants. Scores indicate whether thyroid storm is present, likely to occur, or unlikely to occur.

Mortality rate, as a result of cardiac failure, arrhythmia, shock, or multiple organ failure, is reported to vary widely (from 2% to 75%), even when the crisis is promptly identified and treated. The long half-life of T4 and T3 means that the crisis can persist for a considerable amount of time. The implication for older adults, among whom metabolism is already altered, is that they are at increased risk for crisis, particularly with dose changes of levothyroxine. Further complicating the ability to monitor for changes is that many elderly patients have blunted adrenergic symptoms related to concurrent treatment with β-blockers.

### Table 3: Precipitating Factors for Thyroid Storm

<table>
<thead>
<tr>
<th>Precipitating Factors</th>
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</thead>
<tbody>
<tr>
<td>Withdrawal of antithyroid medications</td>
</tr>
<tr>
<td>Severe infection</td>
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<tr>
<td>Diabetic ketoacidosis</td>
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<tr>
<td>Myocardial infarction and cerebrovascular accident</td>
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<tr>
<td>Cardiac failure</td>
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<tr>
<td>Surgery</td>
</tr>
<tr>
<td>Parturition</td>
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<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Radioiodine</td>
</tr>
<tr>
<td>Drug reaction</td>
</tr>
<tr>
<td>Iodinated contrast exposure</td>
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</tbody>
</table>

### Treatment

Treatment of thyrotoxic crisis or thyroid storm follows the same basic treatment as for non-critical thyrotoxicosis, but the medications are given in higher doses and more frequently, and supportive care is best provided in an intensive care setting. The patient requires several types of medications for adequate treatment.

- **An antithyroid medication to stop thyroid hormone synthesis.** The thioamide antithyroid medications are not available in intravenous form in the United States and therefore must be given via a nasogastric tube or rectally for comatose patients.
- **An iodide to block thyroid hormone release.** The iodide should not be given sooner than 1 hour after the thioamide to avoid increased thyroid hormone synthesis, not to augment gland storage, and to avoid exacerbation of the crisis. If the patient is allergic to iodine, lithium carbonate may be an acceptable alternative.
- **A β-blocker to ameliorate adrenergic symptoms.** β-blockers will address symptoms by decreasing heart rate and oxygen demand and reduce convulsion symptoms, psychotic behavior, agitation, and fever.
- **A glucocorticoid to block T4 to T3 conversion and address potential concomitant adrenal insufficiency.**
Fluids may be needed to maintain vascular stability, although patients with underlying cardiac disease should be monitored closely to ensure appropriate fluid resuscitation. Use of vasoactive medications also may be needed for a short period of time to achieve hemodynamic stability. If fever needs to be treated, acetaminophen is the recommended drug, as salicylates can inhibit thyroid hormone binding.

In some cases, plasmapheresis and therapeutic plasma exchange can be used if medication therapy is not adequate to reduce free thyroid hormone levels. The use of plasma and albumin with these therapies provides new binding sites for the circulating free hormones. However, the use of the therapies results in only transient reductions in T3 and T4 (for up to 36 hours), requiring continued need for definitive therapy. Peritoneal dialysis and oral cholestyramine resin also may provide temporary binding of hormone. In addition, any underlying disease that is exacerbating the crisis must be treated. If the underlying precipitating factor is not obvious, clinicians should search for an infection as a potential contributing factor.

Improvement can be dramatic, occurring within as little as 24 hours. Once a patient has been stabilized, with precipitating causes addressed, iodide therapy and glucocorticoids can be withdrawn. Treatment with β-blockers should continue until thyroid function test results have returned to normal. The thioamide should be titrated, so the patient maintains a euthyroid state. If initiated on propylthiouracil, the patient should be transitioned to methimazole in preparation for discharge, because it has a preferable dosing schedule and improved safety profile.

Whatever the cause for thyrotoxicosis, a definitive plan is needed to manage thyroid hormone levels and prevent repeat crisis. If the cause of thyrotoxic crisis was Graves disease, radioiodine ablation or thyroidectomy should be pursued to prevent a repeat crisis. Hyperthyroidism due to goiter also may be treated before surgical intervention to avoid another crisis during surgery.

Thyroid Storm in Pregnancy
Hyperthyroidism (caused by Graves disease in 85% of cases) occurs in 1 in 500 pregnancies. Not surprisingly, women with thyrotoxicosis who have limited prenatal care or medical or obstetric complications are at increased risk of thyroid storm. Signs and symptoms of thyroid storm in pregnant women are not different from those in nonpregnant adults, but could be mistaken for signs and symptoms of the normal hypermetabolic state of pregnancy. Management of thyroid storm in pregnancy involves the same components as for adults, but provision for fetal well-being adds to the complexity. Propylthiouracil and methimazole cross the placenta. Because of differences in teratogenicity and adverse effects, the use of propylthiouracil is recommended for the first trimester, and then the use of methimazole is recommended for the remaining period of pregnancy. Delivery of the fetus during thyroid storm is not recommended, unless the condition of the fetus calls for it. Once the patient has stabilized, management is continued with the smallest dose necessary of thioamide; radioactive iodine is contraindicated and thyroidectomy is avoided because of increased risk for spontaneous abortion or preterm delivery.

Hypothyroidism
Hypothyroidism is most commonly the result of the autoimmune disorder Hashimoto thyroiditis and also may be caused by thyroid gland destruction as a result of surgery, radioactive iodine, or external radiation of the neck for cancer. Additional causes of primary hypothyroidism are iodine deficiency, medications (iodine excess, lithium, antithyroid drugs, p-aminosalicylic acid, interferon alfa), dietary goitrogens (cassava root, cruciferous vegetables), and congenital defects. Secondary hypothyroidism is caused by pituitary disease, and tertiary hypothyroidism is caused by hypothalamic disorders.

Hypothyroidism causes decreased metabolism manifest in slowed heart rate, diminished oxygen consumption, and deposition of glycosaminoglycans in intracellular spaces (skin and muscle, in particular), producing myxedema in severe cases. Individuals with hypothyroidism may complain of fatigue, cold intolerance, hair loss, difficulty concentrating, constipation, weight gain despite poor appetite, dyspnea, hoarse voice, menorrhagia or oligomenorrhea, impaired hearing, and paresthesia. They may exhibit dry, coarse skin; puffy face, hands, and feet (myxedema); diffuse alopecia; bradycardia; peripheral edema; delayed tendon reflex relaxation; carpal tunnel syndrome; and serous cavity effusions.
Myxedema Coma
Myxedema coma is the end stage of untreated or inadequately treated hypothyroidism, and it, like thyroid storm, may be precipitated by acute events, such as cerebrovascular accident, myocardial infarction, infection, GI bleeding, or trauma (see Table 4). Excessive hydration, sedatives, narcotics, and potent diuretics also may be precipitating factors. Other medications and medication factors that increase the risk of myxedema coma include amiodarone, lithium, phenytoin, and lack of adherence to thyroid replacement therapy. The incidence rate is estimated to be as low as 0.22 per million people per year, with the most common presentation in hospitalized elderly women with a long history of hypothyroidism. Eighty percent of women affected by this condition are older than 60 years, although it can occur in younger patients and also in pregnant women. Cases frequently present in the winter months, leading to the belief that cold weather may lower the threshold for those who are at risk of myxedema coma.

Signs and Symptoms
A typical course can initiate as lethargy, with worsening mental status progressing to coma, then respiratory decompensation and hypothermia. Patients with myxedema coma present with mental status changes, such as disorientation, depression, poor memory, and/or hallucinations, sometimes associated with seizures, as well as other hypothyroid symptoms. Hypothermia may be significant (body temperature as low as 74°F). Cardiovascular symptoms can be significant, with increased risk of arrhythmias (bradycardia, varying types of blocks, prolonged QT intervals, and torsades de pointes), impaired cardiac contractility, and shock from decreased cardiac output and hypotension from low intravascular volumes. Cardiac tamponade can occur from an accumulation of mucopolysaccharide infiltrates in the pericardial sac.

Respiratory decompensation can require mechanical ventilatory support because of depressed respiratory drive from hypoaxia and diminished response to hypercapnia. The patient also needs to be monitored for edema of the tongue and vocal cords, resulting in airway obstruction. Respiratory recovery may be slow, resulting in the need for prolonged mechanical ventilatory support.

The major electrolyte abnormality can be hyponatremia from increased serum antidiuretic hormone and impaired diuresis caused by decreased water getting to the distal nephron. A decreased glomerular filtration rate can result, leading to an increase in total body water. The hyponatremia also affects the patient’s mental status. Patients with symptomatic hyponatremia have higher mortality rates than those without hyponatremia. The bladder can become atonic, with urinary retention. In addition, increased creatinine kinase levels can lead to rhabdomyolysis, increasing the risk of renal failure.

Although general hypothyroidism increases the risk of clotting, patients with myxedema coma are at increased risk of coagulopathy and bleeding, including reduced factors V, VII, VIII, IX, and X, as well as acquired von Willebrand syndrome. Coagulopathy is reversible with the administration of T4. Granulocytopenia can result in development of severe infections and a decrease in cell-mediated immune response. Patients can, therefore, experience disseminated intravascular clotting if they become septic.

Patients may experience decreased GI motility as a result of the mucopolysaccharide infiltration and edema of the gut, sometimes severe enough to result in paralytic ileus. Gastrointestinal bleeding also may occur because of coagulopathy.

Treatment
Management of myxedema coma involves several approaches: thyroid hormone replacement, supportive care, and treatment of concomitant conditions. Optimal hormone replacement treatment is uncertain at this time, with few well-controlled trials because of the infrequency of cases. Hormone

Table 4: Precipitating Factors of Myxedema Coma

<table>
<thead>
<tr>
<th>Precipitating Factors of Myxedema Coma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
</tr>
<tr>
<td>Exposure to cold</td>
</tr>
<tr>
<td>Heart failure</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Surgical procedures</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
</tr>
<tr>
<td>Gastrointestinal bleeding</td>
</tr>
</tbody>
</table>

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replacement may be with T4 or T3 or both. Thyroxine and T3 in combination are advocated by some because of the impairment in T4 to T3 conversion seen in myxedema coma (see Table 5). Overly aggressive replacement of T4 can be a risk, as myocardial infarction can be a consequence.

The patient may require intravenous fluids for vascular volume—0.9% sodium chloride or 5% dextrose in 0.9% sodium chloride or 10% dextrose in 0.9% sodium chloride if the patient requires glucose. Sodium replacement may be needed for severe hyponatremia. Small amounts of hypertonic saline (50–100 mL of 3% sodium chloride) can be administered with a goal to increase the sodium level by 2 to 4 mmol/L, which should be followed by furosemide diuretic to promote diuresis. Sodium levels should not be raised too rapidly, as rapid correction of chronic hyponatremia might put patients at risk for central pontine myelinolysis. Close patient monitoring is required with replacement of fluids and sodium in patients with cardiac disease.

Hyperthermia will resolve with administration of T3 and T4, although a warm ambient temperature and warm blankets also can be used. Aggressive rewarming should be avoided as significant vasodilation can occur as the myxedema coma resolves. Mortality rate with myxedema coma is highest in those patients who exhibit severe hypothermia and low blood pressure. Mortality rates have decreased significantly from 60% to 70% to 20% to 25% over the years, which is likely a result of better recognition of the diagnosis and better treatment strategies. 10,16

**Case Studies**

**Myxedema Coma**

Marietta is an elderly, obese woman with medical history significant for type 2 diabetes, who underwent open reduction and internal fixation of a displaced left humerus fracture. Marietta was hemodynamically stable throughout the surgery, but upon completion and reversal of sedation, she remained apneic and unresponsive. After treatment with naloxone, she responded with shallow breathing and intermittent apnea, prompting resumption of ventilatory support. Although hypothermia was being reversed with a forced air-warming device, Marietta’s laboratory test results showed normal electrolyte levels, absent ketones, and normal blood gas. At this time, her blood pressure was down to 75/50 mm Hg, prompting initiation of dopamine infusion. Her blood pressure improved and 12-lead electrocardiogram failed to reveal any ischemic changes. Thyroid function profile revealed T3, 0.30 ng/mL; T4, 1.25 ng/dL; free T4, 0.85 ng/dL; and TSH, 180 mU/L.

Marietta was treated with thyroid hormone replacement and ongoing supportive care. Within 6 hours, she was exhibiting regular spontaneous respiratory effort and responding to commands. Her blood pressure improved,

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**Table 5: Pharmacological Treatment of Myxedema Coma**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Class/Action</th>
<th>Function</th>
<th>Dose, Delivery</th>
<th>Adverse Effects, Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levothyroxine (T4)</td>
<td>Synthetic thyroxine</td>
<td>Replacement</td>
<td>200-500 mcg IV/NG tube for loading dose, then 50 mcg IV daily</td>
<td>Potential to precipitate Myocardial infarction in high doses</td>
</tr>
<tr>
<td>Liothyronine (T3)</td>
<td>Synthetic triiodothyronine</td>
<td>Replacement</td>
<td>10-25 mcg IV/NG tube every 8-12 h</td>
<td>Cardiac monitoring</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Glucocorticoid</td>
<td>Patient may have hypopituitarism or polyglandular failure</td>
<td>50–100 mg IV every 6–8 h × 48 h or until adrenal suppression ruled out, then taper</td>
<td>Arrhythmias, cardiac monitoring</td>
</tr>
</tbody>
</table>

Abbreviations: IV, intravenous; NG, nasogastric; T3, triiodothyronine; T4, thyroxine.
and the dopamine was weaned off. She was able to be discharged to a rehabilitation facility within 5 days.

1. Marietta’s hypothyroidism was undiagnosed. What perioperative factors, coupled with her hypothyroidism, put her at risk of respiratory and cardiovascular dysfunction?
2. What is the primary pharmacological treatment in myxedema coma?

Thyroid Storm

Thea is a 55-year-old woman with a history of Graves disease. She had been taking methimazole 5 mg daily but stopped taking it sometime after her last endocrinologist visit 2 years ago.

She came to the emergency department with complaints of palpitations, restlessness, nausea, diarrhea, and heat intolerance. Her husband said that she seemed confused and her skin was hot. He was concerned about another infection. He reported that she had knee surgery 2 weeks prior and had been taking antibiotic drugs for surgical wound infection.

Her vital signs were as follows: oral temperature, 103°F; pulse, 130/min; respiratory rate, 20/min; blood pressure 90/40 mm Hg; and O₂ saturation, 95%. Physical examination was remarkable for exophthalmos and enlarged thyroid. She was admitted to the medical intensive care unit. Continuous electrocardiogram monitoring showed atrial fibrillation. A central catheter was placed, and central venous pressure monitoring was begun, as was fluid resuscitation for surgery. By this time, she also had a nasogastric tube and a Foley catheter.

1. What data indicate that Thea might be in thyroid storm?
2. What laboratory tests would be needed and what results would support the diagnosis of thyroid storm?
3. What will be the 4 main pharmacological interventions?
4. What supportive care and nursing assessments need to be incorporated into the care plan?
5. What likely precipitated the thyroid storm?

Conclusion

Thyroid storm and myxedema coma are endocrine emergencies that occur infrequently but can carry a high risk of mortality. These patients require admission to the intensive care unit because they need frequent monitoring and aggressive intervention. Critical care nurses can play a key role in early recognition of signs and symptoms of thyroid storm and myxedema coma, allowing for prompt intervention that will limit the risk of morbidity and mortality.

REFERENCES