Managing Patients With Acute Thyrotoxicosis

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Acute thyrotoxicosis is a systemic syndrome that occurs as a result of excess production and release of the thyroid hormones triiodothyronine and thyroxine. The syndrome is characterized by a hypermetabolic state in which the tissues respond to an excess of circulating thyroid hormones by markedly increasing cellular function. Under normal circumstances, thyroid hormones control metabolic processes in body tissues by increasing the rate of protein, fat, and glucose metabolism. If left untreated, the sustained hypermetabolic state that occurs in acute thyrotoxicosis can lead to death.

Prompt recognition of thyrotoxicosis and aggressive treatment of the associated manifestations are essential. Critical care nurses who have an understanding of this life-threatening condition can quickly intervene to improve patients’ outcomes. In this article, I describe thyroid physiology, discuss the clinical manifestations and treatment of acute thyrotoxicosis, and present a case study of a patient with thyrotoxicosis who was admitted to the critical care unit because of acute congestive heart failure.

THYROID PHYSIOLOGY

The thyroid gland lies inferior to the thyroid cartilage and is composed of 2 lobes that lie on either side of the trachea (Figure 1). The lobes are joined by a small band of tissue called the isthmus. Normally not visible on inspection, the thyroid gland can be palpated during swallowing when it is upwardly displaced. Thyroid tissue is highly vascular and is composed of follicular cells that synthesize and secrete 2 major hormones: thyroxine (T₄) and triiodothyronine (T₃). These hormones are sometimes collectively referred to as thyroid hormone. T₃ and T₄ affect all cells within the body except those in the brain, spleen, testes, and uterus.

Secretion of Thyroid Hormones

T₃ and T₄ are regulated through a feedback loop that involves the hypothalamus, the anterior part of the pituitary gland, and the thyroid gland (Figure 2). The initiating hormone, thyrotropin-releasing hormone, is synthesized and stored in the hypothalamus. Thyrotropin-releasing hormone circulates to the anterior part of the pituitary gland, where it stimulates the release of thyroid-stimulating hormone (TSH). TSH binds to receptor sites located on the thyroid gland, which produces, secretes, and stores T₃ and T₄. Stimulation by TSH results in an immediate release of stored thyroid hormones into the circulation. Serum levels of T₃ and T₄ increase as T₃ binds to cell receptors. Concurrent effects include an increase in the uptake and oxidation of iodine, an increase in the synthesis of T₃ and T₄, and an increase in the synthesis and secretion of prostaglandins.

Under normal circumstances, the feedback loop ensures that circulating levels of thyroid hormones are appropriate for metabolic needs. Disruption of the feedback loop can result in a life-threatening excess or deficit of T₃ and T₄.

Mechanism of Action

The thyroid gland normally produces 90% T₄ and 10% T₃. In the body tissues, T₄ is converted to T₃, which has the greatest systemic
metabolic effects (Table 1). Thyroid hormones are involved in glucose oxidation, which increases metabolic rate, consumption of oxygen by tissues, and production of body heat. Upon binding to its receptor, T₃ initiates a series of biochemical and physiological responses that affect all systems, including the cardiovascular, respiratory, gastrointestinal, and neuromuscular.

At physiological levels, T₃ favors anabolism and growth in general, as well as lipogenesis and protein synthesis. At higher levels, the effects are catabolic and lead to increased production of body heat, increased generation of adenosine triphosphate, and depletion of cellular forms of energy. Because of these diverse effects, critical care nurses must simultaneously balance an array of pathophysiological responses that occur during thyroid emergencies.

**THYROTOXICOSIS**

Thyrotoxicosis is an all-inclusive term used to describe severe hypermetabolic states induced by thyroid hormones. The term hyperthyroidism encompasses a heterogeneous group of disorders, all characterized by elevated levels of thyroid hormones in the blood. Thyroid storm, a relatively rare condition, is a life-threatening decompensated state of thyrotoxicosis that can cause death unless recognized early and treated aggressively. Thyroid crisis or storm is precipitated when the metabolic, thermoregulatory, and cardiovascular mechanisms that compensate for thyrotoxicosis fail (Table 2).

Clinical differentiation between thyrotoxicosis and thyroid storm can be difficult. Classical clinical findings of thyroid storm include sinus or supraventricular tachycardia with or without congestive heart failure and the presence of hyperpyrexia (core temperature >40°C); confusion and delirium often occur and may progress to coma.

Although thyroid storm is rare, early diagnosis and vigorous therapy can prevent a fatal outcome. Mortality rates of hospitalized patients in thyroid storm are 20% to 50%. Critical care nurses must recognize the hypermetabolic state and ensure that
aggressive and supportive treatment is not delayed.

Etiology
Thyrotoxicosis may be due to true hyperthyroidism, in which synthesis and release of hormones from the thyroid gland are increased. Graves’ disease is the most common cause of true hyperthyroidism, accounting for 60% to 90% of all cases. Other causes include a hyperfunctioning thyroid nodule, excessive ingestion of exogenous thyroid hormones, pituitary tumors, pituitary gland abnormalities, and thyroiditis.8-11 A thyrotoxic crisis or thyroid storm may develop precipitously in patients with hyperthyroidism who have had surgery, have experienced trauma, have an infection, are in the postpartum period, or have had antithyroid medication withdrawn.1,2,7

The precipitating event should be determined after the patient’s condition has been stabilized and the hypermetabolic state has been arrested. Critical care nurses must use their assessment skills and clinical judgment to ensure that treatment is not postponed while the precipitating event is determined. Moreover, treatment should not be delayed while waiting for the results of thyroid function tests.

Clinical Manifestations
Patients with thyrotoxicosis have a myriad of clinical features related to increased cellular functioning. Easily recognized characteristics include marked weight loss (>15% of body mass index; index calculated as weight in kilograms divided by the square of

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**Table 1** Effects of thyroxine and triiodothyronine

<table>
<thead>
<tr>
<th>System</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Facilitates cardiac contraction and cardiac output</td>
</tr>
<tr>
<td></td>
<td>With increased secretion, tachycardia and increased velocity and force of contractility</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Increases the rate of cellular respiration and the rate of protein and fatty acid degradation in many tissues, thereby affecting production of carbon dioxide</td>
</tr>
<tr>
<td>Nervous</td>
<td>Enhances the action of catecholamines on nervous tissue</td>
</tr>
<tr>
<td></td>
<td>Promotes the normal development of the nervous system in the fetus and protects the unborn child from cretinism, a form of mental retardation</td>
</tr>
<tr>
<td>Digestive/metabolic</td>
<td>Increases secretion of digestive juices and promotes motility</td>
</tr>
<tr>
<td></td>
<td>Enhances the ability to absorb glucose</td>
</tr>
<tr>
<td></td>
<td>Increases metabolic rate by stimulating enzymes concerned with glucose oxidation, thereby increasing oxygen consumption and body heat production</td>
</tr>
<tr>
<td></td>
<td>Enhances the rate of glucose uptake and promotes protein synthesis</td>
</tr>
<tr>
<td></td>
<td>Lowers blood cholesterol by increasing the rate at which cholesterol is excreted and degraded</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Encourages bone growth until adulthood is reached</td>
</tr>
<tr>
<td></td>
<td>Stimulates the production of proteins needed for muscle contraction and relaxation</td>
</tr>
<tr>
<td>Integumentary</td>
<td>Controls sweat glands and oil glands, ensuring that skin is moist and supple.</td>
</tr>
</tbody>
</table>
height in meters), anxious demeanor, excitable state, fixed stare, and fine tremors of the hands and tongue. Exophthalmos and goiter are commonly associated with hyperthyroidism but are not always present in acute thyrotoxicosis.

Understanding major organ responses to excessive circulating levels of thyroid hormones and recognizing a decompensating hypermetabolic state will help in developing an appropriate plan of care. Aggressive management of acute thyrotoxicosis, including continuous monitoring, can prevent deterioration to thyroid storm.

Cardiovascular Manifestations. Cardiovascular manifestations of thyrotoxicosis are due to the direct effect of thyroxine on the myocardium, alterations in systemic vascular resistance, and increased sensitivity to endogenous catecholamines. Thyroid hormones increase calcium and sodium transport in cardiac muscle, causing a lowered atrial excitation threshold, an increase in sinoatrial node firing, and a shortened refractory period. These changes are compounded by a decrease in systemic vascular resistance and enhanced sympathetic activity.

Tachycardia greater than 140 beats per minute is a common finding, as is atrial fibrillation and various degrees of ventricular dysfunction. The hemodynamic profile in acute thyrotoxicosis includes increased stroke volume, increased oxygen consumption, increased left ventricular stroke work index, and a reduction in peripheral vascular resistance. A patient with failing compensatory mechanisms or heart disease is at risk for high-output heart failure, the clinical hallmark of thyrocardiac disease.

In high-output heart failure, metabolic acidosis is imminent because of the body’s demand for oxygen, so cardiac output is greatly increased. However, despite the elevated cardiac output, the body’s metabolic needs are still not met. Patients in high-output heart failure often have a widened pulse pressure, with elevated systolic pressure. If high-output heart failure is allowed to progress untreated, moderate to severe pulmonary edema develops, indicating marked cardiac and respiratory decompensation.

Respiratory Manifestations. In hypermetabolic states, the respiratory system is affected because of the need for greater oxygen supply and utilization. As a result, an increase in carbon dioxide production also occurs. Tachypnea and dyspnea on exertion are common abnormalities that occur because of a decrease in cardiac contractile reserve in patients with thyrotoxicosis. Weakness of the respiratory and skeletal muscles also occurs and places patients at risk for respiratory failure due to muscle fatigue. Consequently, critical care nurses must be diligent in assessing patients for indications of a decompensating respiratory state. Other abnormalities that contribute to respiratory compromise include decreased lung compliance, an engorged pulmonary capillary bed, and high-output ventricular failure.

Table 2: Clinical features of thyrotoxicosis and thyroid storm

<table>
<thead>
<tr>
<th>System</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Palpitations, Shortness of breath, Tachycardia, Systolic hypertension, Congestive heart failure, Arrhythmias (new onset)*</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Weight loss, Hyperdefecation, Diarrhea, Jaundice*</td>
</tr>
<tr>
<td>Nervous</td>
<td>Restlessness, Emotional lability, Short attention span, Tremor, Hyperreflexia, Apathy*, Agitation*, Delirium*, Coma*</td>
</tr>
<tr>
<td>Thermoregulatory</td>
<td>Warm, moist skin, Heat intolerance, Hyperpyrexia (body temperature &gt; 40°C)*</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Goiter, Exophthalmos</td>
</tr>
</tbody>
</table>

*Cardinal features of thyroid storm.
Patients in high-output heart failure are also at risk for acute respiratory failure because of the accumulation of fluid in the pulmonary capillary beds and alveoli. Pulmonary congestion further decreases gas exchange and impairs ventricular functioning. Acute pulmonary edema, a common sequela of untreated heart failure, must be recognized early and managed aggressively.

**Gastrointestinal Manifestations.** Gastrointestinal manifestations in thyrotoxicosis are due to accelerated intestinal transport and decreased absorption. Nausea, vomiting, and diarrhea are typical findings and may mimic those of an acute abdominal emergency. Abdominal pain may be the chief symptom in patients with acute thyrotoxicosis. A history of weight loss despite hyperphagia is common because of the need for increased energy requirements. In addition, elevations in transaminase levels and hepatotoxic effects may result in clinical jaundice.

**Neuromuscular Manifestations.** Alterations in the levels of thyroid hormones have a profound effect on the neuromuscular system. Sympathomimetic effects result in changes in mental status that range from rapid mentalation, agitation, restlessness, and confusion to delirium, obtundation, and coma if the effects are allowed to progress untreated. Electroencephalograms reveal an increase in alpha-wave activity that can be attributed to increased metabolic activity. Hyperreactivity may occur in response to physical and emotional stimuli. A fine motor tremor is present when the hands are outstretched, and patients have fatigue and muscle weakness. Deep tendon reflexes in patients with thyrotoxicosis are brisk, with a shortened relaxation phase.

Myopathy occurs in more than 50% of patients with thyrotoxicosis and most commonly affects the proximal muscle groups of the shoulders and pelvic girdle. Shoulder weakness, difficulty in going from a sitting to a standing position, and generalized weakness and fatigability are common.

**Integumentary Manifestations.** The integument plays a fundamental role in thermoregulation. The skin contains only α-adrenergic receptors, which are regulated by the sympathetic nervous system. Heat loss is regulated by varying blood flow through the skin in conjunction with the evaporative loss of heat through sweating. In thyrotoxicosis, rapid metabolism causes increased dermal blood flow.

Patients with thyrotoxicosis are heat intolerant and have warm moist skin that is silky and smooth to touch. Increased diaphoresis and hot flushes are common because of the increased blood flow associated with hypermetabolism. Alopecia and nail friability with separation of the nail from the proximal part of the nail bed are also common findings.

**TREATMENT**

Recognition and treatment of acute thyrotoxicosis are based on clinical evaluation and judgment. Thyrotoxicosis is an acute, emergent condition, and patients must be treated aggressively. Patients have an endogenous overproduction of thyroid hormone that must be arrested. If allowed to go untreated, the physiological responses may rapidly progress to coma and to death due to cardiac failure. Strong consideration must be given to initiating hemodynamic monitoring in patients with moderate to severe congestive heart failure. Even though data on the levels of thyroid hormones are usually not immediately available, therapy must not be delayed while the results of laboratory tests are pending. Moreover, laboratory results provide corroborative information and ultimately confirm the clinical assessment findings once the patient’s condition is stabilized.

Specific therapy must be aimed at supporting vital functions, blocking hormone synthesis and release, inhibiting the peripheral conversion of T₄ to T₃, and ameliorating the effects of the hypermetabolic state. Supportive therapy includes ensuring adequate ventilation and oxygenation, suppressing the heightened response to catecholamines, monitoring for and controlling cardiac dysrhythmias, and administering intravenous fluids to replace sensible and insensible losses of fluid. If vomiting and diarrhea occur, careful attention to electrolyte replacement is necessary. Intravenous fluids should include glucose if hypoglycemia occurs and vitamins, because patients are catabolic. Hyperglycemia may also occur because of the stimulatory effect of T₃ on gluconeogenesis and glycogenolysis. Therefore, glucose monitoring should be included in the plan of care.

**Medications**

Critical care nurses must be knowledgeable about the medications that are necessary to meet the goals of therapy for thyrotoxicosis. This knowledge includes understanding how the various drugs work, how they are metabolized, the individual dosage...
parameters, precautions for use, and the nursing implications (Table 3).

**Inhibition of Thyroid Hormone Biosynthesis.** Synthesis of thyroid hormones can be inhibited by the administration of antithyroid thioamide drugs. Specifically, propylthiouracil and methimazole (Tapazole) inhibit biosynthesis of the hormones. However, these drugs do not block the release of previously synthesized thyroid hormones and must be given with iodide preparations. Propylthiouracil is especially therapeutic because in large doses it blocks the conversion of T₄ to T₃ in peripheral tissues, resulting in a more rapid reduction of levels of circulating thyroid hormones. Methimazole has a slower rate of action but is more potent than is propylthiouracil. Both drugs can only be given orally or via a gastric tube. Because these 2 drugs do not block the release of thyroid hormones already stored in the thyroid gland, administration of medications that block the release of the hormones into the circulation, such as iodide preparations, and glucocorticoids are necessary.

Glucocorticoids such as dexamethasone may be given when a patient is thought to have a relative deficiency of steroids. An increased stress response, which often occurs in acute thyrotoxicosis, depletes endogenous cortisol stores even in patients without adrenal insufficiency. Glucocorticoids also inhibit conversion of T₄ to T₃ in peripheral tissues and have an additive effect when given with propylthiouracil.

**Blockade of Thyroid Hormones.** Because the amount of hormones stored in the thyroid gland is generally great enough to last several weeks, release of the hormones must be suppressed by administering potassium iodide. Iodide preparations are rapid acting and have a short duration. They maintain and increase the levels of protein-bound thyroid hormones, thereby decreasing the levels of the free, active forms. Iodide preparations should not be given sooner than 1 hour after the administration of antithyroid drugs because if given too soon, the iodide may be used by the body to synthesize more T₄, thus paradoxically increasing the toxic levels of T₃.

**Table 3** Drugs commonly used in thyrotoxicosis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose/frequency</th>
<th>Reason given</th>
<th>Nursing interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propylthiouracil</td>
<td>1200-1500 mg/d, given in 200- to 250-mg increments every 4 hours orally or via gastric tube</td>
<td>Blocks new synthesis of thyroid hormones</td>
<td>Monitor for bleeding; platelet count may decrease.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inhibits conversion of thyroxine to triiodothyronine</td>
<td></td>
</tr>
<tr>
<td>Methimazole</td>
<td>120 mg given in 20-mg increments every 4 hours orally or via gastric tube</td>
<td>Blocks new synthesis of thyroid hormones</td>
<td>Monitor for bleeding; platelet count may decrease.</td>
</tr>
<tr>
<td>Saturated solution of potassium iodide</td>
<td>8 drops every 6 hours orally or via gastric tube</td>
<td>Prevents release of stored thyroid hormones</td>
<td>Give a minimum of 1 hour after propylthiouracil or methimazole.</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>300 mg intravenously</td>
<td>Adrenal reserves of endogenous cortisol stores may be exceeded during thyrotoxic crisis</td>
<td>Monitor serum levels of glucose and electrolytes. Watch for signs of fluid overload and infection.</td>
</tr>
<tr>
<td>Propranolol</td>
<td>60-120 mg every 6 hours for crisis</td>
<td>Blunts enhanced sympathetic activity</td>
<td>Provide continuous cardiac monitoring and frequent blood pressure monitoring. Do not use in patients with chronic lung disease or hypotension.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ameliorates the manifestations of excess levels of thyroxine and triiodothyronine</td>
<td></td>
</tr>
</tbody>
</table>
state.\textsuperscript{1,7,9-11} Usually, Lugol solution or a saturated solution of potassium iodide is given orally. Iopodate (Oragrafin) and iopanoic acid (Telepaque), radiographic contrast materials, have also been used to block release of thyroid hormones.

Lithium carbonate, a more toxic inhibitor of thyroid hormones has been used in patients who are allergic to iodide. Lithium carbonate must also be given orally or via a gastric tube.

**Antagonism of Peripheral Effects of Thyroid Hormones.** Although plasma catecholamine levels are normal in thyrotoxicosis, increased adrenergic activity is a prominent feature, and patients have a heightened sympathetic response.\textsuperscript{7,9-11} As a result, patients are anxious, tachycardic, and diaphoretic and have tremors. Patients may also report heat intolerance and have tachypnea. Critical care nurses must do frequent assessments and be alert for alterations in cardiac rhythm and for indications of respiratory decompensation due to muscle fatigue and acid-base imbalance.

β-Adrenergic blockade is the treatment of choice for reducing adrenergic-like signs and symptoms in patients with thyrotoxicosis. Propanolol is the drug of choice to counteract the peripheral effects of increased levels of thyroid hormones because it antagonizes the effects of catecholamines that act on β-adrenergic receptors. Propanolol is most effective in reducing the cardiovascular manifestations of thyrotoxicosis. Propanolol and similar β-adrenergic blockers (ie, metoprolol and acebutolol) are metabolized by the liver. Because hepatic blood flow is increased in thyrotoxicosis, higher doses may be required to achieve desired results. Propanolol should not be used in patients with bronchospastic pulmonary disease and/or a history of congestive heart failure.\textsuperscript{7,9} Agents that deplete catecholamines such as reserpine have been used when propanolol alone is not effective. Guanethidine has also been used; however, its delayed onset of action makes it less useful than other medications in the critical care setting.\textsuperscript{1,7}

**Treatment of High-Output Heart Failure.** Acute high-output heart failure is primarily due to impaired myocardial contractility and is aggravatated by atrial dysrhythmias. Conventional measures to treat heart failure include use of antiarrhythmic agents, digoxin, and diuretics.\textsuperscript{18} Definitive therapy for heart failure induced by thyrotoxicosis is reestablishment of euthyroidism or a normal thyroid state. Because the metabolism and clearance of medications can be markedly increased during the hypermetabolic state, increased dosages may be necessary to achieve desired results.

**Diagnostic Studies**

Once the patient’s condition is stabilized, the precipitating or underlying cause of thyrotoxicosis must be determined. In addition to measurement of thyroid hormone levels, diagnostic evaluation may include general laboratory tests, radionuclide imaging, scintigraphy, and thyroid antibody tests.\textsuperscript{10} Because the thyroid gland normally takes up iodine to make thyroid hormones, the test is a useful measurement to determine how much iodine is captured by the gland. The test is often essential for treatment decisions once a patient’s condition is stabilized.

**Conclusions**

Acute thyrotoxicosis is a rare disorder that must be recognized promptly and treated aggressively. Providing optimal nursing care for patients with thyrotoxicosis is based on a solid foundation of knowledge of changes that may occur as a result of increased levels of circulating thyroid hormones. Caring for these patients can be a challenge for critical care nurses, who must continuously...
CASE STUDY

A 29-year-old Spanish-speaking gravida 1, para 1 woman was transferred to the emergency department from a nearby hospital 24 hours after delivery of her infant for evaluation and possible admission to the critical care unit. At the outlying hospital, she had elevated blood pressure (systolic pressure, 160–170 mm Hg) and tachycardia (heart rate, 120/min). She had a history of thyroid disease of unknown cause. One year previously, treatment had been started, but she self-discontinued it after a few months because she felt that she did not need to take medicine. She did not know what medications she had been taking.

On arrival at the emergency department, she was alert and oriented and anxious and hyperactive, as indicated by her constant movement in the bed. Cardiac monitoring indicated sinus tachycardia (heart rate, 130/min). Blood pressure was 160/70 mm Hg; respirations were 38/min and shallow, with crackles the bases of both lungs. Body temperature, measured orally, was 37.4°C. She was transferred from the emergency department to the critical care unit for observation.

During the admission assessment, the critical care nurse noted that the patient had exophthalmos and a slightly enlarged thyroid with no palpable nodules. The patient said that she did not have shortness of breath, chest pain, or palpitations. She was tachypneic (respirations, 32/min) with crackles throughout all lung fields; heart rate was 130/min with bounding pulses. The patient’s abdomen was postgravid and nontender. Deep tendon reflexes were 3+ bilaterally.

She said she felt very warm and refused to be covered with a sheet. She continued to be extremely anxious and hypervigilant.

Laboratory studies were ordered, including a thyroid panel. A complete blood cell count revealed that the patient was anemic (hematocrit, 0.26; hemoglobin, 80 g/L). A blood specimen for typing and crossmatching was sent to the laboratory, and the patient was started on oxygen by nasal cannula at 4 L/min. Intravenous fluid therapy was initiated with 10% dextrose and isotonic sodium chloride solution at a rate of 100 mL/h.

Approximately 45 minutes after arrival in the critical care unit, the patient became confused and restless. Her skin was pale, cool to touch, and diaphoretic. The cardiac monitor showed sinus tachycardia (heart rate, 150/min). Her blood pressure was 160/50 mm Hg, and respirations were 42/min and shallow, with crackles throughout both lung fields, audible without a stethoscope.

About 5 minutes into this critical incident, the patient’s blood pressure plummeted to 90/40 mm Hg, and her body temperature, measured in the axilla, increased to 38.5°C. Arterial blood gas analysis revealed acute respiratory failure, as indicated by respiratory acidosis (pH, 7.30; Pco2, 58 mm Hg; bicarbonate, 20 mmol/L) and a PaO2 of 50 mm Hg. The patient was intubated, and mechanical ventilation was started. An outpouring of pink frothy sputum when the endotracheal tube was inserted indicated acute pulmonary edema, requiring diligent suctioning.

On the basis of the pulmonary edema, hypotension, and tachycardia, the patient was judged to be in decompenated high-output heart failure. It was determined that the patient’s heart failure was due to her untreated thyrotoxicosis, which was exacerbated by her anemic state.

An oral gastric tube was inserted for gastric decompression and administration of medications. The patient was given 80 mg of furosemide and 10 mg of morphine sulfate intravenously to decrease preload and dilate the pulmonary bed. A 500-mg loading dose of propylthiouracil was administered via the oral gastric tube, and a dose of 250 mg was given every 4 hours for a maximum loading dose of 1500 mg over the next 24 hours.

One hour later, the patient was given 450 mg of saturated solution of potassium iodide and 1 g of succinylate via the oral gastric tube. The patient was given 40 mg of propranolol intravenously and then was given 2 mg of hydrocortisone intravenously every 6 hours. Propranolol was continued on an as-needed basis for heart rate greater than 100/min.

Laboratory reports obtained after admission to the critical care unit confirmed thyrotoxicosis: a serum level of free T4 of 86 pmol/L (6.67 ng/dL; reference range, 17-49 pmol/L [1.3-3.8 ng/dL]) and a suppressed serum level of TSH; reference range, <10 mIU/L. The patient’s blood pressure and heart rate remained stable for the next 1 to 2 days at 130/84 mm Hg and 90/min, respectively. The pulmonary edema subsided, and she continued to be afebrile.

Within 24 hours, the patient was successfully weaned off mechanical ventilation and extubated. The next day, she was transferred to the progressive care unit for further evaluation of her thyroid condition and to receive education about hyperthyroidism.

References