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A Devastating Storm

Eve Merrill, MD
Thomas Jefferson University Hospitals, Eve.Merrill@jefferson.edu

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Case Report

A 26-year-old female with no significant medical history presented with palpitations and shortness of breath. Two weeks prior, she experienced rhinorrhea and congestion.Vital signs on admission were temperature 96.6°F, heart rate 252 beats per minute, blood pressure 127/74 mmHg, respiratory rate 24 breaths per minute, oxygen saturation 98% on room air. On exam, the patient was tachycardic and had a large, homogenous thyroid without any palpable nodules. The rest of her physical exam was unremarkable.

Laboratory data revealed total bilirubin 4.4 mg/dL, aspartate aminotransferase 245 units/L, alanine aminotransferase 263 units/L, thyroid stimulating hormone (TSH) 0.02 mIU/L, free thyroxine (T4) 5.5 ng/dL, and free triiodothyronine (T3) 21.4 pg/mL. Electrocardiogram revealed supraventricular tachycardia. Computed tomography angiogram of the chest revealed a grossly enlarged thyroid, a left lower lobe infiltrate, and no evidence of pulmonary embolism. The patient was cardioverted to sinus rhythm and was started on a continuous infusion of diltiazem. She then became hypotensive and tachypneic and was subsequently intubated. Thionamide medications, including methimazole and propylthiouracil, for the patient's hyperthyroid state were initially held due to acute liver failure.

On the second day of admission, the patient developed rapid atrial fibrillation and was started on an esmolol infusion. Shortly thereafter, she became bradycardic and had a cardiac arrest with successful resuscitation. She then had a transvenous pacer placed as her asystolic event was attributed to a long conversion pause. Echocardiogram showed an ejection fraction of 10% with severe global systolic dysfunction, likely representing a stress-induced cardiomyopathy compounded by ischemia from the cardiac arrest.

In light of her rising liver function tests (transaminitis greater than 1000 units/L), the patient was started on methimazole, potassium iodine, and high-dose hydrocortisone for her severe hyperthyroid state. Ultrasound revealed an enlarged, heterogeneous thyroid with increased vascularity and serum studies showed anti-TSH receptor antibodies. Once her thyroid levels and liver function improved, propylthiouracil was used instead of methimazole. The patient also developed acute kidney injury requiring continuous venovenous hemodialysis. As she continued to clinically deteriorate, she was started on Extracorporeal Membrane Oxygenation (ECMO). The plan was to ultimately have the patient undergo a thyroidectomy once she became hemodynamically stable. Unfortunately, the patient had uncontrollable bleeding from her ECMO graft site and passed away on hospital day six.

Discussion

Thyroid storm is a state of severe hyperthyroid crisis (thyrotoxicosis) that causes organ dysfunction. Excess thyroid hormone produces a massive sympathetic (adrenergic) response and can damage the heart, liver, lungs, and central nervous system. Interestingly, the severity of thyroid dysfunction in thyroid storm does not correlate with thyroid levels. Thus, one cannot use thyroid levels alone to distinguish between hyperthyroidism and thyroid storm. Instead, there is scoring system developed by Burch and Wartofsky in 1993 to help establish a diagnosis of thyroid storm. Diagnostic criteria includes a point scale for varying degrees of cardiovascular dysfunction (including tachycardia and atrial fibrillation), heart failure, thermoregulatory dysfunction, central nervous system dysfunction, gastrointestinal-hepatic dysfunction, and a precipitant history event. A score below 25 makes thyroid storm unlikely, while scores between twenty-five and forty-four supports the diagnosis and a score of 45 of more is highly suggestive. Our patient had a score of 85.

Thyroid storm usually develops after an inciting factor in a patient with undertreated or undiagnosed hyperthyroidism. Precipitating stressors include infection, surgery, trauma, and a cardiovascular event. Other possible triggers include discontinuation of hyperthyroid medications or exposure to iodine, including intravenous contrast or amiodarone. The underlying hyperthyroid state is most commonly due to Graves' disease, but other causes of hyperthyroidism include toxic adenoma, toxic multinodular goiter and Hashimoto's thyrotoxicosis. Our patient likely had undiagnosed Graves' disease and an upper respiratory tract illness as her inciting event.

Medical treatment of thyroid storm aims to stop thyroid hormone production within the gland, inhibit the release of thyroid hormone, and inhibit conversion of T4 to T3. In addition, supportive treatment for adrenergic symptoms and management of end organ dysfunction are crucial. Mainstays of treatment include beta-blockers, thionamide (propylthiouracil or methimazole), iodine, steroids and for definitive therapy, thyroidectomy. Propranolol is the first line beta-blocker because it provides anti-adrenergic effects and also inhibits the peripheral conversion of T4 to T3. Propylthiouracil is the thionamide of choice in severe, life-threatening thyroid storm as it blocks peripheral conversion of T4 to T3. Methimazole is recommended for severe non-life threatening thyroid storm as it has a longer half-life than propylthiouracil, normalizes T3 more rapidly, and has less hepatotoxicity. Despite our patient's critical hyperthyroid state, she was initially started on methimazole because of her severe liver failure. She was transitioned to propylthiouracil once her thyroid hormone levels improved and her liver function stabilized. Mortality associated with thyroid

storm is predicted to be twenty to thirty percent, making it crucial to diagnose early.

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"Rock Polishing"
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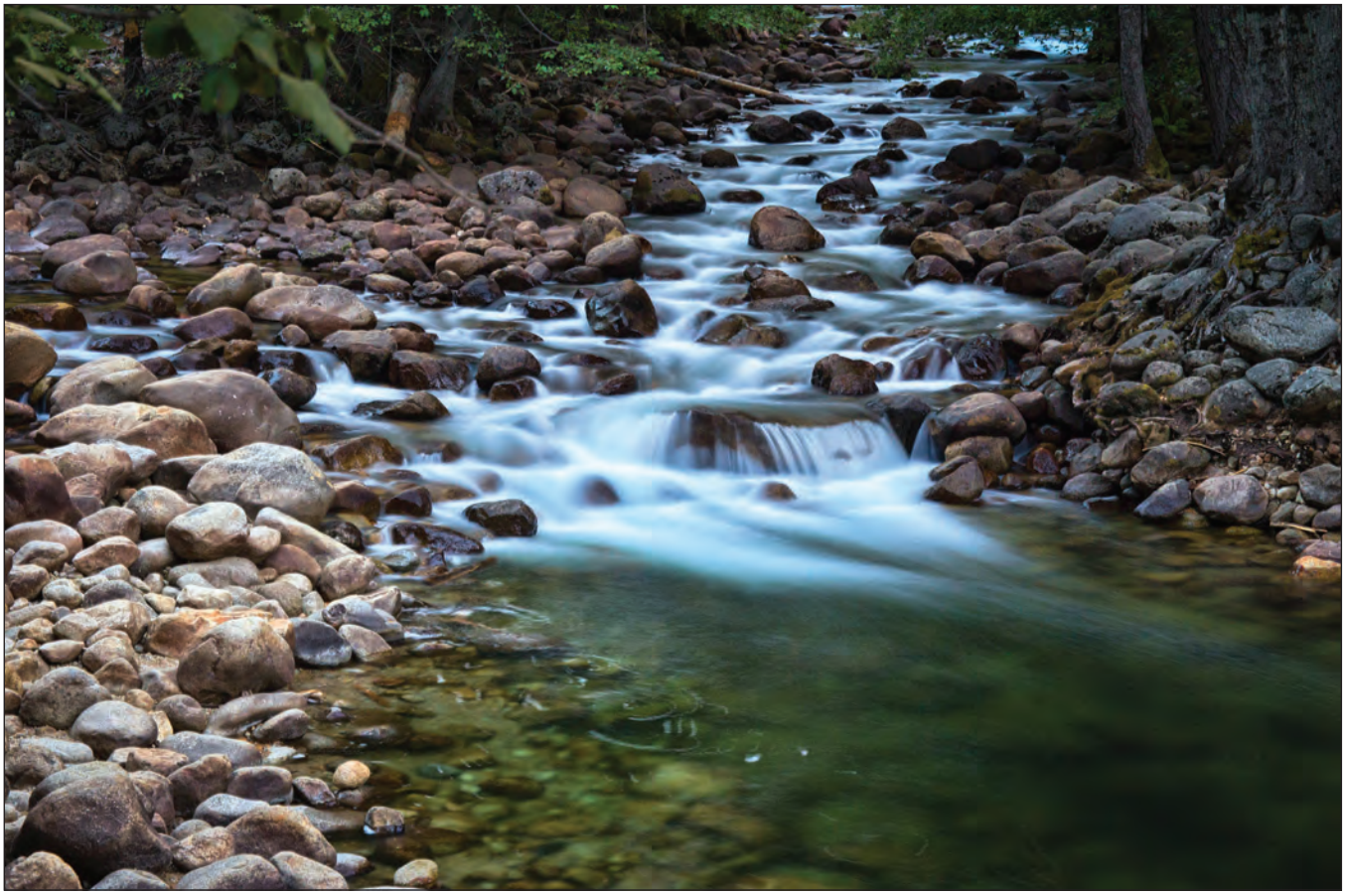

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