

## CLINICAL VIGNETTE

# An Unusual Case of Thyroid Storm

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A 43-year-old female with an eight year history of hyperthyroidism presented to the emergency department with two days of fevers and chills. Her fevers had been as high as 104 degrees. Her husband stated that she had been somnolent, weak, and fatigued. She also had a productive cough with dyspnea even at rest. She also endorsed orthopnea and paroxysmal nocturnal dyspnea over the last 5-6 days. Though she had previously been taking propylthiouracil (PTU) for her hyperthyroidism, she had stopped taking the medication after losing her medical insurance. She had no history of thyroid storm and had refused thyroidectomy in the past.

On presentation to the Emergency Department, she was febrile to 105°F, her blood pressure was 166/110, her pulse was 184 beats per minute, and she was oxygenating at 88% on room air. She was ill-appearing and lethargic with obvious exophthalmus. A goiter was noted, but no bruits were appreciated over the thyroid area. She was tachypneic and in respiratory distress with diffuse crackles on lung exam bilaterally. She was hyperreflexic with mild bilateral upper extremity weakness. Her skin was dry with poor skin turgor but no rashes.

Initial lab work showed a normal complete blood count and basic metabolic panel. Her thyroid tests showed: thyroid stimulating hormone level (TSH) < 0.02 U/ml, free thyroxine >7 ng/dL, and free triiodothyronine > 2900pg/dL. Her brain natriuretic peptide was >1000pg/mL. A troponin test was intermediate at 0.20ng/mL. A chest X-ray showed pulmonary edema. A CT scan of her head showed evidence of bilateral embolic strokes.

In the Emergency Department, the patient was intubated and placed on a ventilator. A bedside echocardiogram showed an ejection fraction (EF) of 20%, global hypokinesis, but no pericardial effusion. Her EKG showed a narrow complex tachycardia at 180 beats per minute determined to be atrial fibrillation; she was treated with diltiazem and amiodarone with no improvement of her extreme tachycardia. Three rounds of synchronized cardioversion were performed without improvement in patient's unstable rhythm. Because of the high likelihood of thyroid storm, Endocrine was consulted and she was started on systematic treatment with PTU, then high doses of propranolol. One hour later, she was started on 5 drops of potassium iodide (SSKI) every 6 hours. She was also treated with hydrocortisone. Her heart failure with associated severe pulmonary edema was treated with a furosemide drip. She was also started on heparin drip for the acute stroke.

Patient's symptoms improved over the next 1 week. She was discharged in good condition after 2 weeks in the hospital.

### Discussion

Thyroid storm is a serious type of thyrotoxicosis with a very high mortality rate between 10-30%<sup>1,2</sup> and diagnosis is made on a high clinical suspicion. The most common etiology of thyroid storm is Graves' disease, which is mediated by excess production of thyroxine hormones when thyrotropin receptors are over-stimulated by thyrotropin (TSH) receptor antibodies.

Common precipitants include infection, trauma, pulmonary embolism, and myocardial infarction.<sup>2</sup> Thyroid storm is also associated with medication non-compliance as was the problem with our patient. The pathogenesis is not very well understood; however, it is believed to be a result of increased burden of beta-adrenergic receptor density or post-receptor modification in signaling pathways. This leads to increased sensitivity to catecholamines.<sup>3</sup>

Thyroid storm remains largely a clinical diagnosis with no T4 or T3 cutoff to distinguish thyroid storm from severe thyrotoxicosis. However, objective measures such as use of the Burch and Wartosky scale<sup>4</sup> can be helpful. The scale can assist in determining the need for aggressive management by differentiating the likelihood of life-threatening thyroid storm from less severe presentations of hyperthyroidism.

Treatment of thyroid storm is based on 3 principles. First is the inhibition of thyroid hormone synthesis. Thionamides such as propylthiouracil (PTU) or methimazole are administered to decrease peripheral conversion of T4 to T3 conversion. The second principle is the inhibition of the release of thyroid hormone from the thyroid gland. Iodine therapy is used to prevent the release of pre-stored hormones and decrease iodide transfer and oxidation. Acutely, iodine therapy can stimulate new thyroid hormone production and therefore should be delayed for 1 hour after thionamide administration. Commonly used formulations of inorganic iodine include potassium iodide (SSKI) and lugol's solution. The third principle in the management of thyroid storm involves counteracting the peripheral effects of thyroid hormones using beta-blockers such as propranolol and steroids such as hydrocortisone.<sup>5,6</sup>

Definitive therapy of thyrotoxicosis after the treatment of life threatening thyroid storm includes use of gradually decreased dosing of thionamides and beta-blockers to maintain

euthyroidism. Radioactive iodine ablation and thyroidectomy should be considered several weeks after treatment of thyroid storm to prevent recurrences.<sup>7</sup>

## REFERENCES

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