Thyroid

THYROID DISORDERS CASE REPORT

Storm's a Brewing

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Intro: Thyroid storm is an often feared but overall rare complication of untreated hyperthyroidism. This severe presentation of thyrotoxicosis has multiple different treatment modalities with actual treatment directed by the patient's clinical course. We present the case of a 34-year-old male who presents in thyroid storm. **Case:** A male presented to the emergency department in southern Arizona in July with complaint of dizziness and tachycardia after working outside in the sun all day. He was rehydrated with symptomatic improvement but had continued tachycardia. The patient refused admission and left the emergency department prior to his thyroid function tests resulting with a TSH of <0.02 and FT4 of 7.43. He had no apparent follow-up or further symptoms until he presented back to the emergency department approximately one year later at age 34. He presented the second time with shortness of breath and palpitations and was found to have atrial fibrillation with rapid ventricular rate >200, a TSH <0.02, and TSH of 6.59. He was tachypneic, but his temperature and blood pressure were within normal limits. He had a CTA of the chest that showed bilateral pulmonary infiltrates. He was started on intravenous metoprolol and diltiazem without resolution of his tachycardia, so an amiodarone drip was started. Hours later, the patient became hemodynamically unstable and went into cardiac arrest. Return of spontaneous circulation was obtained and the patient was intubated with post-arrest cooling protocol initiated. The patient was clinically diagnosed with thyroid storm, which was supported by an elevated Burch-Wartofsky score. Post-arrest treatment of thyroid storm was complicated by acute liver failure, systolic heart failure with ejection fraction <20%, and persistent tachycardia. The patient was treated with high dose methimazole, propranolol, potassium iodine drops, corticosteroids, and cholestyramine. Surgery was considered given the slow progression of medical treatment but no pursued due to cardiovascular risk. Plasmapheresis was considered but not available. The patient's thyroid function was followed daily and improved over a week despite persistent tachycardia and delirium. Eventually, the patient was discharged home with daily methimazole 20mg and planned outpatient follow-up for definitive therapy. **Discussion:** This patient presented to the ED with evidence of thyrotoxicosis that was untreated for one year prior to returning with thyroid storm. This illustrates the ability of a young, otherwise healthy patient to compensate for a prolonged period with relatively few symptoms prior to decompensating. This also shows the importance of having a system in place to catch abnormal laboratory results even if the patient is no longer present in the facility; a project has already been completed to catch abnormal thyroid function testing at our facility in the future.

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Stress Cardiomyopathy Due to Exogenous Thyrotoxicosis From T3 Supplementation

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Background: Wilson's Temperature Syndrome (WTS) refers to a constellation of nonspecific symptoms, some of which include a low-normal body temperature, headaches, and fatigue. WTS was rejected by the American Thyroid Association as a valid medical diagnosis in 2005. Originally proposed in 1990 by Dr. E Denis Wilson, the etiology was theorized to be an impaired ability to convert T4 to T3, although this was not corroborated in thyroid lab abnormalities. Despite being publicly rebuffed as a true medical diagnosis, WTS has continued to gain traction amongst certain alternative medical groups and is treated with T3 supplementation. The development of a stress cardiomyopathy due to T3 supplementation is rare. Extrapolating from endogenous thyroid mediated stress cardiomyopathy and T4 supplementation induced stress cardiomyopathy, the pathophysiologic mechanism is likely excessive sympathetic activation.

Clinical Case: A 58-year old female with no prior cardiac history presented to the emergency department with chest pain. Physical exam revealed a euvolemic appearing women, with intact and symmetric distal pulses, and a normal cardiac exam without murmurs or other abnormal heart sounds. There were no abnormal lung sounds. Her O2 sats were normal on room air. EKG and CXR were unremarkable. Her troponin was elevated (3.7 ng/mL, n< 0.034 ng/mL) and her BNP was elevated (4,568 pg/mL, n< 150 pg/mL). The patient was given aspirin and started on therapeutic heparin given concern for NSTEMI. Echocardiogram revealed an ejection fraction of 30% with hypokinesis of the entire apex and mid ventricle, raising concern for a stress cardiomyopathy.

Coronary angiogram was performed which demonstrated no coronary artery disease. The patient underwent a cardiac MRI which confirmed a stress cardiomyopathy. Meanwhile, her laboratory workup was completed which revealed an undetectable TSH, a low T4 (0.53 ng/dL, n 0.7-1.8 ng/dL), and an elevated T3 (6.37 pg/mL, n 1.71-3.71 pg/mL). Patient endorsed taking oral liothyronine (T3) at doses of 5-40 mcg BID over the past 6 weeks per her alternative medicine provider for treatment of WTS. Her stress cardiomyopathy was presumed to be due to her exogenous thyrotoxicosis from T3 supplementation. She was counseled on the importance of cessation of T3 supplementation, and was started on heart failure medications. On hospital day 3, her T3 normalized to 2.73 pg/mL, and T4 remained low (0.4 pg/mL). Follow-up echocardiogram four months later demonstrated an ejection fraction that had improved to 45%.

Conclusion: This case highlights the importance of physician awareness of alternative medicine diagnoses and treatment regimens that affect thyroid hormones and may cause harm to patients. This case is an important reminder of the effect thyroid hormones have on coronary vasculature, myocytes and myocardial function.