Thyrotoxicosis Presenting as STEMI and Cardiogenic Shock

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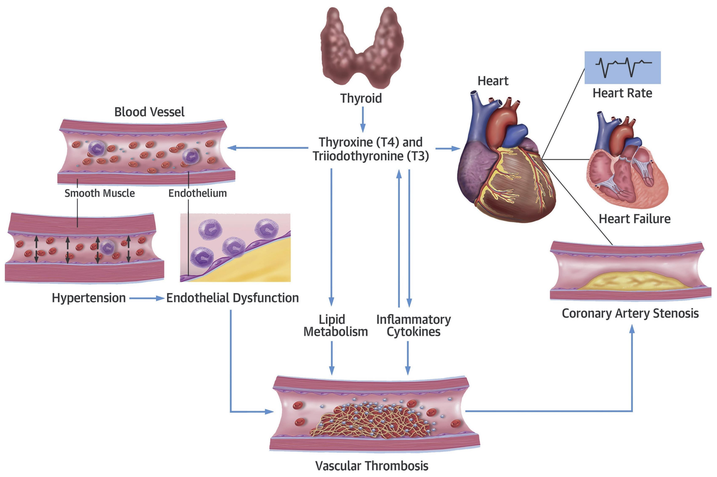
Introduction

Major cardiac manifestations of thyrotoxicosis include tachyarrhythmias, particularly atrial fibrillation. This is mediated by thyroid hormone receptors on myocytes which reduce action potential duration. Secondarily, increased levels of thyroid hormone increase the adrenergic response to catecholamines leading to increased heart rate and contractility.



Razvi, S. et al. J Am Coll Cardiol. 2018;71(16):1781-96.

Figure 1. The effect of thyroid hormones on the cardiovascular system[1]

Less commonly, thyroid storm has been associated with myocarditis (both viral and autoimmune), stress cardiomyopathy and ST-segment elevation myocardial infarction. Cardiomyopathy in the setting of Graves' disease has previously been reported with elevated TSH receptor antibodies, although the exact effect of these autoantibodies on cardiac myocytes during the observed cardiomyopathy has yet to be determined[2]. Thyroid storm has been reported to have a mortality of 30%[3] and these rarer presentations coupled with hemodynamic instability necessitate prompt recognition for appropriate treatment.

Methods

An anxious 39 year-old male with a history of hypertension and opiate abuse presented from home for chest pain. Review of systems was significant for intentional weight loss. EKG demonstrated ST-segment elevation in aVR with progressive depressions in V2-V6 on repeat EKGs. Emergent cardiac catheterization demonstrated no coronary artery disease but did reveal severe pulmonary hypertension with severely elevated pulmonary wedge pressures and low cardiac output. Urgent echocardiogram demonstrated systolic dysfunction (ejection fraction=35%) with decreased apical motion. While there was mild-moderate tricuspid regurgitation and mild mitral regurgitation, there were no other gross valvular abnormalities. There was no Takotsubo cardiomyopathy. Soon after, the patient suffered multiple cardiac arrests with pulseless electrical activity and unstable ventricular tachycardia. ACLS was performed, the patient was endotracheally intubated and ROSC was obtained. The patient was started on vasopressors and amiodarone.

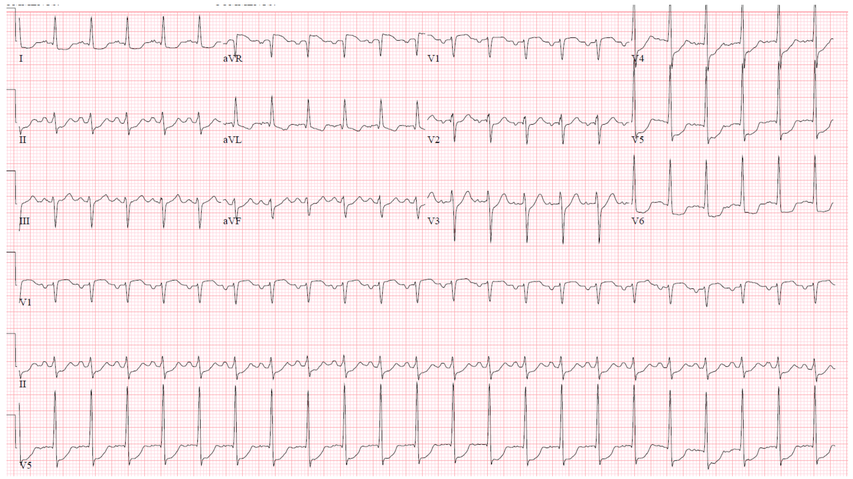


Figure 3. Electrocardiogram demonstrating sinus tachycardia with ST-segment elevation in aVR and depressions in V2-V6.

Results

Soon after the patient's cardiac arrest, admission testing demonstrated undetectable Thyroid Stimulating Hormone (TSH<.01 uIU/mL, reference range 0.36-3.74 uIU/mL) and elevated T4 (4.69, reference 0.76-1.46 ng/dL). The patient was treated for severe hyperthyroidism with propylthiouracil, potassium iodide, cholestyramine and hydrocortisone but was too hypotensive for beta-blocker therapy. Viral panel was negative. TSH receptor antibodies were elevated (8.77 IU/L, Reference <2 IU/L). Thyroid peroxidase antibodies were also elevated (82 IU/mL, Reference <9 IU/mL). Thyroglobulin antibodies were normal (<1 IU/mL, Reference <1 IU/mL). After two days of initial improvement in vasopressor requirement, the patient suffered another cardiac arrest with persistent hypoxia despite mechanical ventilation, pulmonary vasodilators and paralysis. The patient was cannulated for venoarterial extracorporeal membrane oxygenation (VA-ECMO) and oxygen saturation improved with mechanical support.

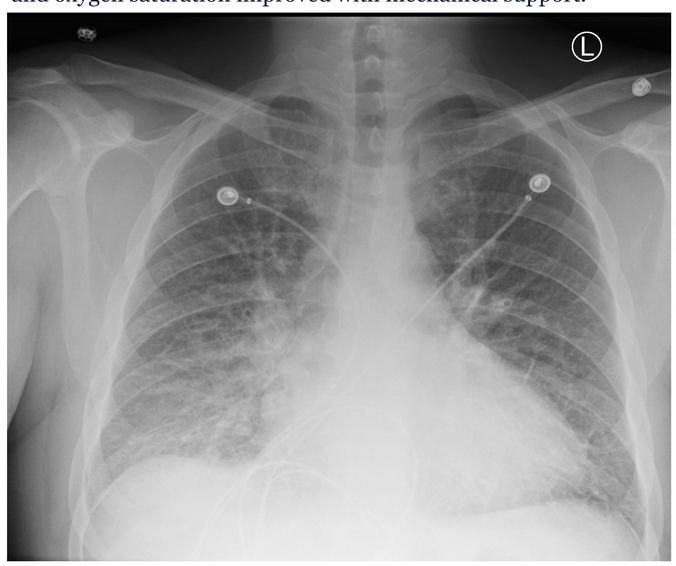


Figure 2. PA and Lateral Chest X-ray films demonstrating increased vascular congestion likely due to pulmonary edema in the setting of cardiogenic shock.

Indications for VA-ECMO
Fulminant Myocarditis
Pulmonary hypertension* and right heart failure
Cardiogenic Shock*: with or without MI
Pulmonary embolus with hemodynamic compromise
Cardiac arrest*
Medication overdose
Nonischemic cardiomyopathy
Bridge to transplant or VAD
Support post cardiac surgery
ARDS
Extracorporeal assistance for lung rest (e.g. contusion)
Lung transplant
Lung hyperinflation (e.g. status asthmaticus)
Congenital diaphragmatic hernia/meconium aspiration

Table 1. Cardiac and Pulmonary indications for VA-ECMO. Asterisks included next to indications relevant to this case. Contraindications include: unrecoverable heart and patient is not transplant or VAD support candidate with destination therapy, disseminated malignancy, known severe brain injury, unwitnessed cardiac arrest, prolonged CPR without adequate tissue perfusion, unrepaired aortic dissection, severe aortic regurgitation, severe chronic organ dysfunction, compliance (e.g. social limitation), PVD in peripheral ECMO. Relative contraindications include anticoagulation, advanced age and obesity. [4]

Conclusions

Uncommon presentations for thyrotoxicosis include cardiomyopathy, myocarditis, vasospasm or in this case, with ST-elevation myocardial ischemic patterns. In this case, the patient likely had Graves' disease with excessive thyroid hormones causing his presentation in cardiogenic shock. The elevated TSH receptor antibodies observed in this patient are consistent with previously reported Graves' disease associated cardiomyopathy. Stress cardiomyopathy, or Takotsubo cardiomyopathy, has also been observed in thyrotoxicosis, likely due to increased action of catecholamines on myocytes in thyroid storm. Unfortunately, the patient's severe hypotension precluded the use of beta blockers, which are more frequently used in patients who survive thyroid storm[5] . In the setting of hypoxic respiratory failure and cardiogenic shock refractory to treatment, access to ECMO should be considered.

References

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