**CLINICAL VIGNETTE**

# A Case of Thyroid Storm: Unusual Presentation in a Young Man

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***Case Report***

A 36-year-old man with a history of alcohol, cocaine, and methamphetamine abuse presented to the emergency department (ED) with abdominal distention and palpitations. He was previously seen at an outside clinic with progressively worsening abdominal swelling and an irregular heartbeat. He had lower extremity edema, dyspnea on exertion, orthopnea, and pruritus, as well as a rapid weight gain of 30 pounds over the 4 weeks prior to his clinic visit. His social history was significant for use of multiple recreational drugs, including prior heavy cocaine and methamphetamine abuse, occasional marijuana use, and 9 years of alcohol abuse, consuming between 5-18 standard drinks per day. The patient's reported no cocaine or alcohol use for 2 weeks prior to presentation. He was told he had "cirrhosis" and was sent to the ED after he was found to be in atrial fibrillation with rapid ventricular response (RVR).

On initial exam, the patient's vitals included: temperature 36.8 C, respiratory rate 20 breaths per minutes, pulse 140-170 beats per minute, blood pressure 143/98 mmHg, and oxygen saturation 96% on room air. He had an irregularly irregular heart rate, elevated jugular venous pressure measured at 12 cm $H_2O$, bibasilar crackles, abdominal distension with flank bulging, positive fluid wave, scrotal swelling, 3+ pitting edema in the lower extremities up to the upper thighs, and jaundice. EKG confirmed a diagnosis of atrial fibrillation with RVR.

Laboratory tests were significant for thyroid stimulating hormone (TSH) <0.02 mcIU/mL (0.3-4.7), triiodothyronine (T3) 1190 pg/dL (222-383), and thyroxine (T4) >7.0 ng/dL (0.8-1.6). Additional thyroid studies included thyroid stimulating immunoglobulin (TSI) 173% (<122%), thyroid peroxidase antibody >600 IU/mL (<=20), and thyrotropin binding inhibitory Ig 53% (<16%). Liver function tests were significant for an AST 55 U/L (13-47), ALT 23 U/L (8-64), total bilirubin 1.4 mg/dL (0.1-1.2), and alkaline phosphatase 196 U/L (37-113). Chest X-ray showed moderate pulmonary edema, bilateral pleural effusions, and moderate cardiomegaly. Echocardiogram revealed an ejection fraction of 25-30% and 4-chamber enlargement. Liver ultrasound was without evidence of cirrhosis, but was suggestive of passive liver congestion.

In the ED, the patient was started on an esmolol infusion along with dexamethasone, iopanoic acid, and methimazole. The patient's volume status was managed with diuretics while atrial fibrillation with RVR was managed with beta-blockade and eventually DC cardioversion. The patient was transferred to ICU for closer monitoring. At discharge, the patient's T3 had normalized and T4 was continuing to downtrend.

***Discussion***

Thyroid storm is a rare complication of thyrotoxicosis that occurs in 1-2% of hyperthyroid patients, and favors women in their third to sixth decade of life.[1,2] It has an overall mortality rate between 10-20%, but can increase to 75% if treatment is delayed.[3] It can present with thyrocardiac disease, of which arrhythmias are the most common, including atrial fibrillation in 15% of patients. Most hyperthyroid patients have higher than normal cardiac output that when left untreated can, lead to myocardial hypertrophy, dilated cardiomyopathy and in a minority of cases, symptomatic heart failure. Heart failure in young patients, is typically "high output" cardiac failure that results from protracted and severe thyrotoxicosis, and typically responds well to diuretics.[4] Heart failure can also be both a trigger or result of thyroid storm. Six percent of thyrotoxic patients can develop this complication.[4] The etiology of congestive heart failure in this patient is multifactorial, including tachycardia from thyrotoxicosis, as well as possible underlying heart disease from heavy cocaine and methamphetamine abuse.[4] While cardiac abnormalities can reverse with antithyroid treatment, possible underlying structural heart disease increases uncertainty in cardiac function improvement.

Diagnosis of thyroid storm requires life-threatening symptoms in the presence of biochemical evidence of thyrotoxicosis. While there is no standard tool for diagnosis of thyroid storm, Burch and Wartofsky introduced a point system that takes into account a number of clinical features of thyroid storm. Points are assigned for fever, heart failure, and the dysfunction of CNS, GI-hepatic, and cardiovascular systems.[5,6] Liver involvement in hyperthyroidism commonly include mild elevations in liver transaminases; however, liver failure is rare.[7]

Standard treatment of thyroid storm involves beta-blockade which can manage tachyarrhythmias and decrease T4 to T3 conversion, iopanoic acid and glucocorticoids which also inhibit T4 to T3 conversion, and thionamides which blocks synthesis of new thyroid hormones. Bile acid sequestrants can decrease the recycling of thyroid hormones through the enterohepatic system. Adequate treatment involve supportive therapy and counteracting the effects of thyroid hormone, which should provide clinical improvement within 12-24 hours.[3]

Because the patient first sought care for rapid abdominal distention, with polysubstance abuse,he was diagnosed with presumed cirrhosis. His presentation was further complicated by the lack of characteristic physical exam findings of hyperthyroidism, including pyrexia, goiter, lid lag, exophthalmos, and weight loss. This young man with signs and symptoms of heart failure and arrhythmia provided adequate suspicion for thyroid storm, and treatment was initiated immediately and he had an uneventful recovery.

## REFERENCES


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