The Journal of Emergency Medicine, Vol. 53, No. 6, pp. e125–e128, 2017

0736-4679/$ - see front matter

http://dx.doi.org/10.1016/j.jemermed.2017.08.096

Clinical Communications: Adults

IODINATED CONTRAST ADMINISTRATION RESULTING IN CARDIOGENIC SHOCK IN PATIENT WITH UNCONTROLLED GRAVES DISEASE

Wes Brundridge, DO* and Jack Perkins, MD, FAAEM, FACP†

*Department of Ophthalmology, Brooke Army Medical Center, San Antonio, Texas and †Virginia Tech Carilion Emergency Medicine Residency, Department of Emergency Medicine, Virginia Tech Carilion School of Medicine, Roanoke, Virginia

Reprint Address: Jack Perkins, MD, FAAEM, FACP, Department of Emergency Medicine, Virginia Tech Carilion Emergency Medicine Residency, Virginia Tech Carilion School of Medicine, 1 Riverside Circle, 4th Floor, Roanoke, VA 24014

☐ **Abstract—Background: Thyroid storm (also known as thyroid or thyrotoxic crisis) is part of the spectrum of thyrotoxicosis and represents the extreme end of that spectrum. The condition is quite rare, yet mortality rates are high and may approach 10–30%. Case Report: A 34-year-old-man who had a history of Graves disease presented in atrial fibrillation with rapid ventricular response and mild congestive heart failure. During the course of his Emergency Department (ED) stay he deteriorated into cardiogenic shock. Roughly 10 h transpired between his presentation and the development of cardiogenic shock. He had received an intravenous contrast load of iohexol shortly after initial presentation, and the associated iodine bolus, we suspect, contributed to his abrupt deterioration into cardiogenic shock. Why Should an Emergency Physician Be Aware of This?: Thyroid storm is infrequently seen in the ED, and there is potential for management errors that can lead to a detrimental patient outcome.**

☐ **Keywords—thyroid storm; iodinated contrast; cardiogenic shock; Graves disease**

INTRODUCTION

Thyroid storm is an acutely exaggerated manifestation of the thyrotoxic state. It is not uncommon for patients with thyrotoxicosis to present to the emergency department (ED), however, it is very uncommon for those patients to present or develop thyroid storm. We report a case of a patient with a history of Graves disease who initially presented with mild congestive heart failure and atrial fibrillation with rapid ventricular response. He received an iohexol contrast load and subsequently developed cardiogenic shock. We review the literature and discuss the risks of iodinated contrast in patients with uncontrolled hyperthyroid disease.

CASE REPORT

A 34-year-old African-American man with a history of Graves disease presented to our ED with a chief complaint of shortness of breath. He stated that a few weeks prior to presentation he began having dyspnea and palpitations. The dyspnea was progressive, and by the time of presentation he endorsed orthopnea as well. He also reported lower-extremity edema, insomnia, anxiety, and a 30-pound weight loss in the past few months. He denied any recent fever and had not had any chest pain. The review of systems was remarkable for a decreased appetite, thinning hair, dizziness, and fatigue. He was initially diagnosed with Graves disease in 2011 and was briefly on antithyroid medications before he stopped taking them due to cost. Most recently he was taking propranolol, but he discontinued this a few weeks prior to presentation. He reported daily marijuana use, no

Received: 16 March 2017; Final submission received: 25 August 2017;
Accepted: 30 August 2017

alcohol consumption, daily cigarette use, and denied other illicit drug use.

On examination his temperature was 36.9°C, heart rate 119 beats/min, blood pressure 92/65 mm Hg, respiratory rate of 26 breaths/min, and pulse oxygenation of 100% on room air. He was a cachectic-appearing, ill man who looked older than his stated age. His hair was coarse and his thyroid was noted to be enlarged diffusely. His heart rate was tachycardic and irregularly irregular. Lung sounds were diminished at the right base but he was not in distress and had no wheezes or rales. His abdominal and neurologic examinations were unremarkable. He was noted to have 2+ lower-extremity pitting edema.

He had an electrocardiogram (Figure 1), chest X-ray study (CXR; Figure 2), and serum laboratory values obtained. His electrocardiogram revealed atrial fibrillation at a rate of 164 beats/min without any ischemic changes. His CXR showed a moderate-sized right pleural effusion, cardiomegaly, and a widened mediastinum, which was new compared with a CXR 7 years earlier. His laboratory results are reflected in Table 1. Computed tomography (CT) of the chest with intravenous (i.v.) contrast was ordered for the mediastinal abnormality seen on CXR, and it revealed a 6-cm ascending aortic aneurysm. The patient was administered 40 mg of i.v. furosemide and started on a diltiazem drip. He was admitted to a telemetry unit under the hospitalist service. He was placed in a holding area in the ED, as no bed was available at the time of admission.

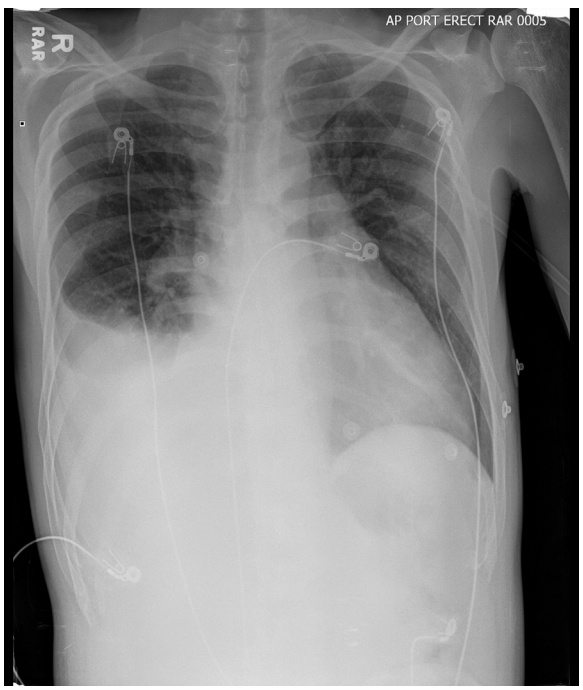

Figure 1. A portable chest X-ray study reveals cardiomegaly, a moderate right pleural effusion, and a slightly enlarged mediastinum.

Roughly 10 h after initial presentation the patient decompensated rapidly and a rapid response was initiated at his bedside. He was found at this time to be bradycardic at 48 beats/min, hypotensive at 62/28 mm Hg, poorly responsive to verbal or painful stimuli, and had agonal respirations. He received 1 mg of epinephrine and 200 μg of phenylephrine to stabilize his blood pressure prior to endotracheal intubation. He also received 2000 mL of intravenous normal saline. A rapid ultrasound for shock and hypotension (RUSH) examination was performed and free fluid was observed in Morrison's pouch. A central line was placed and he was started on an epinephrine drip. When his mean arterial pressure was > 65 mm Hg, he was taken for a CT angiogram of his chest, abdomen, and pelvis, as there was significant concern for dissection or rupture of the known proximal aortic aneurysm, and to further investigate the free fluid seen on the RUSH examination.

The CT angiogram of the chest/abdomen/pelvis did not reveal any aortic dissection and yielded no answer for his abrupt decompensation. The patient was given 200 mg of hydrocortisone and empirically administered cefepime 2 grams i.v. and vancomycin 30 mg/kg i.v. A thyroid-stimulating hormone level returned at < 0.01 uIU/mL, and a free thyroxine (T4) level returned at 26.0 ug/dL (normal 4.5–12.0 ug/dL). He was started on 100 mg propylthiouracil every 4 h, and an esmolol drip in addition to phenylephrine and vasopressin drips. Cardiology was consulted and a bedside echocardiogram revealed an ejection fraction of 40–45%, with reduced left and right ventricle systolic dysfunction. He was admitted to the Intensive Care Unit, where he had a complex 28-day hospital course but was discharged neurologically intact. His final diagnosis was cardiogenic shock secondary to thyroid storm.

DISCUSSION

Thyroid storm is an acutely exaggerated manifestation of the thyrotoxic state. Many of the manifestations of thyrotoxicosis are related to the increase in oxygen consumption and use of metabolic fuels associated with the hypermetabolic state (1). Thyroid hormone has many effects on the heart and vascular system. Many of the clinical manifestations of hyperthyroidism are due to the ability of thyroid hormone to alter cardiovascular hemodynamics. Excess thyroid hormone causes palpitations, with some degree of exercise impairment and a widened pulse pressure, independently of the cause of the hyperthyroidism. The changes in heart rate result from both an increase in sympathetic tone and a decrease in parasympathetic tone. In patients with hyperthyroidism, cardiac output is 50–300% higher than in normal subjects. The