Post Coronary Artery Bypass Graft (CABG) Surgery Thyroid Storm: A Near Miss

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Altered mental status (AMS) is a challenging symptom, commonly encountered in the acute hospital setting. We present a challenging case of AMS where an early assessment could have potentially prevented a complicated hospital course. A 42-year-old male presented to emergency room with chest pain. He was tachycardic, hypotensive and electrocardiogram showed new ST depressions. He underwent emergent cardiac catheterization showing triple vessel disease with placement of intra-aortic balloon pump, followed by CABG. Post-operatively he could not be extubated and had persistent fever, tachycardia and deterioration in mental status. Initial work up for fever and AMS was unremarkable. He was treated with broad spectrum antibiotics and beta blockers but remained intubated, febrile, and altered. Thyroid function tests done around second week of hospitalization revealed TSH < 0.03uU/ml, fT4 > 4.5 ng/dl, T3 411.9 ng/dl, thyroid stimulating immunoglobulin 269%. A thyroid sonogram revealed diffusely enlarged, heterogeneous and hypervascular thyroid lobes. He was started on stress doses of steroids, propylthiouracil and continued on beta blocker. Within the next 72 hours his heart rate, fever and mental status improved and he was extubated. Thyroid storm, also referred to as thyrotoxic crisis, is an acute life threatening hypermetabolic state induced by excessive release of thyroid hormones in a patient with thyrotoxicosis. It is often precipitated by an acute event such as surgery, trauma, infection or iodine load. The Burch and Wartofsky scoring system is helpful in distinguishing this from severe thyrotoxicosis. It should always be kept in the differential diagnosis of AMS, fever, tachycardia of unknown cause.

Key words: thyroid storm; hyperthyroidism; CABG

Introduction

Altered mental status (AMS) is a challenging symptom, commonly encountered in the acute inpatient setting (1). The underlying etiology could be metabolic, infectious, toxic, cardiac, neurological or trauma (2). We present a challenging case of AMS where an early comprehensive assessment could have potentially prevented a prolonged and complicated hospital course.

Case presentation

A 42-year-old otherwise healthy male, presented to the emergency room with chest pain and worsening shortness of breath. On examination, he was noted to be diaphoretic, pale, hypotensive and tachycardic (heart rate was 114/min, BP 75/44 mmHg). The rest of the physical examination was benign. The electrocardiogram (EKG) showed new ST depressions with elevated troponins. He underwent emergent cardiac catheterization, showing severe 3-vessel disease with left main disease and moderate to severe left ventricular dysfunction. Due to cardiogenic shock, an intra-aortic balloon pump was placed and coronary artery bypass graft (CABG) surgery was performed the next day. On post-operative day 1, patient developed fever (100.8F to 103.1F) and sinus tachycardia. Mental status also became progressively altered with episodes of agitation requiring increasing levels of sedation. He was treated with broad spectrum antibiotics and beta blockers. Over the next few days, he failed multiple spontaneous breathing trials in an attempt to extubate. The work up for persisting fever and AMS included blood, urine and sputum cultures, respiratory viral panel, sputum for acid fast bacilli (AFB), stool for ova and parasite, serology for leptospirosis, testing for human immunodeficiency virus (HIV) and hepatitis, lumbar puncture, electroencephalogram, magnetic resonance imaging (MRI) of the brain and venous Doppler studies. The patient was found to have left posterior tibial vein thrombosis and was started on anticoagulation. But he continued to be febrile, tachycardic, altered and intubated.

Thyroid function tests done around week 2 of hospitalization revealed thyroid-stimulating hormone (TSH) $< 0.03 \mu\text{U/ml}$ ($0.27 - 4.2 \mu\text{U/ml}$), $\text{fT}_4 > 4.5 \text{ ng/dl}$ ($0.9 - 1.7 \text{ ng/dl}$), $\text{T}_3 411.9 \text{ ng/dl}$ ($80 - 200 \text{ ng/dl}$), and thyroid stimulating immunoglobulin 269% ($< 122\%$). A thyroid sonogram revealed diffusely enlarged, heterogeneous and hyper-vascular thyroid lobes. Further history from wife revealed that the patient has had unintentional 25-pound weight loss over the last 3 months. He was started on hydrocortisone 100 mg every 8 hours, propylthiouracil 100 mg every 8 hours and continued on beta blocker, titrated to heart rate. Over next 72 hours his heart rate, fever and mental status improved and he was successfully extubated. He was discharged on oral methimazole and tapering dose of steroids. The patient continues to remain euthyroid and asymptomatic in the most recent endocrinology clinic follow up.

Discussion

Thyroid storm, also referred to as thyrotoxic crisis, is an acute life threatening hypermetabolic state induced by excessive release of thyroid hormones in patients with thyrotoxicosis (3). It is a rare condition with a multisystem involvement with an incidence of 0.2 per 100,000 per year in hospitalized patients (4). Thyroid storm most commonly occurs in women, with Grave's disease being the most commonly reported underlying pathology (3). It is often precipitated by an acute event like trauma, surgery, infection, iodine load or parturition. Infection is the most commonly reported precipitant in inpatient setting (4-6). However, in about 25% - 43% of cases a clear precipitating event is not identified (7).

In our patient it is possible that stress of cardiac surgery along with iodine load during cardiac catheterization precipitated the episode of thyroid storm (3, 6). There is also a probability that he may have had undiagnosed hyperthyroidism without overt manifestations prior to this hospital encounter. To the best of our knowledge, there are only two previously reported cases of thyroid storm occurring in the setting of CABG (8, 9). Our case had minimal pre-operative findings of

thyrotoxicosis, no known risk factors of cardiovascular disease, post-operative thyroid storm and prompt response to treatment.

High fever is seen in almost all patients presenting with thyroid storm and can help to distinguish thyroid storm from thyrotoxicosis (10). Anxiety, agitation, delirium and coma are also commonly seen and are considered by many as essential to the diagnosis (4). In one study, altered mental status was the only reliable differentiating feature between thyroid storm and thyrotoxicosis (11). Cardiac manifestations include cardiac arrhythmias, congestive heart failure, hypotension and death may also occur from cardiovascular collapse (12). The degree of elevation of thyroid hormones or suppression of TSH does not correlate with the clinical severity (13). The Burch and Wartofsky scoring system, based upon clinical criteria can be helpful in recognizing thyroid storm and differentiating from thyrotoxicosis (14).

Patients in thyroid storm are critically ill and prompt initiation of resuscitative measures is required. Treatment guidelines recommend a multifactorial approach for the management of thyroid storm including monitoring in the intensive care unit, respiratory support, volume resuscitation, cooling measures with acetaminophen and cooling blankets, beta blockers, anti-thyroid drugs, inorganic iodide and corticosteroids (15). Propranolol is the most commonly used beta-blocker in this setting due to its non-selective beta blocking property and decreased conversion of T4 to T3 in the periphery (3, 6). Doses as high as 60 mg to 120 mg every 6 hours, with appropriate adjustment for heart rate and blood pressure, may be required due to increased drug metabolism in thyroid storm (16). Regarding the use of anti-thyroid drugs there is no strong data showing clinical benefit of a specific thionamide agent over another, but in the acute setting, propylthiouracil is generally used. Propylthiouracil blocks T4 to T3 conversion and there is some evidence that in the first few hours after administration, propylthiouracil reduces serum T3 concentration more rapidly than methimazole (17). Glucocorticoid administration prevents adrenal crisis, as there is a potentially limited adrenal reserve in this acute setting. It also decreases the T4 to T3 conversion and in patients with Grave's disease may have an effect on the underlying autoimmune disease process (18). In patients who fail medical therapy, therapeutic plasma exchange is an option (19).

Thyroid storm should always be kept in the differential diagnosis of altered mental status, fever and tachycardia of unknown cause. Prompt recognition and aggressive treatment is of utmost importance since this condition carries a high mortality if left untreated (6).

Notes

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