

An Unusual Case of Thyroid Storm

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Abstract

Administration of a large dose of radioactive iodine leading to excess thyroid hormone release is a rare cause of thyroid storm. A 56 year-old Hispanic female presented to the Emergency Department (ED) complaining of shortness of breath and lightheadedness. The patient was given iodine-131 three days prior to presentation. Thyrotoxicosis and thyroid storm are very rare complications of radioactive iodine administration. We present a case of an unusual cause of thyroid storm and discuss the pathophysiology, diagnosis and treatment.

Key Words: Thyroid Storm, Iodine 131, Iatrogenic, Thyrotoxicosis

Introduction

Thyroid storm is a life-threatening syndrome of decompensated thyrotoxicosis (1,2). Thyroid storm usually develops in an undiagnosed hyperthyroid patient who has a major stress or continues without anti-thyroid treatment. The precipitating stress may be a medical illness, trauma, surgical procedure or iodine load (3). We present an unusual case of thyroid storm resulting from prior treatment with iodine-131 and a brief discussion of thyroid storm, its proposed mechanism, pathophysiology and treatment.

Case Report

A 56-year-old woman presented by ambulance to the ED with the chief complaint of shortness of breath that awoke her. In the ambulance she was given one sublingual nitroglycerin and 40 mg intravenous furosemide. The patient also complained of lightheadedness, 1-2 episodes of nausea, vomiting and chest pain described as tightness without radiation. She also described being profoundly weak. She gave no prior history of similar symptoms. Past medical history included rheumatic heart disease, a transient ischemic attack 2 months ago, hypertension and hyperthyroidism. She was allergic to propylthiouracil. She mentioned that she had been to another hospital's clinic three days previously for treatment of her thyroid condition but was unsure what treatment she had received. Her only medication was propranolol 10 mg twice a day. She denied any smoking history, illicit drug or alcohol use.

Physical examination revealed a tremulous and anxious diaphoretic woman in moderate to severe respiratory distress. Vital signs: blood pressure of 168/100 mm Hg; pulse of 120 beats/minute; respiratory rate of 28-30 breaths/minute; and an oral temperature of 100.1°F. Patient's oxygen saturation was 77% on room air. The examination of the head, eyes, ears, nose and throat were unremarkable. There was no lid lag or exophthalmos present. Her neck revealed jugular venous distention and no carotid or thyroid bruits. There was no palpable goiter. Her chest revealed that she had rales up to the mid-lung fields and her heart examination revealed a tachycardia with no rubs, gallops or murmurs. Her abdomen was soft and benign. There was no clubbing no edema of the extremities and her pulses were equal and symmetrical. Rectal examination revealed a guaiac negative stool. Her neurological examination was normal except for symmetrically brisk deep tendon reflexes.

The patient was given 100% oxygen via a non-rebreather placed on a cardiac monitor, an intravenous line was inserted and a laboratory work-up sent off. A portable chest x-ray revealed pulmonary edema with a normal sized heart and the electrocardiogram

showed a sinus tachycardia with T wave inversion in leads I, aVL, II, III, aVF and V₂-V₆. The patient was given an additional 40 mg of intravenous furosemide, 4 mg of intravenous morphine sulfate for her pain and anxiety and started on nitroglycerin for relief of her shortness of breath and chest pain.

An initial arterial blood gas obtained on room air showed a pH of 6.95; a PaCO₂ of 46mm Hg; a PaO₂ of 77 mm Hg; and an oxygen saturation of 83%. A repeat blood gas revealed a pH of 7.15; a PaCO₂ of 43 mm Hg; a PaO₂ of 80; and an oxygen saturation of 91% with a urine output of 300 cc. There was mild improvement of her respiratory distress. The complete blood count, electrolytes, blood urea nitrogen and glucose measurements were normal. Two sets of cardiac enzymes were negative for cardiac insult.

Despite the 100% oxygen therapy and the medication administered, the patient remained anxious, tremulous and short of breath. She was initially thought to have acute pulmonary edema secondary to myocardial ischemia but the possibility of pulmonary embolus was also considered. She was started on intravenous heparin, given aspirin and sent for a ventilation/perfusion scan.

Only the ventilation part of the ventilation/perfusion scan was preformed. There was a large area of radioactive uptake in the region of the thyroid blacking out both lung fields. The radiologist questioned the ED physicians whether the patient had undergone a thyroid scan recently. At this point we considered the diagnosis of thyroid storm precipitated by iatrogenic exposure to iodine. Later, the patient's son had arrived and was able to relate that the patient had received an unknown dose of iodine-131 to ablate her thyroid gland as treatment for her Grave's disease three days ago at her clinic.

She then received 100 mg of hydrocortisone, low dose propranolol and Lugol's solution. The Lugol's solution was given, after consultation with the endocrine service, to prevent any further release of thyroid hormone. In the intensive care unit a third set of cardiac enzymes revealed a creatine phosphokinase of 1274 mg/dl with the MB fraction positive. The thyroid function tests were abnormal with free T4 measuring at 5.1 ng/dL, T3 was 273 ng/mL and the TSH low - less than .01 mIU/mL. The patient did well but did have some residual heart problems.

Discussion

Thyrotoxicosis is a hypermetabolic condition that develops after body tissues are exposed to increased concentrations of thyroxine (T4), free triiodothyronine (FT3) or both. Thyrotoxicosis is a subset of hyperthyroidism generally affecting approximately 1-2% of women and 0.2-0.3% of men (1). Clinical manifestations of thyrotoxicosis affects every organ system and the presentation can vary with the person's age (2-4). Thyroid storm, a severe form of thyrotoxicosis, is a life-threatening medical emergency. It presents as a clinical syndrome: a severely ill patient with fever (>101 F), tachycardia out of proportion to the fever, central nervous system excitation, hypertension and gastrointestinal dysfunction. Tachydysrhythmias are common and peripheral effects are especially prominent. Elderly patients may exhibit apathy or confusion, cachexia and atrial fibrillation (1-3). The patient may present with cardiovascular collapse. Thyroid storm results from untreated or inadequately treated thyrotoxicosis and may be precipitated by infection, trauma, surgery, an embolic phenomenon, uncontrolled diabetes, toxemia of pregnancy or labor. Thyroid storm most commonly occurs in patients who have Grave's disease, thyroiditis and toxic multinodular goiter. Less common causes include excess thyroid stimulating hormone (trophoblastic tumors, pituitary tumors), ectopic thyroxine production (struma ovarii and thyroid carcinoma) and iodine-induced thyrotoxicosis.

Iodine-induced hyperthyroidism is also known as the Jod-Basedow phenomenon. The name combines the German word for iodine and a tribute to Karl Adolph von Basedow, a German contemporary of Robert Graves(4). Iodine ingestion may cause hyperthyroidism with a low thyroid radioactive uptake. Since the thyroid radioactive iodine uptake is inversely proportional to iodine intake, low radioactive iodine uptake is readily explained. It is most often seen in patients with underlying multinodular goiter or thyroid adenoma who are given drugs that contain inorganic iodine (e.g., amiodarone, iodine-containing expectorants) or whose radiologic and cardiac studies use iodine-rich contrast agents (3,4). Thyrotoxicosis occurs a few weeks after a large dose of iodine is administered, typically in a contrast medium. The phenomenon is far more common in areas of the world where iodine intake is low or marginal such as mountainous regions, but can occur with adequate dietary iodine intake. A related cause of thyrotoxicosis and thyroid storm is radiation induced thyroiditis secondary to the administration of iodine-131.

Radioactive iodine causing thyrotoxicosis or thyroid storm is a very well described entity but is rare in the literature (5,6). In 1983, McDermott et al. discussed a case report of radioiodine-induced thyroid storm and reviewed 15 other cases of radioactive iodine induced thyroid storm. In his review, McDermott noted the mean age of the patients in the 16 cases was 53.4 years and of the 16 cases, only ten were pre-medicated with either a thionamide or beta-blocker but still had been symptomatic before radioiodine therapy (6). They concluded that the patients at high risk for going into thyroid storm after radioiodine therapy include "the elderly, those with severe thyrotoxicosis or significant weight loss and those with underlying or thyrotoxic related cardiovascular disease or cerebrovascular disease" (6). They stressed the adequate treatment of underlying or complicating illnesses and recognized the importance of pre-treating thyrotoxic patients with either thionamides and/or beta-blockers.

Treatment of thyroid storm involves supportive care, blocking peripheral effects, inhibition of thyroid hormone synthesis and release and inhibition of peripheral hormone conversion. Supportive care involves antipyretics and cooling measures, fluids, correction of electrolytes, pretreatment thyroid function tests and symptomatic treatment. Peripheral effects are controlled with beta-blockers. Calcium channel blockers can be used as an alternative if beta-blockers are contraindicated or poorly tolerated. Next, high dose anti -thyroid drugs are administered to prevent further thyroid synthesis. After one hour Lugol's solution is given to saturate receptors to prevent hormone release. Lastly, steroids are given to prevent peripheral conversion of T4 to T3 and to treat the relative adrenal insufficiency that develops.

Radioactive iodine is being increasingly used as first line therapy for Grave's disease and is the treatment of choice for recurrent hyperthyroidism after thionamide drug therapy (7-9). The objective of radioactive iodine-131 therapy is to destroy sufficient thyroid tissue to cure the patient's hyperthyroid state or to render the patient either euthyroid or hypothyroid. It is safe, effective, convenient and much less expensive than surgery. Most patients are treated as outpatients. Elderly or especially ill patients are treated with thionamides until euthyroid; then the thionamides are discontinued for 4-7 days while the beta-blocker is continued and the radioactive iodine is given (9,10). This reduces the amount of thyroid hormone released after the radiation induced injury and avoids the cardiovascular effects that may occur with a sudden surge of thyroid hormones (9,10). Younger patients are pretreated with higher doses of a beta-blocker. Within 6-12 weeks after the radioactive iodine treatments, the patients are generally euthyroid. If the patient is hyperthyroid 6 months after radioactive iodine-131, a second treatment is necessary.

We speculate that our patient went into thyroid storm because she was inadequately pre-treated. The patient was started on a low dose of propranolol rather than the recommended higher doses needed to suppress the cardiovascular and neurological effects of excessive thyroid hormone release. Kaplan et al. recommended long acting propranolol, 80 to 160 mg/day, or atenolol, 50 to 150 mg/day, but larger doses may be necessary in more severely symptomatic patients until the radioactive iodine takes effect (10). Becker and Hurley advise pre-treating older patients or severely ill patients with a thionamide and/or beta-blockers (7,9). Mechanick et al. recommend that all patients should be made euthyroid before radioactive iodine therapy (11) and Franklyn treats most patients with either antithyroid drugs or beta-blockers before therapy (8). Burch et al., however, reported recently that pretreatment with thionamides does not appear to protect against worsening thyrotoxicosis before radioactive iodine treatment and delays iodine-131 administration (12). Of interest to note in Burch's study is that most patients were given beta-blockers throughout the radioiodine therapy to achieve good symptom control (12).

Conclusion

Whether radioactive iodine induces thyroid storm through thyroid follicular disruption or due to excess iodine supply to a multinodular goiter via a Jod-Basedow phenomenon is speculative. It is difficult to ascertain whether our patient went into thyroid storm through either routes, but the importance of pretreatment and posttreatment after radioactive iodine is essential to prevent thyroid storm. In conclusion, we present an unusual cause of thyroid storm. Though rare, ED physicians should be aware that exogenous iodine and radioactive iodine administration is a potential cause of thyrotoxicosis and thyroid storm.

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