



THYROID STORM: A CASE REPORT

Emergency Medicine

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ABSTRACT

Background: Thyroid storm is an endocrine emergency which occurs in patients of hyperthyroidism. As diagnosis is primarily a clinical emergency physician should be aware of clinical presentation of thyroid storm. **Case presentation:** A young male patient who was known case of hyperthyroidism and family history of hypothyroidism with history of omission of antithyroid drug due to abdominal pain associated with previous enteric fever presented with clinical features of thyroid storm. A prompt clinical diagnosis was made and treated accordingly. **Summary:** Emergency physician should be aware of clinical presentation of thyroid storm as diagnosis is clinical and it is important for prompt management. Patient education about precipitating factors and compliance to drug is equally important for prevention of thyroid storm.

KEYWORDS

INTRODUCTION :

In 1928, Lahey gave this early description of thyroid storm: "The following remarks concerning thyroid crises and their management are prompted by the fact that so many patients are constantly being brought to the hospital in advanced stages of thyroid crisis and because so many of these inoperable patients die in spite of employment of every measure with which we are acquainted"^{1,2}. The estimated incidence of thyroid storm in Japan was reported to be 2.0 per million per year in a nationwide questionnaire survey from 2004 to 2008, conducted by the Japan Thyroid Association.³ The mortality of thyroid storm is in the range of 8% to 25%.^{3,4} Thyroid storm occurs in around 1-2% of patients with hyperthyroidism⁵. Nationwide survey by the Japan Thyroid Association suggested that shock, disseminated intravascular coagulation, and multiple organ failure were associated with mortality in patients with thyroid storm, but did not include microdata of individual patients³. Emergency physician should be aware that treatment should be started without awaiting for lab results as diagnosis is clinical.

Case presentation :

A 33 year old male patient presented to Emergency Department with chief complaint of difficulty in breathing since 2 hours. The patient also had complaints of high grade fever without chills, tremors, abdominal pain, vomiting, headache, dryness of mouth, difficulty in swallowing and throat pain since 2 days. The patient was admitted for treatment of enteric fever for 5 days 15 days ago. Patient was a known case of Hyperthyroidism for 6 months but he had stopped taking anti thyroid drugs since last 1 month because of fever and abdominal pain. Mother of the patient also had a history of hypothyroidism for which she was taking treatment regularly. On examination, patient had tachycardia with pulse rate of 130/min regular rhythm, wide pulse pressure (Blood Pressure -140/78 mm of hg), Respiratory rate of 24/min, normal oxygen saturation of 98% on room air, fine tremors of hand with warm and moist skin. He was conscious and oriented to time, place and person but was agitated and was also having hyperreflexia. His blood sugar level was 92 mg/dl. His electrocardiogram on admission revealed sinus tachycardia. Based on these clinical findings a strong suspicion of thyroid storm was made in the Emergency Department. Patient was resuscitated with fluid administration (bolus of 0.9% saline of 30 cc/kg) intravenous steroid (hydrocortisone 300 mg i.v. stat followed by 100 mg i.v. 8 hourly), beta blocker (Tab propranolol 40 mg every 4 hourly), propylthiouracil (Tab propylthiouracil 1000 mg followed by 250 mg every 4 hourly), lugol solution (6-10 drops every 6-8 hourly) and other supportive treatment.

His echocardiogram, lung ultrasound and chest x ray was essentially normal. His clinical findings were supported by TSH value of < 0.01, free T3 >20, free T4 more than 7.37 and Anti TPO antibody level :183.6. During course of hospitalization USG thyroid done which revealed enlargement of both lobes and isthmus, altered echo pattern and increased vascularity. The entire hospital stay was uneventful. Then the patient was discharged in a hemodynamically stable condition.



Images Showing Movement of Thyroid Gland During Deglutition

DISCUSSION:

Thyroid storm is an acute, severe, life-threatening hypermetabolic state caused either by excessive release of thyroid hormones causing adrenergic hyperactivity or an increased peripheral response to thyroid hormone in response to one or more precipitants⁶. It is usually precipitated by infection, trauma, surgery, excess of ingestion of thyroid hormones or withdrawal of anti thyroid drugs, diabetic ketoacidosis, hyperosmolar hyperglycemic coma, myocardial infarction, stroke, labor, eclampsia and radioactive iodine administration⁶. In this patient who is already a case of hyperthyroidism, previous infection due to enteric fever and noncompliance with antithyroid drugs may be the precipitating factor for the thyroid storm. In thyroid the main systems to concentrate on are the thermoregulatory system (rise in temperature), cardiovascular system (ranging from tachycardia to atrial fibrillation and congestive cardiac failure), CNS (ranging from being agitated to seizure), and the GI-hepatic system (ranging from nausea to vomiting and jaundice)⁶. Thyroid storm is a clinical diagnosis for patients with preexisting hyperthyroidism. Treatment is not delayed while confirming diagnosis by waiting for lab results. In determining whether or not a patient has thyroid storm⁶, Pathophysiology of thyroid storm is poorly understood. Several hypothesis include increase thyroid hormone release, absolute increase in free thyroid hormone levels, and increase in the receptors of thyroid hormones which leads to increase in the sympathetic activity which leads to sign and symptoms of thyroid storm.^{6,7,8} According to newer theories, thyroid storm results from allostatic failure in a situation where thyrotoxicosis hampers the development of non-thyroidal illness syndrome,⁷ which would help to save energy in critical illness and other situations of high metabolic demand.⁸

Fever is often present in thyroid storm. It may herald the onset of thyrotoxic crisis in previously uncomplicated disease⁶. Heightened sympathetic activity leads to tachycardia, arrhythmia, hypertension, wide pulse pressure and can lead to a high cardiac output induced cardiac failure⁶. CNS involvement includes agitation, delirium, anxiety, psychosis, or coma⁶. Gastrointestinal (GI) symptoms include nausea, vomiting, diarrhea, abdominal pain, intestinal obstruction, and acute hepatic failure⁶. A Japanese study found the CNS involvement to be a poor prognostic factor for increased mortality.³ This patient had tachycardia, wide pulse pressure, abdominal pain, agitation, tremors, and dyspnea. In 1993 Burch and Wartofsky Point Scale (BWPS) was introduced for thyroid storm diagnosis. The system is practical

because it is based on clinical and physical criteria and it is sensitive for thyroid storm, but it is not very specific.⁹ Apart from this scoring system, in 2012, Akamizu et al formulated diagnostic criteria for thyroid storm and clarified its clinical features, prognosis, and incidence based on nationwide surveys in Japan.² After this, the Japanese Thyroid Association proposed diagnostic criteria of TS1 and TS2 for thyroid storm, taking into account laboratory evidence of increased free thyroid hormones with any CNS symptoms and non-CNS symptoms such as fever, tachycardia, heart failure presentation, or GI-hepatic derangement manifestations.² Nevertheless, a Burch and Wartofsky Point Scale score ≥ 45 appears more sensitive than a Japanese Thyroid Association classification of TS1 or TS2 in detecting patients with a clinical symptoms.^{4,10}

Burch-Wartofsky Point Scale (BWPS)⁹

Temperature: 5 points per 1 F above 99 F (maximum 30 points)

- CNS dysfunction: 10 points for mild (agitation), 20 for moderate (delirium, psychosis or extreme lethargy), and 30 for severe (seizure or coma)
- Tachycardia: 5 (99-109), 10 (110 -119), 15 (120 -129), 20 (130 -139) and 25 (greater than 140)
- Presence of atrial fibrillation: 10
- Heart failure: 5 for mild (pedal edema), 10 for moderate (bi-basilar rales), 15 for severe (pulmonary edema)
- GI dysfunction: 10 for moderate (diarrhea, nausea/vomiting or abdominal pain) and 20 for severe (unexplained jaundice)
- Presence of Precipitating factor: 10 points

Diagnosis:

A total score of more than 45 is highly suggestive of thyroid storm, 25 to 44 supports the diagnosis, and less than 25 makes the diagnosis unlikely in this patient score is of 55.

Leucocytosis, hyperglycemia, pre renal azotemia, elevated liver enzymes, high total and free levels of thyroid hormones are common laboratory findings of thyroid storm.⁶ This patient had all of the above. Ecg findings in thyroid storm are of tachycardia. Atrial fibrillation is present in around 10-35 % of patients.¹¹ Echocardiography usually reveals increased Left ventricular contractility with dilated inferior vena cava.⁶

Treatment of thyroid storm⁶:

Treatment of thyroid storm is directed towards following aims 1. Inhibition of peripheral adrenergic effects: It is achieved by beta blockers like propranolol (Propranolol 0.5–1 milligrams IV over 10 minutes, then 1–2 milligrams every few hours adjusted to vital signs. For the less toxic patient, PO dose of 60–80 milligrams every 4 h) or esmolol (250–500 micrograms/kg IV load, then 50–100 micrograms/kg/min titrated doses to vital signs). Propranolol has advantage of inhibition of peripheral conversion of T4 to T3. In patients with absolute contraindications to beta blockers ganglion blockers like Reserpine or Guanethidine can be used.

2. Inhibition of new thyroid hormone synthesis :

It is achieved by Methimazole 20 milligrams every 6 h orally or propylthiouracil (PTU) a loading dose of 500–1000 milligrams given PO and followed by 250 milligrams every 4 h. Methimazole is avoided in pregnant women as it has teratogenic risk which is highest in first trimester. PTU is associated with severe and fatal hepatic dysfunction. PTU is strictly preferred only in the case of pregnant patients during the first trimester.

3. Inhibition of thyroid hormone release:

Lugol Iodine solution, potassium iodide, ipodate (Oragrafin®), or lithium carbonate can be given to stop thyroid hormone release. Nevertheless, iodine-containing solution should not be given to patients with iodine hypersensitivity, iodine overload, or iodine-induced hyperthyroidism or to those with amiodarone-induced thyrotoxicosis.

4. Preventing peripheral conversion of T4 to T3:

Glucocorticoids such as hydrocortisone or dexamethasone are essential in treatment by inhibiting peripheral conversion of T4 to T3. Glucocorticoid use in thyroid storm also improves survival rates.⁹

5. Preventing free thyroid hormones reabsorption:

Cholestyramine is used to inhibit thyroid hormone reabsorption by inhibiting enterohepatic circulation.

6. Supportive measures and control of precipitating factors.:

which includes maintenance of hydration, control of fever with paracetamol, antibiotics for infection, control of blood sugars in diabetic ketoacidosis and treatment of other precipitating factor according to the cause.

7. Definitive therapy :

like radioactive iodine therapy or surgery after control of hyperthyroidism.

8. Alternative therapy :

Plasmapheresis, charcoal hemoperfusion, resin hemoperfusion, and plasma exchange may be effectively used to rapidly reduce thyroid hormone levels in thyroid storm patients who respond poorly to traditional therapeutic measures.¹²

This patient was treated with all of above mentioned aims and discharged without adverse event.

CONCLUSION:

Thyroid storm is a life threatening health condition that is associated with untreated or undertreated hyperthyroidism which can lead to acute heart failure and pulmonary edema (3). The diagnosis of Thyroid Storm is clinical, so as an emergency physician it is very crucial to diagnose it in early stage and to start immediate treatment to reduce mortality. Patient education for compliance to treatment and precipitating factors are also important for prevention of future episode.

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