Thyroid storm following trauma: A rare case report

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ABSTRACT

Thyroid storm, a complication of hyperthyroidism, can present with features such as altered sensorium, fever, and tachycardia. Similar features are common in trauma patients and may mask any underlying hyperthyroidism. A thorough history, recognition of risk factors, and early analysis of thyroid function in patients with unresolving fever, altered sensorium, and tachycardia can circumvent unnecessary investigations and the high mortality associated with the missed diagnosis of thyroid storm.

Key words: *Hyperthyroidism, Thyroid crisis, Thyroid storm*

Thyroid storm, also known as thyroid crisis, is a rare complication of thyrotoxicosis. Despite the early diagnosis, the overall mortality rate remains high at 10–30% [1]. Four main features characterize the clinical scenario: Altered mental status, hyperpyrexia, tachycardia, and gastrointestinal dysfunction. Thyroid storm is typically precipitated by several concomitant events such as infection, iodine-containing contrast agent use, medication, pregnancy, surgery, and acute illness. Trauma is a rare precipitating factor [2].

Recognition of thyroid storm in a trauma patient is a difficult task, as its manifestations such as tachycardia and altered consciousness could be considered to be trauma related. Thorough patient history and awareness of the risk factors of thyroid storms may reduce the rate of misdiagnosis and prevent catastrophic outcomes. We report a case of thyroid storm occurring after a road accident, as the diagnosis was initially elusive in the face of clinical features being misinterpreted as trauma related.

CASE REPORT

A 36-year-old man, known case of chronic pancreatitis and a chronic smoker, presented to the emergency department after a road traffic accident. On arrival, the patient was extremely agitated and diaphoretic. During the primary survey, he was dyspneic, with respiratory rate of 32/min, heart rate of 160/min, and blood pressure of 100/70 mmHg. Glasgow Coma Scale score was 15, and his pupils were normal in size, equal, and reactive to light. The injuries noted were multiple abrasions over his face and all four limbs, and a deformity of both lower limbs and open left knee joint. There was no response to 1 L of the intravenous fluid bolus.

Computed tomography (CT) scan showed bilateral, multiple rib fractures with mild left hemothorax, posterior dislocation of the right hip, comminuted fracture of the right acetabulum and acetabular wall, right femoral head fracture, and the left femoral fracture. He underwent emergency debridement of both lower limbs, left distal femur articular reconstruction with minimal internal fixation, and knee spanning external fixator under general anesthesia.

Post-procedure, the patient was extubated and shifted to intensive care unit (ICU). He had continuous fever spikes and tachycardia and was empirically started on broad-spectrum antibiotics. He was reintubated in view of worsening hypoxemia and altered sensorium. Post-intubation, chest X-ray showed bilateral lung infiltrates. CT scan of the chest showed diffuse ground-glass opacities in bilateral lung fields suggestive of pulmonary edema or infective etiology (Fig. 1).

He was ventilated as per the acute respiratory distress syndrome net protocol. Despite all supportive measures and broad-spectrum antibiotics, he had persistent fever and tachycardia. As the initial cultures were negative, the thyroid profile was sent for as part of the work-up for non-infectious causes of fever. It showed thyroid-stimulating hormone (TSH) 0.00 µIU/ml (normal range 0.34– 5.60 µIU/ml) and total T4 of 16.38 µg/dl (normal range 4.6–11.2 µg/ dl) (Table 1).

Further, a history from the family revealed that the patient had palpitation, weight loss, and heat intolerance several weeks before the accident, which had not been evaluated. CT neck showed diffuse enlargement of the thyroid gland (Fig. 2). Thyroid storm was diagnosed using the Burch and Wartofsky score (Table 2): The patient had a score of 80 (temperature = 20, delirium = 20, precipitant history = 10, tachycardia = 15, and pulmonary edema = 15) and laboratory results.

The treatment was instituted according to the guidelines of the American Thyroid Association with intravenous hydrocortisone 100 mg 8th hourly, tablet propranolol 40 mg 6th hourly, and tablet

carbimazole 20 mg 6th hourly in addition to continued sedation, ventilation, and cooling using ice packs and a cooling blanket. The dose was adjusted according to hemodynamic condition and total T4 level. Percutaneous tracheostomy was done in view of the need for long-term ventilation and subsequent orthopedic surgeries. The patient's clinical status improved gradually. He underwent right hip open reduction, acetabular reconstruction, and definitive stabilization of distal femur under general anesthesia. He was slowly weaned off ventilator support and transferred to the general ward. The tracheostomy tube was downsized and subsequently decannulated. The patient was discharged with advice to follow-up with an endocrinologist.

DISCUSSION

Thyroid storm, which occurs in approximately 1–10% of inpatients with thyrotoxicosis, has been associated with diagnostic and therapeutic challenges in clinical practice since it was first characterized in 1926. It occurs more commonly in women and among patients with Graves' disease [3-5]. In the present case, there were no features of Graves' disease such as goiter, tremor, orbitopathy, acropachy, or dermopathy. Although the exact pathogenesis of thyroid storm is not definitive, numerous precipitating factors, namely, infection, major trauma, surgery, parturition, diabetic ketoacidosis, vascular accidents, non-compliance with thyroid medication, iodine exposure from radiocontrast dyes, amiodarone exposure, and emotional stress, have been identified [2]. Trauma as a cause has been sparsely reported. In a recent nationwide survey of Japanese hospitals, trauma accounted for 3.9% of cases of thyroid storm [6].

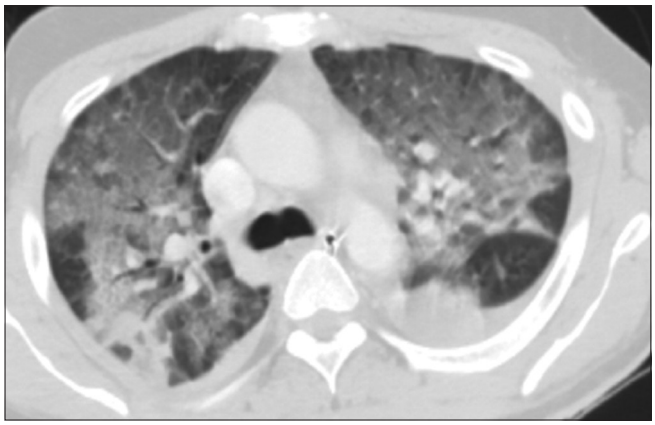

Figure 1: Computed tomography scan of the chest showing acute respiratory distress syndrome

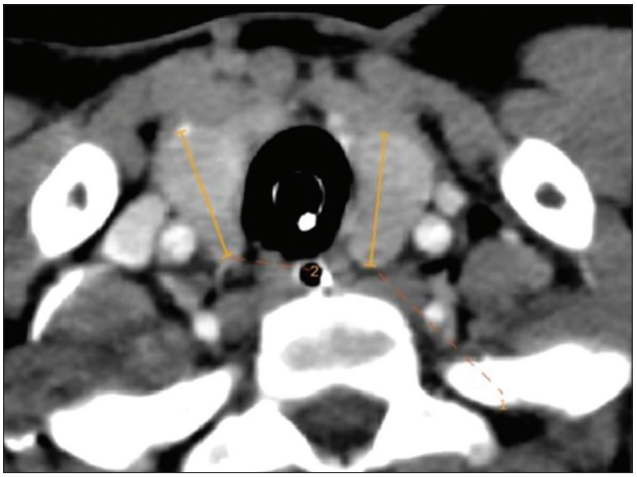

Figure 2: Computed tomography scan of the neck showing enlarged thyroid gland

The symptoms of thyroid storm, which include altered consciousness, tachycardia, and hypertension, may be confounding in cases of trauma [3]. A delay in the diagnosis of thyroid storm is potentially catastrophic, as even early diagnosed thyroid storm has an associated mortality rate of 10–30% [1].

The differentials to consider in thyroid storm are infection, sepsis, cocaine use, psychosis, pheochromocytoma, neuroleptic malignant syndrome, and hyperthermia [7]. The diagnosis of thyroid storm depends on clinical findings. In 1993, a landmark article by Burch and Wartofsky created a thyroid storm scoring system (Table 2). Each of the different signs and symptoms was given a numerical score [8]. A score of 45 or greater is highly suggestive of thyroid storm, a score of 25–44 is suggestive of an impending storm, and a score of <25 denotes that thyroid storm is unlikely. Although the scoring system is not extremely specific, its application in patients with post-traumatic thyroid storm seems to be helpful in making a diagnosis and providing further management.

Thyroid function tests in patients with post-traumatic thyroid storm have revealed indicators of hyperthyroidism, including low TSH (100%) and increased T4 (95.7%) and T3 (80.9%) levels [9]. There is no definitive serum T3 or T4 cutoff level to differentiate uncomplicated thyrotoxicosis from thyroid storm, but a complete evaluation of TSH, free T4, and total T3 levels may facilitate confirmation of the diagnosis. Because thyroid storm is a syndrome and the presentation so variable, there is no pathognomonic constellation of signs and symptoms. Serum total T3 levels may actually be within normal limits, presumably due to the superimposition of the "low T3 syndrome" caused by the underlying illness that precipitated the storm.

Trauma-induced thyroid storm is characterized by increased levels of cytokines, especially interleukin-6, causing impairment in the synthesis of albumin and thyroid-binding globulin in the liver, as well as increased concentration of circulating inhibitors. The consequences may include reduced protein binding, leading to increased levels of free thyroid hormone, activation of the adrenergic nervous system, and enhanced sensitivity of peripheral cellular responses to thyroid hormone [10].

Due to its high mortality rate, the treatment should start as soon as thyroid storm is suspected. Patients require admission

Table 1: Progress in thyroid function over the course in ICU

Thyroid hormone	Day 1	Day 4	Day 7	Day 10	Day 15
Total T4 (mcg/dl)	16.38	16.76	15.81	11.14	10.37
Total T3 (ng/ml)	1.03		0.75	0.68	
FREE T4 (ng/dl)	2.71				

ICU: Intensive care unit

Table 2: Burch-Wartofsky scale

Temperature (F)		Cardiovascular dysfunction	
99–99.9	5 points	Tachycardia (beats/min)	
100–100.9	10	99–109	5 points
101–101.9	15	110–119	10
102–102.9	20	120–129	15
103–103.9	25	130–139	20
≥104	30	≥140	25
Central nervous system effects		Atrial fibrillation	10
Absent	0	Heart failure	
Mild (agitation)	10	Mild (pedal edema)	5
Moderate (delirium, psychosis, and extreme lethargy)	20	Moderate (bibasilar rales)	10
Severe (seizure and coma)	30	Severe (pulmonary edema)	15
Gastrointestinal–hepatic dysfunction		Precipitant history	
Moderate (diarrhea, nausea/vomiting, and abdominal pain)	10	Positive	0
Severe (unexplained jaundice)	20	Negative	1

Total <25, storm unlikely; 25-45, impending storm; >45, thyroid storm

to an ICU for close monitoring. The medical management of thyroid storm has three objectives: Inhibiting the synthesis and release of thyroid hormone, inhibiting the peripheral effects of thyroid hormone, and increasing thyroid hormone clearance [2]. Thioamides, which stop new thyroid hormone production, include thiouracils (6-propyl-2-thiouracil) and imidazoles (methimazole and carbimazole), and are the first-line treatment for thyroid storm. The administration of iodine can block thyroid hormone release [8]. Lugol's solution and potassium iodide are drugs with proven efficacy in reducing thyroid hormone release.

Lithium may also be considered as a second-line agent to decrease thyroid hormone release from the gland and reduce the iodination of tyrosine residues. However, the mechanism is not well known, and the subsequent toxicity is complicated. Beta-blockers, corticosteroids, and digoxin are agents that block the peripheral effects of thyroid hormone. Propranolol is the most commonly used beta-blocker due to its non-selective beta-adrenergic antagonism, and because it has the advantages of intravenous administration and the ability to reduce the conversion of T4–T3.

Wilkinson reported a case of a 38-year-old woman who presented initially as the driver in a road traffic accident with a Glasgow Coma Score of 14, agitated, tachycardic, tachypneic, and hypertensive. The combination of an abdominal CT scan and clinical findings led to the decision to perform an emergency laparotomy. Her persisting symptoms and laboratory results revealed her to be manifesting acute thyroid storm, TSH of <0.10 IU, free T4 of 59.8 pmol, and free T3 of 20.20 pmol [11].

Yoshida reported a case of a 21-year-old woman presented to the emergency department complaining of head, neck, and abdominal pain after being assaulted. She denied a previous history or significant symptoms of hyperthyroidism. The physical examination was remarkable for lethargy, low-grade fever, tachycardia, facial abrasions, swelling of the anterior neck, and a tremor. A diagnosis of thyroid storm was made. An extensive work-up excluded other causes of the patient's tachycardia, altered mental status, and neck swelling. A review of the clinical features and management of thyroid storm is presented. Relatively, minor trauma can be a precipitating event for thyroid storm [12].

Sepsis was thought unlikely as our patient was fit and well before the accident with no identifiable focus of infection either during the initial evaluation or during his initial acute phase. There was no prior evidence of typical episodic hypertension or any other symptoms suggestive of pheochromocytoma either in his medical history or on presentation. In the case presented here, it appears that hitherto undiagnosed and untreated hyperthyroidism was responsible for the clinical picture. On the basis of this constellation of clinical and laboratory findings, the most likely diagnosis was thyroid storm, precipitated by trauma, in a patient with pre-existing hyperthyroidism.

CONCLUSION

Trauma is a rare cause of thyroid storm, and the diagnosis of thyroid storm may be difficult due to the overlap of many clinical findings between thyrotoxicosis (tachycardia, altered mental status, and abdominal pain) and trauma. The critical care physician should always consider a diagnosis of thyroid storm if the patient has a history of hyperthyroidism and trauma of moderate severity. The scoring system and laboratory data could help to diagnose and monitor the outcome of treatment.

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