Trauma Precipitating Thyroid Storm


Abstract
A 40 year old male victim of a road traffic accident presented to our emergency trauma services with multiple limb injuries and a Glasgow Coma Score (GCS) of 15/15. Soon after admission, he became confused, stuporous, febrile and tachycardic. A clinical diagnosis of thyrotoxic crisis precipitated by trauma was confirmed by relevant investigations, with appropriate therapeutic response. A review of the clinical features and management of this rare medical emergency, with only few cases reported worldwide, is presented.

Introduction
Thyroid storm - a dramatic exacerbation of pre-existing hyperthyroidism of sudden onset associated with hyperthermia, tachycardia and CNS symptomatology remains a life threatening disease.1 It is a catastrophic metabolic crisis with multisystem dysfunction. It is relatively rare, accounting for approximately 1.2% of hospital admissions for thyrotoxicosis.1 Thyroid storm precipitated by trauma is even rarer, with few reported cases in literature.1,2,3,4 Prompt recognition and aggressive, multifaceted therapeutic intervention as outlined herein are critical to obtaining a satisfactory outcome. We report this case both for its rarity and the invaluable clinical lessons imparted.

Case Report
A 40 year old man injured in a road traffic accident was admitted to the trauma ward of our tertiary care centre with multiple contusions and abrasions on all extremities, no obvious head injury and a GCS of 15. He was hemodynamically stable with pulse rate of 94/min and BP of 116/70 mm Hg. Complete blood count, blood glucose, electrolytes and renal function tests were all within normal limits. However, within a few hours, his sensorium deteriorated with increasing confusion and stupor. Body temperature rose to 38.4°C and pulse rate to 140/min. Urgent contrast enhanced CT brain was done to rule out concussion or intracranial bleed, but was absolutely normal. A medicine reference was sought for unexplained altered sensorium.

On enquiry, the spouse gave a history of progressively growing neck swelling over the last one year, with no associated complaints. Alcohol abuse was admitted; however, the patient had not binged in over two months.

On examination, the patient was stuporous, febrile and profusely diaphoretic despite ambient air conditioning. There was a staring look with extensive exposure keratoconjunctivitis. Heart rate was 144/min with bounding peripheral pulses. BP was 140/60 mm of Hg. A prominent asymmetric goitre (Fig. 1) was present with overlying erythema and a loud bruit on auscultation. There was no murmur or gallop. Crepitations were present in both lung bases. Pupils were equal and reacting. He moved all 4 limbs on painful stimulus. Both plantar responses were flexor and deep tendon reflexes were brisk. Chest X Ray was unremarkable; ECG showed sinus tachycardia. Abdominal ultrasound and 2D echocardiography were normal.

Thus, a diagnosis of thyroid storm precipitated by trauma was made. Serum T3, T4 and TSH levels were ordered and urgent therapy was instituted with intravenous hydrocortisone, propylthiouracil and propranolol tablets followed by Lugol's iodine through a nasogastric tube. Supportive management in the form of hydration under central venous pressure monitoring, cooling and eye care were meticulously provided.

By the third day of treatment, his heart rate was controlled at 70-80/min. Local warmth and intensity of the bruit diminished. Sensorium improved dramatically with the patient conscious and well oriented in time, place and person.

Reports: T3- 7 pg/ml (normal 1.4-4.4) T4 - 2.2 ng/dl (normal 0.8-2) TSH < 0.01 uIU/ml (normal 0.35-4.94) (suggesting T3 toxicosis)

On enquiry after recovery, the patient did give history of

Fig. 1: Prominent asymmetric midline neck swelling (toxic multinodular goitre) with exophthalmos, lid retraction and exposure conjunctivitis

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Thyrotoxicosis becomes storm are absent. To overcome this obstacle, Burch and Wartofsky developed a rating scale using criteria of body temperature, neurological effects, gastrointestinal and hepatic dysfunction, cardiovascular dysfunction and precipitant history. This point scale helps differentiate uncomplicated thyrotoxicosis (≤24) from impending thyroid storm (25-44), and established thyroid storm (≥45). Mortality rates of hospitalized patients have ranged from 20-30% even with the best treatment.

Management

Treatment of thyroid storm is multimodal and is best managed by the endocrinologist and medical intensivist. Supportive therapies such as intravenous hydration, oxygen, pressor agents (in the event of circulatory collapse), electrolyte correction, arrhythmia management, and aggressive reversal of hyperthermia should be delivered promptly. Medical therapy is directed at stabilizing the patient, correcting the hyperthyroid state, managing the systemic decompensation, and treating the underlying cause. In general, management of the hyperthyroid state can be accomplished by (1) reducing thyroid hormone production with propylthiouracil or methimazole, (2) inhibiting thyroid hormone release with iodine or lithium, and (3) reducing peripheral conversion of T₄ to T₃ with propylthiouracil, steroids, propranolol or iodate. It is essential that iodine therapy should not be administered until an effective blockade of new hormone synthesis has been established with antithyroid drugs (approximately one hour) as iodine will further fortify the thyroid hormone stores, thus increasing the risk of exacerbation of the toxic state. Plasma phaeohypha may be needed in unresponsive cases for the removal of excess circulating hormone. After the patient has been stabilized, definitive treatment in the form of RIA or surgery should be undertaken; until then, patients should be continued on antithyroid medications and a beta blocker.

Conclusion

Thus, a high index of suspicion, timely diagnosis and aggressive multipronged therapy help produce gratifying results in this catastrophic endocrine emergency with otherwise high mortality.

References