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Hyperthyroidism and Blunt Multi-System Trauma

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Abstract

We report a case of multi-system trauma after a motor vehicle collision with documented hyperthyroidism. Intensive care was required including a tube thoracostomy and a tracheostomy. Elevated thyroid function tests were noted and the patient responded to anti-thyroid medication. The patient was decannulated prior to discharge to home.

48 year-old male driver was involved in a rollover motor vehicle collision with ejection. He was intubated in the field and transported to a Level 1 trauma center. At the time of admission his blood pressure was 184/132 with a pulse of 118 and a respiratory rate of 24. The initial abdominal ultrasound was negative and the pelvis was stable on exam.

The chest x-ray revealed a moderate right pneumothorax with 1st and 2nd rib fractures. A right chest tube was inserted. An admission arterial blood gas revealed a PH of 7.11, PC02 of 63 and a Pa02 of 134 with a bicarbonate of 20 and a base deficit of 9. A CT survey revealed thyromegaly with surrounding blood and a spleen laceration with a blush. Intensive care unit observation ensued and no further therapy for the spleen injury was required. The patient remained hypertensive and tachycardic. Thyroid function tests were obtained. Free T4 and T3 were both elevated and TSH was very low. He appeared toxic and a presumptive diagnosis of thyrotoxicosis was made with impending thyroid storm.

Methimazole was initiated along with beta-blockade (propranolol). The thyroid function tests remained elevated and he was switched to Propylthiouracil. His vital signs stabilized and he was eventually extubated. Post-extubation stridor developed and a tracheostomy was performed. He was weaned and eventually decannulated. He was discharged to home in satisfactory condition on daily anti-thyroid medication.

Discusssion

The clinical presentation of any trauma victim mandates that the Advanced Trauma Life Support principles are followed [1]. The airway is secured, breathing assessed along with circulation and balanced resuscitation is initiated. All acute life-threatening injuries are detected during the primary survey and a complete head to toe evaluation is performed during the secondary survey. For the patient described above, the airway was secured in the field with an endotracheal tube. A right pneumothorax was detected on the primary survey and treated with a chest tube. Profound acidosis (respiratory and metabolic) was evident and hypertension and tachycardia were noted on admission. Trauma patients may be hypertensive because of pain and pre-existing essential hypertension that has been poorly controlled. If they are hypotensive on admission it is most often related to blood loss or profound tissue injury. In the patient described above the spleen injury was a potential site of hemorrhage but proved to be clinically inconsequential with close monitoring. No transfusions were required during the monitoring phase. Hyperthyroidism was suspected based on the persistent tachycardia and the thyroid enlargement noted on initial imaging. Thyroid function tests confirmed the over activity and appropriate treatment was implemented. Graves disease is the most common cause of hyperthyroidism overall. Toxic multinodular goiter and toxic adenoma are additional causes. Amiodarone has been recognized as a cause of hyperthyroidism in populations that are iodine deficient. Graves disease usually presents at a young age (between 20 and 40 years) in a typical pattern including heat intolerance, agitation, palpitations, tachycardia, hypertension and often altered mentation [2]. Exophthalmos may be present. Increased thyroid hormone production is the hallmark. Thyroid stimulating hormone is suppressed. Free T4 is elevated and Free T3 may be increased as well. Thyrotropin

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receptor antibodies (Trab) act as a TSH receptor agonist. Trab can be used to monitor progress and possibly predict prognosis regarding remission. The principles of clinical management include; decreasing thyroid hormone production and blocking the adrenergic effects of thyroid hormone excess. Methimazole is a mainstay in this regard along with propylthiouracil. Methimazole inhibits the conjugation

of monotiodtyrosine with di-iodotyrosine to form thyroxine. This shuts down synthesis. When combined with beta blockade, usually in the form of propranolol, clinical improvement is often seen. Propylthiouracil offers the advantage of inhibiting the conversion ofT4 to T3 peripherally [3].

The patient described was started on methimazole but rapid clinical improvement did not occur and the thyroid function tests did not decrease. Once he was switched to propylthiouracil, clinical improvement ensued. Proopylthiouracil may be preferred in the management of patients with thyroid storm because of the peripheral effects.

Thyroid storm can be defined with the Burch-Wartofsky point scale [4]. The seven domains of this scale define the clinical presentation. These domains include thermoregulatory dysfunction, cardiovascular manifestations — tachycardia, the presence of atrial fibrillation, the presence of congestive heart failure, GI-hepatic dysfunction, central nervous system disturbance and precipitating history. If the total score is above 45 points — thyroid storm is present.

Table 1: Burch and Wartofsky's scoring system.

Pirameters	Scoring sirstem
Thermoregulatory dysfunction	
Oral Temperature (F)	
99-99.9	5
100-100.9	10
101-101.9	1S
102-102.9	20
103-103.9	25
104	30
Cardiovascular dysfunction	
Tachycardia	
90-109	5
110-119	10
120-129	15
130-139	20
>140	25
Congestive heart failure	
Absent	0
Mild (pedal edema)	5
Moderate (bibasal rales)	10
Severe (pulmonary oedema)	15
Atrial fibrillation	
Absent	0
Present	10
Central nervous system symptoms	
Absent	0
Mild agitation	10
Moderate (Delirium, psychosis, extreme lethargy)	20
Severe (Seizure, coma)	30
Gastrointestinal /hepatic dysfunction	
Absent	0
Moderate (Diarrhea, nausea, vomiting, abdominal pain)	10
Severe (Unexplained jaundice)	20
Precipitating event	
Absent	0
Present	10

Burch HB, Wartosfsky L. Endocrinol Metab Clinics North America. 1993; 22: 263-277.

25-44 points reflects impending storm and less than 2S points means storm is unlikely (Table 1). Precipitating history may include abrupt cessation of anti-thyroid drug therapy, thyroidectomy or non-thyroidal surgery in a patient with undiagnosed hyperthyroidism. For the patient described, trauma may have been a precipitating cause. His clinical manifestations included; marked tachycardia, hypertension and altered mental state. Impending storm was likely. One can only speculate if the hyperthyroidism in this case contributed to the motor vehicle collision.

There was no history given of prior anti-thyroid medication and no previous history of hyperthyroidism obtained from the patient or his family.

The clinical approach must be based on system support. Fever is controlled with acetaminophen. Other agents like aspirin should be avoided as they may contribute to increased thyroid hormone levels by impacting the binding to thyroid binding globulin [5]. As noted above anti-thyroid medications should be administered immediately. Potassium iodide may be needed to decrease the oxidation of iodine and its' incorporation into thyroglobulin. Corticosteroids should be utilized to decrease thyroid hormone production and to address the presence of relative adrenal insufficiency. Hydrocortisone in a dose of 300 mg per day or dexamethasone in a dose of 8 mg per day is recommended. Beta- blockade is essential and a selective blocker that is rapid acting like esmolol may be needed acutely [6]. Clinical monitoring in the intensive care unit is essential.

For those patients unable to take anti-thyroid medications because of allergies or severe side effects, cholestyramine may offer a way to interfere with enterohepatic circulation and decrease thyroid hormone levels. 4 grams in three or four divided doses is recommended [7,8].

Patients in coma will need airway protection and mechanical ventilation until the hypermetabolic state has resolved. They must be assessed for infection as this may be the precipitating factor.

For those patients in cardiogenic shock, vasopressor support with dobutamine, norepinephrine or additional agents may be needed. Hemodynamic monitoring with an arterial line and central line should be initiated. Echocardiography will be helpful and serial measurements of ventricular function are recommended.

Cardiovascular collapse may mandate extracorporeal membrane oxygenation therapy. This bridge technique with or without Intra-aortic balloon pump support buys precious time to allow the anti-thyroid medications to work and the heart to stabilize [9,10].

Renal replacement therapy may be required if the acute kidney injury related to low flow occurs. For those patients who fail to respond within a 48-hour period therapeutic plasmapheresis may hold some promise [11-13]. Most of the data is retrospective but removing thyroid hormone and cytokines using this technique may provide an effective treatment in selected patients. Circulating hormone levels drop and patients can then safely proceed to total thyroidectomy [13].

Mortality remains significant, varying from 17-30%. From a nationwide Japanese study of 3S6 patients with thyroid storm there were several factors associated with increased mortality including: a pulse rate above 150 and the presence of atrial fibrillation. Multi-organ failure and congestive heart failure were the most common causes of death [7]. A French multi-center long term 17- year retrospective analysis of 92 patients with thyroid storm revealed that cardiogenic shock was a powerful predictor of poor outcome [10].

A high index of suspicion for unusual causes of tachycardia must be maintained during the care of any multi-system trauma victim. Unexplained tachycardia and signs of catecholamine excess should raise the issue of hyperthyroidism. Aggressive antithyroid medication along with beta-blockade should achieve control in the vast majority of these patients.

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