Case Report

Trauma triggering thyrotoxic crisis with lactic acidosis

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ABSTRACT

Thyrotoxic crisis (TC) is defined as a life-threatening exacerbation of the hyperthyroid state that causes multiple autonomic and metabolic disturbances. It is considered to be an endocrine emergency that must be urgently diagnosed and treated. We describe a case of TC precipitated by trauma with a resultant lactic acidosis. The patient is a 24-year-old male with a history of hyperthyroidism who presented to the emergency department following a motor vehicle accident. The patient was initially tachycardic and hypertensive, however, was afebrile. Initial laboratory analysis showed an anion gap of 26, lactic acid 7.6, free T4 5.61 and thyroid stimulating hormone <0.015. A diagnosis of TC was made, and he was treated with intravenous fluids, propranolol, and methimazole with improvement of tachycardia and lactic acidosis. We discuss the features of this case, which reviews the presentations of TC as well as its metabolic sequelae.

Key Words: Lactic acidosis, thyroid storm, thyrotoxic crisis, trauma

INTRODUCTION

Thyrotoxic crisis (TC) is an emergent and life-threatening exacerbation of thyrotoxicosis, which requires prompt diagnosis and treatment. This can be challenging as TC can have atypical presentations. We report a patient who developed TC as a result of a motor vehicle accident (MVA) and consequently developed an anion gap metabolic acidosis due to high serum lactate. On review of the current literature, there are case reports of TC with resultant lactic acidosis and multisystem organ failure as well as case reports of trauma precipitating TC. However, to our knowledge, there is not a case of trauma precipitating TC with resultant lactic acidosis.

CASE REPORT

A 24-year-old male with a past medical history of hyperthyroidism, noncompliant with methimazole treatment, presented as a patient to our level I trauma facility. He was a restrained driver in a rollover MVA with the unknown loss of consciousness. He self-extricated from the wreckage and was ambulatory at the scene. His only complaints were right lower quadrant abdominal pain and right wrist pain. The patient reported consuming ethanol only. His initial vitals per emergency medical services demonstrated heart rate 170, blood pressure 156/78, without a documented temperature. His initial vitals upon arrival demonstrated a heart rate of 163, blood pressure of 153/84, and temperature of 37.3°C.

Upon physical examination, Glasgow Coma Score was 15. He was anxious with a fine tremor. Other pertinent findings included a hematoma over the left eye, seat belt sign to the left chest not involving the neck, right-sided abdominal tenderness to palpation, left hand abrasions, and right wrist pain without deformity on inspection. Notably, there were no neck abrasions or contusions, and there was no goiter present. A focused abdominal sonography for trauma exam was negative. Head, maxillofacial, chest, abdomen, and pelvis computed tomography scans were all negative for acute traumatic abnormalities. Plain radiographs of the extremities were negative for acute osseous abnormalities as well.

The patient was resuscitated with 2 L of normal saline. He was administered intravenous (IV) lorazepam 2 mg and fentanyl 50 mcg for agitation and pain control. Complete blood count, complete metabolic panel, lactic acid, thyroid studies, ethanol level, and rapid urine drug screen laboratory testing were performed. Laboratory results were as follows: sodium 149, potassium 3.4, chloride 108, carbon dioxide 15, anion gap 26,
BUN 11, creatinine 0.51, aspartate transaminase (AST) 43, alanine transaminase (ALT) 58, lactic acid 7.6, free T4 5.61, and thyroid stimulating hormone <0.015. Rapid urine drug screen was positive for cannabinoids, and ethanol level was 101. Serum osmolality, ethylene glycol, and methanol levels were ordered secondary to the anion gap metabolic acidosis that was noted. Serum osmolality was 288 with an osmolar gap of ~6 and both ethylene glycol and methanol results were negative.

The patient remained hypertensive and tachycardic. Due to his history of hyperthyroidism with medication noncompliance TC was suspected. The patient was given methimazole 5 mg, propranolol 1 mg injection, fluid resuscitation was continued and the patient was admitted to the internal medicine service.

After 3 L of normal saline, the patient’s lactic acidosis improved to 0.8 7 h following his initial evaluation. Additional repeat laboratory findings at this time were as follows: Sodium 143, chloride 109, CO$_2$ 21, anion gap 13, AST 24, and ALT 50. Blood glucose ranged from 102 to 85 during hospitalization. After 24 h, the initial tachycardia and hypertension had resolved. The patient was continued on methimazole 5 mg 3 times daily and propranolol 10 mg 3 times daily. His blood cultures were negative for growth. He was instructed to follow-up with endocrinology as an outpatient and was discharged after 48 h in stable condition. He did not follow-up with endocrinology, however, was seen again in the emergency department 6 months later again with TC, this time secondary to tonsillitis as the patient remained medication noncompliant. He has still yet to follow-up with endocrinology clinic.

DISCUSSION

Thyrotoxic crisis is defined as a life-threatening exacerbation of the hyperthyroid state that causes multiple autonomic and metabolic disturbances. It is considered to be an endocrine emergency that must be urgently treated. Standard treatment involves supportive care, inhibition of thyroid hormone release, inhibition of new hormone production, peripheral β blockade, and prevention of the conversion of T$_4$ to T$_3$. This is accomplished by IV fluid resuscitation with dextrose, thioamides either propylthiouracil (PTU) or methimazole to decrease new hormone release, followed by iodine to stop new hormone synthesis. It is important to remember that either PTU or methimazole must be given prior to iodine in order to avoid stimulation of new thyroid hormone synthesis that can occur if iodine is given initially. Next, in order to prevent conversion of T$_4$ to T$_3$, glucocorticoids are an essential aspect of treatment followed by β adrenergic receptor blockade for which propranolol is most often used as it also causes some decrease in the conversion of T$_4$ to T$_3$.\[1\]

The thyrotoxic crisis is frequently precipitated by physical stressors such as infection, surgery, medication noncompliance and trauma.\[2\] Presentation of TC can be varied and, therefore, difficult to diagnose especially in a trauma patient who is likely to be tachycardic and may also have altered mental status.\[3\] The trauma patient may also have been consuming sympathomimetic drugs causing a very similar clinical picture and further complicating and delaying the diagnosis of TC. In addition, even without trauma as a precipitating event the presentation of TC can easily be confused with that of the septic patient. A patient may present with lactic acidosis as well as multiple organ dysfunction that is eventually attributed to TC as opposed to infection with resultant sepsis.\[4,6\] Although the diagnosis is not always straightforward, timely diagnosis is important since mortality from TC has been reported to be between 10% and 30%.\[4\]

On review of the current literature, there are case reports of TC with resultant lactic acidosis and multisystem organ failure as well as case reports of trauma precipitating TC. However, to our knowledge, there is not a case of trauma precipitating TC with resultant lactic acidosis. The case reports of patients with TC that developed a lactic acidosis presented with a severe sepsis picture that required Intensive Care Unit management.\[8,4\] They had multiple organ failure that resolved following TC treatment. It is hypothesized that the lactic acidosis was secondary to TC causing increased lactate production secondary to increased basal metabolic rate and decreased hepatic lactic acid clearance.\[5,6\]

Concerning TC precipitated by trauma, these patients presented status post trauma and MVA, respectively; both patients had TC.\[5,7\] These cases highlight the difficulty in differentiating symptoms of a thyrotoxic state from symptoms of head trauma and abdominal trauma as tachycardia and altered mental status can be observed in hypovolemia, traumatic brain injury, drug abuse, etc. In addition, a goiter can easily be mistaken for a neck hematoma in the trauma setting. Considering these factors, TC in a trauma patient is a very difficult diagnosis to make.

Upon further evaluation of this case, a thorough examination of the chart was performed to investigate if there was an additional or alternate cause of the lactic acidosis in this patient. The lactic acidosis, in this case, is hypothesized to be acute secondary to Type A lactic acidosis due to tissue hypoxia from increased basal metabolic rate causing increased oxygen utilization therefore elevating lactate production. Type B causes of lactic acidosis include diabetes, renal insufficiency, liver disease, sepsis, medications such as biguanides and salicylate as well as toxic alcohols such as methanol and ethylene glycol. In our patient, there was not an additional or alternate cause of the patient’s lactic acidosis.

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