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Case Report: Thyrotoxic Hypokalemic Periodic Paralysis

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Abstract:

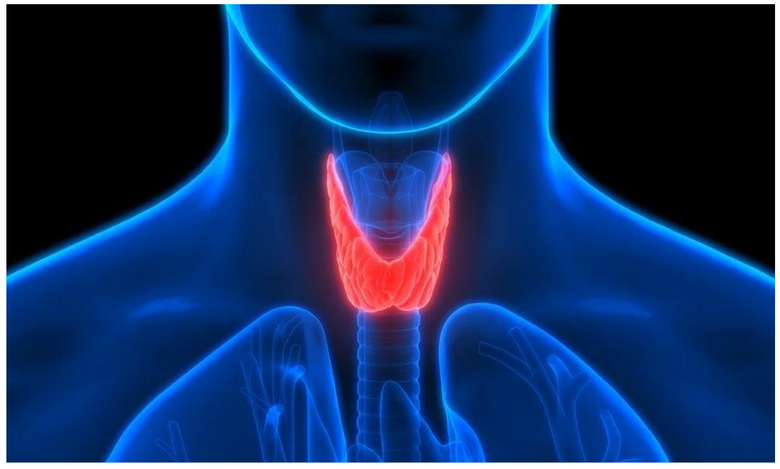
Thyrotoxic crisis, or thyroid storm is an acute, life threatening event caused by hyperthyroidism with mortality up to 8-25% that can present with multi-system organ involvement. It is a rare, but well studied complication in the emergency department but can lead to further, less common complications. In this study we will discuss a case of thyrotoxic hypokalemic periodic paralysis. Thyrotoxic hypokalemic periodic paralysis (TPP) most commonly affects Asian men. The key features of the syndrome include acute onset of hypokalemia and paralysis. The hypokalemic aspect of the disorder is secondary to the shift intracellularly by thyroid hormones' sensitization of Na+/K+-ATPase as opposed to body's depletion of potassium. TPP has an incidence of 2% in patients with thyrotoxicosis of any cause. The paralysis occurs because a large majority of the body's potassium is located in skeletal muscle. When the potassium moves intracellularly the muscles are unable to contract in their normal fashion.

Case Presentation:

A 38 year old Asian male presented to the emergency department for paralysis of bilateral lower extremities and palpitations that started about an hour prior to arrival to the ER. The patient explained that he got up and could not use his lower extremities at all so was attempting to pull himself across the floor but his arms started to go weak as well. The patient's only medical history was chronic lower back pain. He was also experiencing palpitations that started around the same time without associated shortness of breath or pain. His vital signs when he first arrived included a blood pressure 135/84 in the right arm, temperature 98.3F, heart rate 146 bpm, respiratory rate 29, and pulse oximetry 99%. An EKG was immediately performed which showed the patient was in atrial fibrillation.

A physical exam was performed which showed a young, healthy appearing male but appeared very anxious. His bilateral lower extremity strength was 2/5, 3/5 strength on the left upper extremity and 4/5 strength in the right upper extremity. He also had significant sensory deficit to bilateral lower extremities. He was alert and oriented with no further focal deficits. He had chronic back pain that he stated was his baseline, with no reproducible back or leg pain. He had no urinary incontinence or retention and no fevers, chills or midline tenderness. He denied any inciting event or recent trauma.

The patient had a mildly elevated white blood cell count at 11.2 B/L but CBC was otherwise normal. His high sensitivity troponin was 19, potassium was 2.8 mmol/L, and TSH was <0.01ulU/mL. His lab work was otherwise unremarkable. We had the T3 and T4 sent off and were pending. Due to the new weakness we did get a CT head and CTA head and neck, both of which were negative.



Case continued:

We were able to make the diagnosis relatively quickly after the patient arrived in the emergency department and we started treating as soon as possible after confirming our diagnosis with endocrinology. He was given 40mEq of oral potassium as well as 20mEq of KCl intravenous. He was given 60mg oral propranolol and 20 mg methimazole. These medications together in a short time span improved his paralysis and his heart rate normalized.
The patient arrived in the ER at just before 2am and was discharged the following afternoon at which time his symptoms had completely resolved. The patients total T3 was 446 ng/dL and free T4 was 4.8 ng/dL. The patient was ultimately diagnosed with Grave's disease and had a total thyroidectomy a few months later.

Discussion:

While thyroid disorders are relatively common, the severe complications of them are not frequently observed. It is important to be on the lookout for these extreme cases, particularly in a young patient with a constellation of seemingly unrelated symptoms. Thyroid storm is the better known complication of hyperthyroidism and is most common in patients with Grave's disease (2). Due to the high mortality, it is very important to recognize early and start aggressive treatment. TPP is a complication that presents with acute proximal but progressive symmetric lower extremity weakness. When the disease progresses it can involve all limbs and becomes life threatening if it involves the diaphragm and respiratory muscles (3). The incidence of TPP is much higher in the Asian population with up to 1.9% of thyrotoxic patients, compared with incidence in North American thyrotoxic patients which ranges between 0.1-0.2%. Though in general females are at higher risk of thyrotoxic events, TPP specifically is much more common in males. The incidence in Japanese thyrotoxic patients with TPP was 4.3% in Asian males compared to 0.04% females (3).

Discussion Continued:

The exact mechanism behind TPP is not entirely known however it is thought to involve intracellular shift of potassium by the thyroid hormones sensitization of the Na/K-ATPase. Because skeletal muscle holds a significant portion of the total body potassium it is necessary in order to maintain potassium homeostasis extracellularly and with significant drops in potassium extracellularly, muscles are unable to function normally. There is thought to also be a component of hyperinsulinemia contributing to the drop in extracellular potassium. (3). The presentation of TPP involves acute onset of proximal muscle weakness with myalgias. The weakness is more common in the lower extremities than upper extremities. Patients will frequently present with significant weakness prior to showing symptoms of hyperthyroidism which frequently include palpitations, tachycardia, and visual changes. Complications to be aware of in these patients includes rhabdomyolysis, ectopic heart beats and decreased or lack of reflexes. It is most common to have these episodes of paralysis in the early morning and after high amounts of carbohydrates.
In treating patients with TPP, the primary goal is to replete the potassium and reduce the thyroid hormones. Treatment suggests 30mEq of potassium orally every two hours until recovery starts. Non-selective beta blockers are helpful in decreasing the shift intracellularly of phosphate and potassium (3). If patients are unresponsive to potassium 1mg of propranolol can be given every 10 minutes up to 3 doses (3).

Conclusion:

Thyrotoxicosis is a life threatening emergency caused by a rapid rise in thyroid levels. While typical complications to look out for include elevated heart rate, heat intolerance, and tremors, it is important to be on the look out for less common presentations as well (4). When someone presents with TPP there frequently will not yet be symptoms of thyrotoxicosis so in a young patient it is important to keep this on your differential.

TPP is caused by intracellular shift of potassium from thyroid hormones so can occur before the thyroid levels come get to a toxic level which is why frequently patients present with paralysis prior to thyrotoxicosis. TPP is a life threatening emergency but with proper treatment is very treatable if diagnosed early.

References:

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