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CASE REPORT

Thyrotoxic periodic paralysis after urethral dilatation

Moayyad Alziadat, Mourad Ismail

Department of Critical Care Medicine, St Joseph's University Medical Center, Paterson, New Jersey, USA

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare and serious manifestation of thyrotoxicosis that causes flaccid paralysis. In severe cases, it can be life-threatening due to respiratory failure and cardiac arrhythmias. TPP is due to increased sodium/potassium ATPase activity during thyrotoxic states, which is due to mutations encoding potassium channels. It is precipitated by situations that cause a surge in catecholamines, insulin, or both. It can be treated with potassium supplementation and nonselective beta blockers, and it can be prevented by establishing euthyroid state. With the increasing numbers of outpatient procedures performed nowadays and the stress related to these procedures, patients with TPP may develop paralysis after these procedures, so clinicians should be aware of this condition and the importance of identifying it in patients presenting with flaccid paralysis.

Key words: Hypokalemia, paralysis, periodic, thyrotoxic, thyrotoxicosis

Key messages: Thyrotoxic periodic paralysis is a rare but serious condition that should be considered in the differential diagnosis of patients presenting with flaccid paralysis, as prompt identification and treatment will prevent serious complications and can be curative.

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a rare and serious manifestation of thyrotoxicosis, more commonly seen in Asian patients but also reported in patients from other races.[1-3]

We are reporting a case of TPP precipitated by a urological procedure, which to the best of our knowledge has not been reported before.

CASE HISTORY

A 44-year-old Hispanic male presented to our emergency department (ED), unable to move any of his extremities or his neck. The patient has urethral stricture for which he undergoes outpatient urethral dilatation on a regular basis uneventfully. He had a urethral dilation procedure on the day before his presentation, and received one dose of ciprofloxacin perioperatively. Around 10 pm, he noticed mild weakness in all his extremities. The next morning, the weakness got worse to the extent that he was unable to move any of his extremities. He denied any trauma or falls before these symptoms, any symptoms of hyperthyroidism, any urinary or stool incontinence, ingestion of high-carbohydrate meal, exercise, or alcohol consumption. He denied experiencing similar symptoms in the past, any family history of similar symptoms or thyroid disease, or taking any medications on a regular basis.

On presentation, the patient's blood pressure was 135/98 mm Hg, heart rate was 105 beats per minute, or beats/min, respiratory rate was 18 breaths per minute or breath/min, and temperature was 37.5°C. His physical exam was impressive for 0/5 strength, decrease tone, and absent deep tendon reflexes in all four extremities, with normal sensation.

Address for correspondence: Dr. Moayyad Alziadat, Pulmonary and Critical Care Department, St Joseph's University Medical Center, Paterson, New Jersey 07503, USA.
E-mail: muayad_ziadat@hotmail.com

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Electrocardiogram showed sinus rhythm, first degree atrioventricular block (AV) block with U waves, and prolonged QT interval.

Blood work was significant for potassium level of 1.7 mEq/L (reference range, 3.5–5.0 mEq/L), phosphorus level of 1.4 mg/dL (reference range, 2.5–5.0 mg/dL), magnesium level of 1.6 mg/dL (reference range, 1.7–2.5 mg/dL), serum lactate level of 3.5 mmol/L (reference range, 0.5–2.2 mmol/L), troponin I of 0.879 ng/mL (reference range, 0–0.030 ng/mL), thyroid stimulating hormone less than 0.010 micro IU/mL (reference range, 0.45–5.33 micro IU/mL), free T4 of 2.96 ng/dL (reference range, 0.61–1.12 ng/dL) and free T3 of 6.1 pg/mL (reference range, 2.3–4.2 pg/mL), thyroglobulin antibodies of 219.6 IU/mL (reference range, 0–0.9 IU/mL), TPO antibodies of 247 IU/mL (reference range, 0–34 IU/mL), and TSI of 3.53 IU/mL (reference range, 0–0.55 IU/mL).

Ultrasound of the thyroid gland showed heterogeneous thyroid gland with increased vascularity.

The patient was diagnosed with TPP secondary to Graves' disease. He was given intravenously a total of 80 mEq of potassium chloride, 15 mmol of potassium phosphate, and 2 g of magnesium sulfate. The patient was given 40 mg of propranolol and 60 mg of methimazole orally, and then maintained on 10 mg three times daily for each.

Symptoms of the patient improved gradually while receiving potassium replacement, he was initially able to move his fingers, and by the next day, he had complete resolution of all his symptoms.

The patient was discharged with follow-up with the endocrinologist office to arrange for radioactive iodine ablation as an outpatient.

DISCUSSION

TPP is a serious manifestation of thyrotoxicosis. It causes paralysis, usually of the lower extremities, but can ascend and cause respiratory failure requiring mechanical ventilation support. It can also result in cardiac arrhythmias though less common.[1,4]

This condition is frequently confused in the United States with the more commonly known condition of hypokalemic periodic paralysis because it is more common in the Caucasian population, whereas TPP is more commonly seen in Asian population. However, TPP has been described in patients of other races. Also, hypokalemic periodic paralysis is an autosomal-dominant condition and runs in families, whereas TPP is seen in sporadic cases.[3,5]

TPP is usually seen in patients with mild or subclinical thyrotoxicosis, making the diagnosis challenging without high index of suspicion. The degree of paralysis is related to the degree of hypokalemia rather than the severity of thyrotoxicosis.[1,3]

Unlike thyrotoxicosis, TPP is seen much more commonly in males.[1,5,6] Attacks are precipitated by high-carbohydrate meals, rest after strenuous exercise, trauma, exposure to cold, emotional stress, infection, alcohol ingestion, menses, and drugs such as diuretics, insulin, or steroids.[5,6] Interestingly, in our patient, the attack was precipitated by a urologic procedure, probably causing increased catecholamines levels due to stress.

The paralysis usually starts in the lower limbs, proximal muscles more than the distal, lasting few hours to few days. It rarely affects ocular, bulbar, or respiratory muscles, but there have been reports of ventilatory impairment.[1,4]

Patients also develop hypophosphatemia and hypomagnesemia; both are caused by intracellular shift due to endogenous catecholamine surge. In two-thirds of patients, creatine phosphokinase is elevated, especially if precipitated by exercise, rarely complicated by rhabdomyolysis.[3]

Electrocardiographic changes in patients with TPP, include tachycardia, increased P-wave amplitude, prolonged PR interval, widened QRS complexes, and decreased T-wave amplitude and U waves. More serious changes include AV blocks, atrial fibrillation, ventricular fibrillation, and asystole.[1]

The exact mechanism of TPP is not well understood, but it is believed to be due to a subclinical channelopathy that manifests during thyrotoxic states; it is known that thyroid hormone stimulates sodium–potassium ATPase activity, resulting in increased potassium shift intracellularly. Thyroid hormone also sensitizes ATPase receptors to epinephrine and insulin and increases the expression of beta adrenergic receptors, resulting in further shifting of potassium intracellularly.[1,3,5] Actually, in patients with TPP, the urinary and fecal potassium loss is normal if not low. The acid–base balance state in these patients is also normal.[3] The increased sensitivity to insulin in these patients, explains the precipitation of attacks with carbohydrate-rich meals with the resulting hyperinsulinemic state. The enhanced ATPase activity in patients with TPP during thyrotoxic state is much more prominent when compared to patients with

thyrotoxicosis without TPP, and the normalization of the ATPase activity during euthyroid state supports the theory that patients with TPP have an abnormally exaggerated thyroid hormone–mediated ATPase activity.[3]

In fact, mutation in the gene encoding for Kir2.6, a skeletal muscle potassium channel that is transcriptionally regulated by the thyroid hormone, was noted in some TPP cases.[7]

In other TPP cases, a different mutation causing decreased expression of *KCNJ2* (Kir 2.1) during thyrotoxic state, which normalizes during euthyroid states, was identified.[8]

Treatment of TPP includes conservative potassium chloride supplementation to prevent cardiac complications without causing rebound hyperkalemia after resolution of the attack and shift of the intracellular potassium extracellularly.[1,3,5]

High-dose propranolol is also used to abort and prevent attacks by preventing intracellular potassium shift.[3]

Patients should avoid precipitating factors until euthyroid state is achieved, which is curative. This can be achieved using antithyroid medications, radioactive iodine therapy, or surgically.[1,3,5]

In conclusion, TPP should be considered in the differential diagnosis for any patient presenting with symmetrical flaccid paralysis, as prompt identification and appropriate treatment can be curative and can prevent serious complications.

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Conflicts of interest
There are no conflicts of interest.

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