

CASE REPORT

General Medicine

“I’m so weak I feel paralyzed. Can you help me?” A case on thyrotoxic periodic paralysis

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Abstract

Thyrotoxic periodic paralysis (PP) is a rare condition characterized by muscle weakness in the form of hyperthyroidism and hypokalemia. We present the case of a 38-year-old Hispanic male with PP that had been occurring over the course of 4 years. After several emergency department visits, the cause of his symptoms was still unknown. Initial laboratory testing revealed critical hypokalemia with associated electrocardiogram (ECG) changes. Further laboratory evaluation revealed hyperthyroidism, and the diagnosis of thyrotoxic PP was made. The patient received potassium supplementation with complete resolution of his symptoms and resolution of pathologic ECG changes. Our patient was started on propranolol and methimazole to treat his hyperthyroidism. Not well documented in previous reports, we stress the importance of using serial ECGs as an adjunct to patient improvement.

KEYWORDS

emergency medicine, endocrine, hyperkalemia, thyroid, weakness

1 | INTRODUCTION

Thyrotoxic periodic paralysis (PP) is a rare condition characterized by muscle weakness in the form of hyperthyroidism and hypokalemia. Thyrotoxic PP is most commonly diagnosed in Asian populations with an incidence of approximately 2%.^{1,2} Because the incidence of hypokalemic PP in the United States' non-Asian population is 0.1%, it can be easily misdiagnosed and/or overlooked.³ If left untreated, the condition can be fatal because of paralysis of respiratory muscles or development of arrhythmias caused by critical hypokalemia. Treatment for affected patients should address the underlying hyperthyroidism and correct potassium levels. Our case is unique in that it shows the use of an ECG as an adjunct in following patient electrolyte and symptomatic improvement.

2 | CASE REPORT

A 38-year-old Hispanic male with a medical history of mild persistent asthma and obesity presented to the emergency department (ED) with the chief complaint of acute bilateral upper and lower extremity paralysis. Onset was on waking up at 4:00 a.m., when he noticed was unable to lift his arms and legs. The patient also reported sharp and shooting pain originating proximally and traveling distally into the extremities. He had no respiratory or swallowing difficulties and was able to move his facial and neck muscles normally. On further questioning, the patient revealed he had been experiencing similar symptoms for 4 years. He had been seen 4 times in different EDs since the onset of his symptoms and had been given several diagnoses, including acute muscle spasm, acute stress disorder, and neuropathy. The patient reported that most

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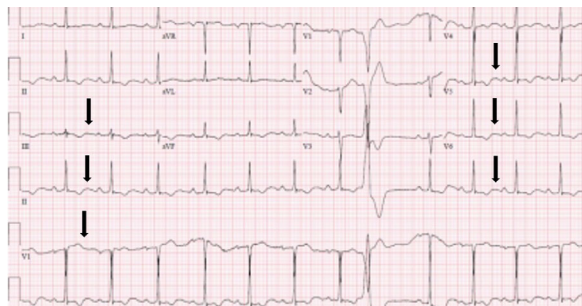


FIGURE 1 Electrocardiogram with sinus tachycardia at T-U wave fusion

of his symptoms were usually minimal or resolved before arriving at prior EDs. The patient during these visits never had his labs drawn and was frequently given NSAIDs and a muscle relaxer for symptomatic relief. The patient's significant social history included recent homelessness as well as a carbohydrate-rich diet. He had eaten an entire pizza the night before his symptom onset.

During the physical exam, the patient was sitting in a wheelchair with his hands positioned on both knees. His vitals indicated a blood pressure of 155/87 mm Hg, pulse of 120 beats/min, and respiratory rate of 18 breaths/min. The patient was afebrile, and his oxygen saturation was 98% on room air. He was mildly obese with a body mass index of 28 and was wearing mismatched shoes and tethered clothing. His skin was warm and dry, the oral mucosa was moist. No goiter or lymphadenopathy was appreciated. The patient was making direct eye contact, had symmetrical facial expressions, and was answering questions appropriately. The patient had no nystagmus on exam with 4-mm equal round and reactive pupils. A cardiac exam revealed tachycardia without obvious murmurs. Lung sounds were equal to auscultation bilaterally, without rales, rhonchi, or wheezes. There was +1 pitting edema of the lower extremities up to the mid-shin without evidence of skin discoloration or erythema. Neurologic examination yielded intact cranial nerves II–XI with no loss of sensation in bilateral upper and lower extremities. When asked to grip the examiner's fingers, the patient struggled to lift his hands to form a grasp. Grip strength was noted to be +3/5 bilaterally. Muscle strength was assessed at +1/5 in flexion and extension in bilateral shoulders, elbows, and wrists. Muscle strength of the lower extremities was assessed at +3/5 in ankle flexion and extension, +4/5 in knee flexion and extension, and +1/5 in hip flexion and extension bilaterally.

Analgesia in the form of 15 mg Ketorolac was administered intravenously, resulting in some resolution of the patient's pain. Initial laboratory tests were significant for potassium of 2.2 mEq/L (normal: 3.5–5.5 mEq/L), magnesium 1.6 mEq/L (normal: 1.3–2.1 mEq/L), TSH 0.04 IU/mL (normal: 0.34–5.6 uIU/mL), total T4 of 13 μ g/L (normal: 6–12 μ g/L), free T4 of 2.54 ng/dL (normal: 0.82–1.77 ng/dL), and total T3 of 190 ng/dl (normal: 87–178 ng/dL). Liver enzymes and complete blood count were also obtained and were within normal limits. Electrocardiogram revealed sinus tachycardia with T-U wave fusion (Figure 1).

The patient was subsequently given 40 mEq of oral potassium chloride and 40 mEq of intravenous potassium chloride, resulting in

complete resolution of his pain and paralysis. Repeat potassium was 2.9 mEq/L. An additional 40 mg of intravenous potassium chloride was given. Repeat ECG was normal sinus rhythm with resolution of TU fusion (Figure 2). The patient was diagnosed with thyrotoxic PP and was started on methimazole and propranolol. Similar to previous cases, the decision was made to trial the patient on medication management prior to discussing surgical options. He was admitted to a monitored unit and discharged 2 days later with endocrinology follow up after an uneventful hospital course.

3 | DISCUSSION

Thyrotoxic PP belongs in the family of diseases called “channelopathies” and is characterized by episodes of muscle weakness, often referred to as “attacks.” Although most cases of PP are hereditary, there is an association between hyperthyroidism and hypokalemic PP, referred to as thyrotoxic PP. Thyrotoxic PP is mostly described in Asian populations, with an incidence of ~2%.^{1,2} In the United States, the incidence of thyrotoxic PP in the non-Asian population is 0.1%.³ The differential for patient presenting with weakness and paralysis are broad. Although this case clearly demonstrated thyrotoxic PP, bilateral upper and lower extremity weakness and paralysis may include but are not limited to hypoglycemia, transverse myelitis, Guillen Bare, botulism, cerebrovascular accident, multiple sclerosis, tick paralysis, and acute intermittent porphyria.

Although the pathogenesis of thyrotoxic PP is poorly understood, it is thought to be caused by increases in sodium-potassium-adenosine triphosphate (Na/K-ATPase) pump activity within the skeletal muscles. Thyrotoxicosis leads to an increased beta-adrenergic response that increases Na/K-ATPase activity. The thyroid hormone itself also leads to increased activity of Na/K ATPase, which acts to drive potassium into cells, causing hypokalemia.⁴ A carbohydrate-rich diet can lead to insulin secretion from beta islet cells in the pancreas and cause further stimulation of the Na/K-ATPase activity. Hypokalemia is thought to hyperpolarize muscle cell membranes, leaving the muscle fibers unexcitable.

Thyrotoxic PP is most seen between the third and fifth decades of life. Patients will experience transient, recurrent episodes of flaccid paralysis. Proximal muscles are usually affected to a greater degree than distal muscles. Although rare, cases of bulbar and respiratory weakness requiring ventilatory support have been reported.⁵ Paralysis onset is usually sudden and can be preceded by stress, heavy physical exertion, or a meal rich in carbohydrates.⁴ This patient had the social stressor of recent homelessness and had consumed a carbohydrate-rich meal before the onset of his symptoms.

Hypokalemia is almost always present during paralysis. However, the degree of hypokalemia varies. One study found that patients had a mean potassium level of 2.1 mmol/L.⁶ Hyperthyroidism is a defining feature of thyrotoxic PP, and patients will have elevated serum thyroxine (T4) and decreased thyrotropin (TSH) levels. This helps to distinguish the condition from familial hypokalemic PP. Additionally, urine calcium–phosphate ratio >1.7 is a sensitive and

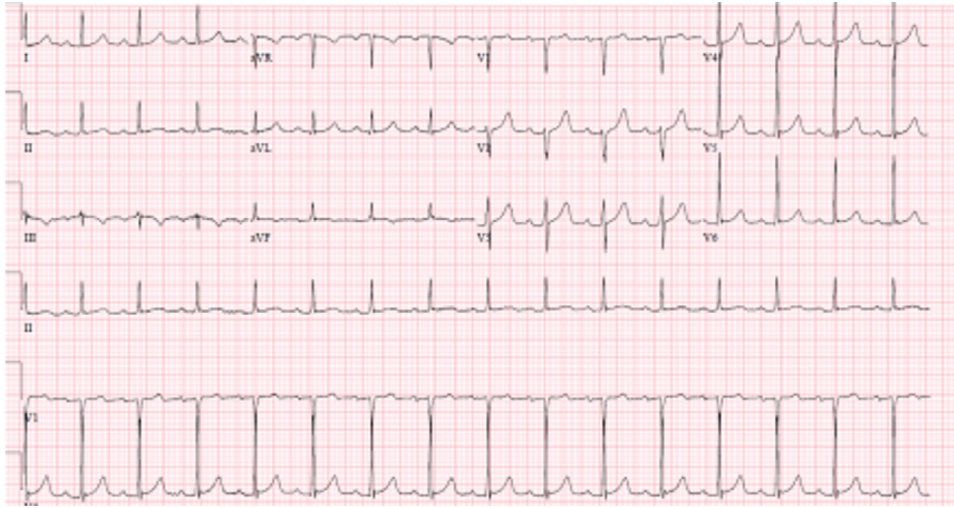


FIGURE 2 Electrocardiogram with sinus tachycardia and resolution of T-U waves

specific test to distinguish thyrotoxic PP from familial hypokalemic PP.⁷

Electrocardiogram (ECG) changes are common during attacks and reflect underlying hypokalemia. Changes include sinus tachycardia, prolonged QT, prolonged PR, widened QRS, U waves, T-U wave fusion, and ST depression. This patient's ECG exhibited both sinus tachycardia and T-U wave fusion. Similarly, hypomagnesemia can also cause similar ECG findings including prolonging the QT interval with possible U waves and T-U wave fusion. Hypocalcemia can also cause QT prolongation. Fatal arrhythmias, such as ventricular fibrillation and ventricular tachycardia, are uncommon but do occur.⁸

During acute attacks, potassium supplementation leads to improvement of weakness. One prospective study compared intravenous potassium chloride to normal saline infusion. Shorter recovery times were noted in patients who received potassium chloride.⁷ When comparing intravenous to oral supplementation, those receiving intravenous potassium chloride recovered more quickly than those who received oral supplementation. Of patients receiving more than 90 mEq of potassium chloride in a 24-hr period, 80% developed rebound hyperkalemia.⁹ This can be prevented by replacing potassium at slower rates of < 10 mEq per hour or by administering 30 mEq every 2 hr with a maximum dose of 90 mEq.¹⁰ Potassium replacement may not be enough to resolve an attack in patients with thyrotoxic PP. Magnesium is usually an adjunct to severe potassium repletion and should be considered for all patients. A non-selective beta blocker may reverse excess stimulation on the Na⁺/K⁺ ATPase and halt the excess drive of potassium into cells. 1 mg of intravenous propranolol every 10 min with a maximum dose of 3 mg has been shown to reverse weakness and hypokalemia in patients with thyrotoxic PP.¹¹

4 | CONCLUSION

The patient presented with a sudden episode of paralysis and reported a history of several similar episodes in the past, but for unknown rea-

sons was not worked up and discharged from several EDs in 3 years. After an astute physical exam and examination of the patient's history, an ECG was ordered to unearth insights on the patient's diagnosis. Lab work revealed abnormal values for potassium, TSH, T3, and T4. Coupled with the history of a carbohydrate-rich diet and a recent social stressor, the patient's history made him prone to a typical presentation of thyrotoxic PP. His ECG revealed characteristic changes along with paralysis, which were resolved with potassium administration. The patient was subsequently started on medications to restore a euthyroid state.

Thyrotoxic PP is an important diagnosis to consider in patients who experience a sudden onset of weakness or paralysis. With symptoms that may often self-resolve spontaneously, the thyrotoxic PP diagnosis can easily be missed, as demonstrated in this patient's case. Continued misdiagnoses would have put the patient at risk for a life-threatening outcome. However, after his paralysis was differentiated from other causes of weakness, the patient was able to be treated promptly with great success.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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