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Case Report

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Thyrotoxic Periodic Paralysis as an Initial Presentation of Graves' Disease Induced by Acute Alcohol Intake

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Abstract

Thyrotoxic periodic paralysis (TPP) is a rare medical emergency, that presents with acute muscle weakness or paralysis in the setting of hypokalemia and thyrotoxicosis. We highlight the case of a patient who presented with acute onset flaccid quadriparesis induced by acute alcohol intake in undiagnosed Graves' hyperthyroidism.

Keywords: Thyrotoxic Periodic Paralysis, Thyrotoxicosis, Graves' Disease, Hypokalemia, Acute Flaccid Paralysis

Background

TPP is an uncommon presentation of thyrotoxicosis. The primary defect in TPP is the increased activity of the sodium/potassium (Na/K) ATPase pump resulting in an increased shift of extracellular potassium into the intracellular space resulting in hypokalemia [1]. This causes paradoxical hyperpolarization of muscle membrane with decreased excitability of muscle fibers leading to paralysis. We discuss the pathophysiology, clinical manifestations, diagnostic evaluation, and therapy of TPP, as well as the prevention of recurrence.

Objective

To raise the awareness of Thyrotoxic Periodic Paralysis. Although rare, TPP can cause life-threatening conditions that warrant early recognition and prompt management.

Case Report

A twenty-seven-year-old man with no significant medical history presented to the ED with bilateral weakness of the upper and lower extremities. He was in his usual state of health until he woke up at 3:30 AM the night prior and found that he could not move his legs and arms. The patient reported drinking 3-4 glasses of beer before sleep. He denied pain in the extremities, bladder and bowel dysfunction, headache, abnormal body movements, sensory loss, and vision loss. A month ago, he had experienced slight leg weakness after drinking alcohol, which did not affect his ability to walk and resolved within an hour. The patient also had a history of fall due to a sudden-onset weakness after drinking alcohol two years ago, resulting in a scalp laceration requiring stitches.

On exam, his vital signs were significant for a heart rate of 115 per minute and a temperature of 99.2 F. On physical assessment, he appeared anxious but was alert and oriented with intact memory and fluent speech. Neurological examination revealed quadriparesis with 0/5 power in both upper and lower extremities except 2/5 power in finger abductors/adductors and 5/5 power in plantar flexors. Reflexes were 0/5 throughout, and the sensation was intact in all the dermatomes.

The significant initial labs (Table 1) revealed severe hypokalemia and hypophosphatemia with hyperthyroidism. The fractional excretion of potassium was 3.2, and the fractional excretion of sodium (FENA) was 0.4 percent, which was indicative of the extrarenal cause of hypokalemia. The EKG showed prolonged QT with diffuse ST depression (Fig 1). Subsequent positive serum thyroid antibodies confirmed autoimmune thyroid disease of Graves' disease.

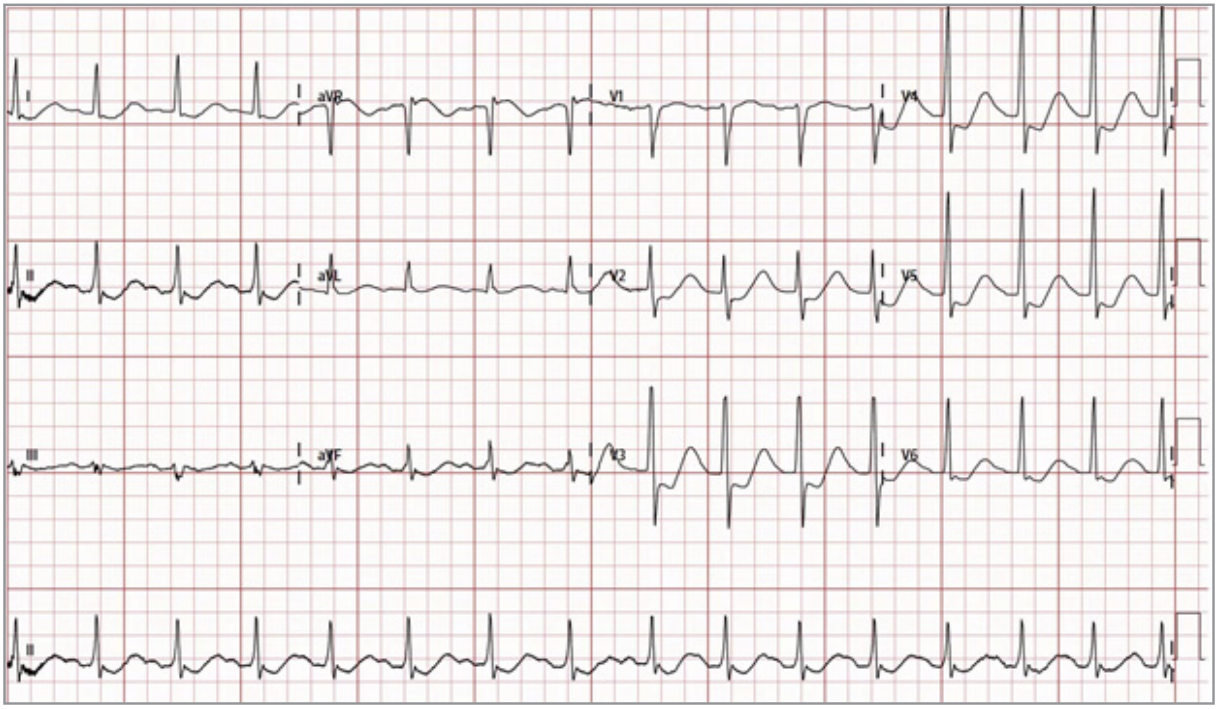

Figure 1: Initial EKG - prolonged QT with QTc of 678 milli-seconds with diffuse ST segment depression

Table 1: Initial laboratory findings

Tests (Normal ranges)	Initial results
Hb (14-18 gm/dl)	14.3 gm/dl
RBC (4.7-6.1 M/UL)	5.73 M/UL
WBC (4.8-10.8 K/UL)	8.8 K/UL
Neutrophil (1.4-6.5 K/UL)	7.8 K/UL
Platelet (150-400 K/ul)	210 K/ul
Na (135-149 mmol/L)	138 mmol/L
Potassium (3.6-51 mmol/L)	**1.4 mmol/L**
Calcium (8.2-10.1 mg/dl)	9.9 mg/dl
Chloride (93-105 mmol/l)	104 mmol/L
Bicarb (23-32 mmol/L	21 mmol/L
Glucose (59-140 mg/dl)	133 mg/dl
Creatinine (0.5-1.3 mg/dl)	0.7 mg/dl
Phosphate (2.2-5.5 mg/dl)	**1.6 mg/dl**
Magnesium (1.6-2.2 mg/dl)	1.7 mg/dl
T3 Free, serum (2.53-3.87 pg/ml)	**9.6pg/ml**
T4 Free serum (0.58-1.64 ng/dl)	**4.03ng/dl**
TSH (0.39-4.08 uIU/ml)	**<0.01 uIU/ml**
TSI (0.00-0.55 IU/L)	**3IU/L**
Thyrotropin Binding Inhibitory Immunoglobulin (0.00-1.75 IU/L)	**18.2 IU/L**
Thyroid Peroxidase Ab (<34.9 IU/ml)	**88.7 IU/ml**
Urine Creatine (Spot)	62mg/dl
Urine Potassium (Spot)	10mmol/L
Urine Sodium (Spot)	44mmol/L

The patient was admitted to the Medical ICU with the diagnosis of TPP secondary to Graves' disease and precipitated by alcohol intake. Brush and Wartofsky's score for thyroid storm was 35 (Fever 99.2 F, HR 117/min, one episode of vomiting, alcohol intake as precipitant factor).

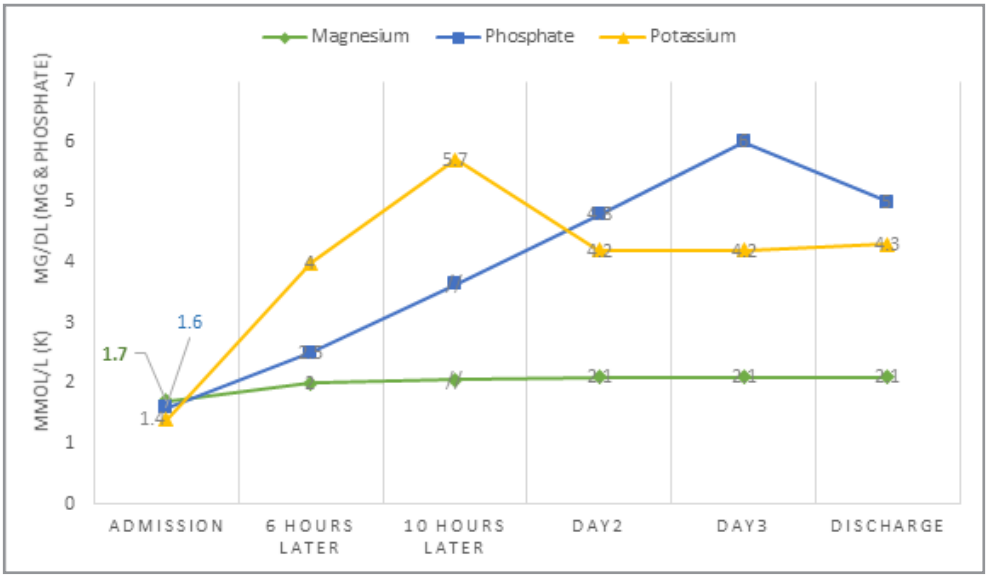

Figure 2: Trend of serum potassium, magnesium, and phosphate

The patient obtained initial 20mEq of intravenous potassium chloride and 80mEq of oral potassium chloride. He also received total of 2g magnesium sulphate for low normal serum levels. Since he had resistant hypokalemia, another 80mEq of oral potassium chloride and intravenous propranolol 1mg were given after 4 hours of initial presentation. His serum potassium improved from 1.4 to 4mmol/L and overcorrected to 5.7mmol/L. Potassium normalized to 4.1mmol/L after oral Kayexalate 30g and sodium zirconium cyclosilicate 10mg.

The long QT on the initial EKG was improved with the resolution of hypokalemia (Fig 3).

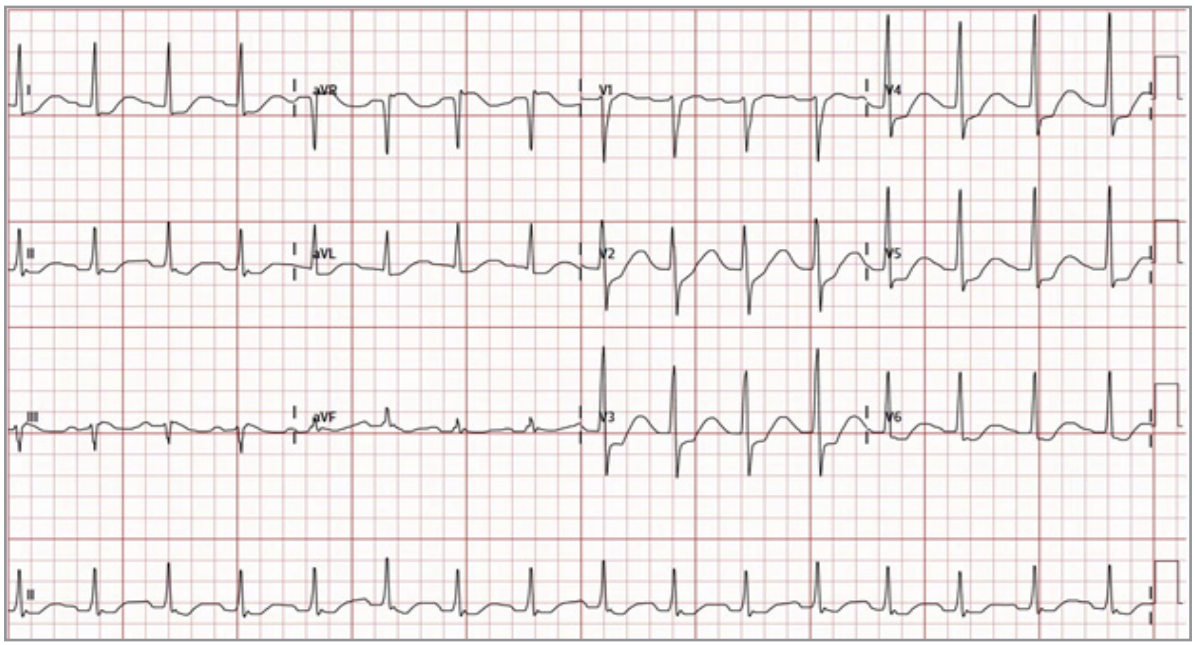

Figure 3: EKG with QTc of 543 milli-seconds (4 hours after presentation)

For impending thyroid storm, intravenous hydrocortisone 100mg every day, oral propranolol 20mg three times a day, oral methimazole 10mg twice a day, and oral potassium iodide 0.25ml every 6 hourly were given. The patient's muscle power recovered two days after admission and the patient was discharged after a few days of close hemodynamic monitoring.

The patient followed up in the endocrine clinic for two years. Propranolol was weaned off and methimazole was gradually tapered to 5mg once a week from the treatment regimen after one year of starting therapy. On this current regimen, the patient is clinically and biochemically euthyroid and had no further episodes of TPP.

Discussion

TPP is predominant in the Asian population, but it may occur in other races [1]. Although thyrotoxicosis is more common in

women with a male-to-female ratio of 1:4 to 1:10, TPP is more prevalent in males with a male-to-female ratio of 22:1 to 76:1 [2]. Due to increased immigration, TPP should also be considered a differential diagnosis for any patient presenting with weakness and hypokalemia. It can manifest in any condition that presents as thyrotoxicosis like Graves' disease, toxic adenoma, thyroiditis, toxic nodular goiter and factitious thyrotoxicosis [3, 4].

Approximately only two percent of hyperthyroid patients develop TPP which might be explained by two shared loci between Graves' disease and TPP. Increase K/Na ATP activity with defect in K+ efflux results in decreased extracellular potassium which ends up reduction in excitability of the muscle membrane culminating in acute flaccid paralysis [2, 5, 6].

Multiple risk factors like high carbohydrate meals, trauma, infections, strenuous exercise, and alcohol ingestion can trigger TPP [6]. Although electrolyte abnormalities are usually commonly seen in chronic alcoholism, an exaggerated electrolyte depletion can occur acutely with binge alcohol ingestion [7, 8]. All these risk factors increase the activity of the Na/K ATPase pump on top of the hyperthyroid state, which results in the influx of potassium inside the cell, causing hypokalemia.

After the initiation of therapy, physicians should monitor for rebound hyperkalemia. One study suggests treating patients with more than 90mEq of potassium chloride increases the risk of rebound hyperkalemia [9]. Early beta-blockage with propranolol can decrease the total potassium chloride requirement. Some literature and case reports suggest hypokalemia can be resolved even without potassium replacement [4]. However, potassium replacement should be initiated early as this can cause fatal cardiopulmonary complications. There can be concomitant hypomagnesemia and hypophosphatemia with hypokalemia, like in our patient [8, 10], and these electrolytes should also be repleted. Improvement in symptoms can be expected rapidly with the improvement of serum potassium, as hypokalemia is the primary factor for weakness. After recovery, maintaining a euthyroid state is critical so as to prevent the recurrence of TPP [4, 11].

Conclusion

TPP should always be considered in any patient who presents with a sudden onset of flaccid paralysis especially with known triggering factors. It is vital to recognize and treat early to avoid life-threatening complications such as cardiac arrhythmias and respiratory failure. Rapid improvement in symptoms can be expected with the recovery from hypokalemia. Watchful potassium administration and monitoring are required as the patients are prone to rebound hyperkalemia. Maintaining a euthyroid state helps to prevent the recurrence of TPP.

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