Subtle Signs of Thyrotoxicosis in Thyrotoxic Periodic Paralysis

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Introduction

- Thyrotoxic periodic paralysis may be difficult to diagnose as it often mistaken for more familiar causes of hypokalemia and lower extremity paralysis.

Case Description

A 26 year old Asian male with a past medical history of Graves disease presented to the hospital after he woke up with bilateral lower and upper extremity weakness that started the morning of presentation. The patient had a similar episode several years prior but the etiology of his symptoms were not elucidated. On admission, the patient was tachycardic with decreased muscle strength in the upper and lower extremities. Physical examination revealed normal thyroid size with no nodules, decreased deep tendon reflexes in the lower extremities bilaterally, and no focal neurological deficits. Laboratory studies revealed a potassium of 1.5 mEq/L, Magnesium 1.2 mg/dl, TSH of .007, and an elevated T4 and T3. EKG revealed sinus tachycardia with U waves. Thyroid ultrasound revealed a mildly enlarged thyroid with heterogenous appearance and several nodules, moderate vascularity and no microcalcifications. Thyroid uptake scan was remarkable for increased radioiodine uptake of 83% at 21 hours which was consistent with Graves Disease. The patient's presentation was presentation was consistent with thyrotoxic periodic paralysis secondary to Graves Disease. The patient was treated with propranolol 20 mg TID, methimazole 10 mg BID with aggressive electrolyte repletion. The patient returned to clinical baseline with full strength and was arranged for radioactive iodine ablation as an outpatient

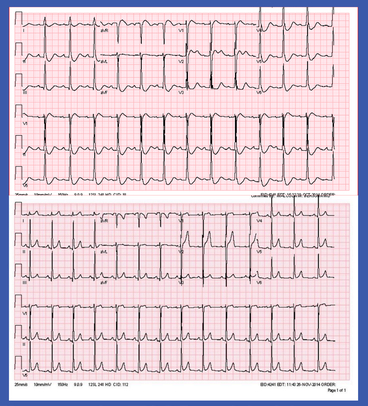

Figure 1. EKG with U waves and post electrolyte repletion

Differential Diagnosis of Acute Proximal Muscle Weakness

- Thyrotoxic periodic paralysis
- Hyperkalemic/hypokalemic periodic paralysis
- Andersen Syndrome
- Tick paralysis
- Myasthenia Gravis
- Guillain Barre
- Thyrotoxic myopathy
- Botulinum
- Spinal cord compression

Discussion

Epidemiology

- Asian males have an incidence of TPP of roughly 2% as opposed to other demographics .1-.2%
- 95% of TPP cases occur in male patients despite hyperthyroidism being more common in female patients.
- Majority of cases present between ages 20-40.

Pathophysiology

- While not fully understood, thyroid hormone increases skeletal muscle, liver and kidney sensitivity to beta adrenergic activation which increases Na/K ATPase activity; this drives K into cells
- Insulin resistance thought to play a role which drives K into cells
- Ion channel defects; Kir2.6 inward rectifying potassium channel expressed on skeletal muscle (also implicated in Graves Disease)
- Testosterone may increase Na/K ATPase activity
- K plays critical role in muscle contraction

Etiologies of TPP

- Graves Disease
- Toxic Nodular Goiter
- Iodine induced thyrotoxicosis
- Adenoma
- Thyroiditis
- Pituitary adenoma
- Amiodarone induced thyrotoxicosis

Evaluation

- Chem: Low K, Mg, Phos, nml acid base
- Elevated TSH, T4, T3
- EKG: U waves, prolonged PR,

Treatment

- Electrolyte repletion: K 30 mEq Q2H
- Propranolol +/- Methimazole
- Treat underlying cause

Implications

- TPP is a rare diagnosis and should be considered in patients that present with weakness or flaccid paralysis of the proximal. muscles
- It is important to keep TPP in the differential even if overt signs of thyrotoxicosis are not present