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Thyrotoxic Hypokalemic Periodic Paralysis

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Abstract Thyrotoxic hypokalemic periodic paralysis (THPP) is a sporadic form of hypokalemic periodic paralysis (HPP), a rare disease which is manifested as painless muscle weakness in the presence of hyperthyroidism and hypokalemia. Our patient was recently diagnosed with hyperthyroidism due to Graves' disease and was started on antithyroid therapy. He presented few months later with upper and lower extremity weakness along with finding of hypokalemia in the background of hyperthyroidism. He was given potassium replacement therapy with resolution of symptoms.

Keywords: *thyrotoxic hypokalemic periodic paralysis, hyperthyroidism, hypokalemia, potassium replacement*

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1. Introduction

Periodic paralysis (PP) is a muscle disease manifested by episodes of painless muscle weakness, usually precipitated by heavy exercise, fasting, or high-carbohydrate meals. PP is classified as hypokalemic or hyperkalemic depending on serum potassium levels. Most cases of hypokalemic periodic paralysis (HPP) are hereditary, usually with an autosomal dominant inheritance pattern. HPP is caused by mutations in the alpha subunits of either the skeletal muscle L-type calcium channel gene CACN1AS (HypoPP1) [1] or the skeletal muscle sodium channel gene SCN4A (HypoPP2) [2]. Acquired cases of HPP have been described in association with hyperthyroidism, also termed as thyrotoxic hypokalemic periodic paralysis (THPP). We present a case of hypokalemic periodic paralysis in the background of hyperthyroidism.

2. Case

Our patient is a 36 year old gentleman who presented to the emergency department with chief complaint of weakness in the upper and lower extremities that began while he was at rest. Patient stated that he was unable to get out of the chair or get in and out of the car without assistance. He denied any recent illness, blurry vision or headache. His past medical history is significant for neurofibromatosis, restless leg syndrome, hyperlipidemia, irritable bowel syndrome and hyperthyroidism secondary to Graves' disease. Our patient was diagnosed with Graves' disease 5 months prior when the patient presented with diffuse toxic goiter with thyrotoxicosis and was found to have a TSH of <0.01, free T4 of 2.48, thyroid stimulating immunoglobulins (TSI) elevated at 323 and iodine uptake scan test positive for 70% uptake. The patient was started on propranolol and methimazole.

Physical exam revealed a temperature of 35.8 Celsius, a heart rate of 80 beats per minute, blood pressure of 141/92 mm Hg, respiratory rate of 16 per minute with an oxygen saturation of 99% on room air. He was found to have 4/5 muscle strength in bilateral upper and lower extremities with multiple nodules seen throughout the body consistent with his neurofibromatosis. The remainder of the physical exam was normal.

Laboratory data, included a complete blood count that was within normal limits. A basic metabolic profile that revealed a sodium of 138 mEq/L (136-145), potassium 2.4 mEq/L (3.5-5.1), chloride 110 mEq/L (98-107), CO2 23 mEq/L (22-29). Other tests including BUN, creatinine, GFR, glucose, CK total, lactic acid, magnesium were normal. TSH was <0.01 mIU/L (0.35-4.94) and free was T4 0.82 ng/dL (0.7-1.48). Urine electrolytes in random urine sample included chloride 149 mEq/L (110-250), sodium 122 mEq/L (40-220), potassium 15.1 mEq/L (25-125). CT head was negative for acute intracranial abnormalities. EKG was consistent with sinus rhythm, mild intra ventricular conduction delay, nonspecific T-wave flattening in the septal and limb leads.

The patient was admitted for further management. Patient was treated with potassium during hospital stay and his weakness fully resolved. He was evaluated by endocrinologist who ruled out other causes of hypokalemia such as hyperaldosteronism. Because of history of neurofibromatosis, renin-aldosterone levels were checked to rule out adrenal tumor. Both renin and aldosterone levels were within normal limits. An increase in methimazole dose was recommended along with definitive therapy for I-31 ablation. He remained stable during his hospital stay.

Table 1. Potassium levels of the patient on presentation and after replacement

Component *Latest Ref Rng*	Day 1, on presentation	Day 1, after K replacement	Day 2
BUN *(9-21) MG/DL*	19	16	10
Calcium *(8.5-10.3) MG/DL*	9.3	9.0 (L)	9.4
Chloride *(98-107) MMOL/L*	110 (H)	111 (H)	107
CO2 *(22-29) MEQ/L*	23	26	23
Creatinine, Ser *(0.7-1.3) MG/DL*	0.7	0.7	0.6 (L)
EXT GFR *mL/min*	128	128	152
Glucose *(70-105) MG/DL*	107 (H)	122 (H)	96
Potassium *(3.5-5.1) MMOL/L*	2.4 (L)	4.1	4.3
Sodium *(136-145) MMOL/L*	138	138	136

Table 2. TFTs of the patient on presentation

Component *Latest Ref Rng*	Day 1
TSH, POC *(0.35-4.94) uIU/ML*	<0.01 (L)
T4 (Thyroxine), Free, Ser (0.7-1.48) NG/DL	0.82

3. Discussion

THPP is an endocrine disorder presenting with proximal motor weakness, typically greatest in the lower extremities, hypokalemia, and signs or laboratory findings consistent with hyperthyroidism [3]. THPP is a rare manifestation of thyrotoxicosis, characterized by hypokalemia, recurrent episodes of muscular weakness and complete inability to walk, which regress with correction of hypokalemia [4]. THPP is most widely reported and studied in Asian populations, in whom there is a 10-fold higher incidence of this complication than in Caucasian populations [4]. Because of its rare prevalence especially in the western population, THPP could be easily overlooked [5]. Most cases of THPP occur in men despite the higher incidence of hyperthyroidism in women [5]. The age of onset of symptoms is between 20 and 39 years in approximately 80% of patients [5].

Our patient is a young Caucasian male, who initially presented with signs and symptoms of thyrotoxicosis and was diagnosed with hyperthyroidism secondary to Graves' disease. He was started on methimazole and propranolol. He presented few months later with bilateral upper and lower extremity weakness with his laboratory data significant for hyperthyroidism and hypokalemia and was suspected for hypokalemic periodic paralysis secondary to hyperthyroidism. The etiology of THPP is not completely clear [5]. It has been suggested that it is caused by an increase in intracellular shift of potassium due to hyperactivity of Na/K ATPase pump [5]. Na/K ATPase, in fact, is activated by insulin and β 2 adrenergic receptor stimulation [5]. Thyroid hormones seem to increase the number of β 2 adrenergic receptors in the skeletal muscles and, consequently, the Na/K ATPase activity [6]. Hyperthyroidism, moreover, increases not only Na/K ATPase activity, but also the passive transmembrane flux of these ions [6]. Before making a diagnosis of THPP, it is necessary to exclude other causes of paralysis and other and more common causes of hypokalemia [5]. We ruled out other common causes of hypokalemia such as eating disorders, GI losses, hyperaldosteronism, hyperreninism, use of diuretics or steroids and subsequently diagnosis of THPP was established.

Therapy of THPP is based on both correcting thyroid hyperactivity and providing oral or intravenous supplements of potassium until complete recovery of the symptoms and improvement of clinical status [5]. As our patient was already on antithyroid therapy, he was treated with potassium with resolution of his symptoms.

4. Conclusion

Thyrotoxic hypokalemic periodic paralysis is a rare manifestation of hyperthyroidism, especially in Caucasian males, which can lead to significant morbidity and can be life threatening if it remains undiagnosed and untreated. Patients with hyperthyroidism presenting with periodic paresis or paralysis should be screened for THPP and if diagnosed should be promptly treated with potassium therapy for rapid resolution of symptoms and prevent life threatening events.

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