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Case Report

Thyrotoxic Hypokalemic Periodic Paralysis: A Case Report

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ABSTRACT

Thyrotoxic hypokalemic periodic paralysis is a rare and fatal complication of hyperthyroidism and is associated with low serum potassium levels and muscle weakness. Herein, we presented a young male patient who did not use the antithyroid drugs given for Graves' disease and was admitted with the complaint of weakness in the extremities and diagnosed with thyrotoxic hypokalemic periodic paralysis.

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Introduction

Thyrotoxic hypokalemic periodic paralysis (THPP) is a rare complication of hyperthyroidism, characterized by low serum potassium levels and acute muscle weakness without any sensory deficit or confusion.[1] We presented a Graves' disease patient who developed weakness in the extremities after stopping the antithyroid medication and was diagnosed as thyrotoxic hypokalemic periodic paralysis.

Case Report

A 22-year-old male patient diagnosed with Graves' disease three years ago was admitted to the emergency department with complaints of weakness in his upper and lower limbs. At the time of diagnosis, his symptoms were hand tremors and irritability. In his thyroid function tests, thyroid-stimulating hormone (TSH) was <0.0025 mU/L (normal range [NR]: 0.35-4.94), free thyroxine (fT4) was 1.43 ng/dL (NR: 0.7-1.47), free tri-iodothyronine (fT3) was 4.96 ng/L (NR: 1.71-

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3.71), TSH-receptor antibody was 9.25 IU/L, which was highly positive (NR <1.5 IU/L). His thyroid scintigraphy showed diffuse enlargement of both thyroid lobes with uniform isotope uptake. After diagnosing Graves' disease, methimazole 10 mg twice a day, propranolol 40 mg daily, and potassium citrate tablets once daily was started. However, the patient stated that he did not use his medications for the last eight months.

On physical examination, the patient was conscious, oriented, afebrile. His pulse rate was 110/min regular, and blood pressure was 100/80 mmHg. His electrocardiogram (ECG) showed sinus tachycardia. Neurologic examination revealed his upper and lower limb power as grade 1/5 with intact sensation. Higher mental function, including speech, cranial nerves and autonomic nervous system examination revealed no abnormality. Complete blood count showed total leukocyte count 13,110 K/mcl, neutrophils 10,960 K/mcL, hemoglobin 15.7 g/dL, platelet 269,000 K/mcL and venous blood gas analysis showed pH of 7.34, pCO_2 46 mmHg, HCO□ 21 mmol/L. Random blood sugar was 112 mg/dL, blood urea 34 mg/dL, serum creatinine 0.67 mg/dL, sodium (Na) 143 mmol/L, potassium (K) 2.2 mmol/L, total calcium 9.8 mg/dL and all liver enzymes were within normal limits (Table 1).

Based on the neurological examinations and the biochemical results, THPP was diagnosed. 30 mEq intravenous potassium chloride in %0.9 sodium chloride solution was started immediately, and the patient was treated with 40 mg oral propranolol per six hours and 30 mg methimazole daily. Serum potassium levels increased to 4.2 mmol/L after five hours of treatment, and neurologic examination revealed upper and lower limbs power was grade 5/5. The patient was examined in the endocrinology clinic the day after. Complete clinical recovery was seen. Thyroid function tests revealed TSH <0.01 mU/L, fT4 2.92 ng/dL, fT3 14.31 ng/dL, potassium was 4.91 mmol/L and the other parameters were within normal limits. The patient was educated to take his medications regularly, avoid strenuous exercise, alcohol use and a high-carbohydrate diet.

Discussion

The incidence of THPP, which is commonly identified in East Asian men, is also increasing in other parts of the world. In THPP, □ncreased beta-adrenergic stimulation and overactive Na-K-ATPase channels cause potassium leak into muscle cells, and muscle cells can not be stimulated due to hyperpolarization. Factors such as a high

Table 1. Laboratory findings at diagnosis, at admission to emergency department and at discharge.

Parameters	At the time of diagnosis of Graves	At the admission to emergency department	At discharges
Leukocyte count (K/mcL)	8,730	13,110	6,730
Hemoglobin (g/dL)	15.5	15.7	14.8
Urea (mg/dL)	25	34	21
Creatinine (mg/dL)	0.82	0.68	10
Sodium (mmol/L)	137	143	139
Potassium (mmol/L)	4.52	2.2	4.4
Thyroid-stimulating hormone (mU/L)	<0.0025	<0.01	<0.01
Thyroxine (ng/dL)	1.43	2.92	0.61
Free triiodothyronine (ng/L)	4.96	14.31	2.49
TSH-receptor antibody (IU/L)	9.25	14.1	

carbohydrate diet, strenuous exercise, emotional stress can precipitate an attack of THPP.[2] The ion channel defect in familial periodic paralysis is not found in the thyrotoxic form. Some genetic mutations on proteins called "kir 2.6", regulated by thyroid hormones and responsible for membrane potassium transitions, can cause THPP.[3]

Thyrotoxicosis is the most common cause of acquired periodic paralysis.[4] In the absence of a family history of paralysis, renal tubular acidosis should also be considered.[5] Symptoms of hyperthyroidism may not always be evident in patients, or muscle paralysis could be the first manifestation of thyrotoxicosis. THPP can be complicated with ventricular fibrillation or hypercapnic respiratory failure if not diagnosed and treated promptly.[6]

In summary, THPP is an infrequent complication of hyperthyroidism that can be mortal. The most critical step in preventing THPP is to achieve euthyroidism.

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Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contribution

Study Conception: SE, EA; Study Design: SE; Supervision: SE, EA, CE; Materials: SE, EA, CE; Data Collection and/or Processing: SE, EA, CE; Statistical Analysis and/or Data Interpretation: SE, CE; Manuscript Preparation: SE; Critical Review: SE, EA, CE.

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