

THYROTOXIC HYPOKALEMIC PERIODIC PARALYSIS PRESENTING WITH PARAPARESIS

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ABSTRACT

Thyrotoxic hypokalemic periodic paralysis (THPP) is a rare hyperthyroidism-related endocrine disorder seen predominantly in men of Asian origin. The main characteristics of the disease are hyperthyroidism, hypokalemia, muscle weakness and acute paralysis. We describe a 24-year old male patient with thyrotoxic hypokalemic periodic paralysis presented with symmetrical motor weakness in lower extremities. When he woke up in the morning, he noticed the weakness in both arms and

legs, also he recognized that he can not walk. According to his history, similar symptoms occurred and resolved a few months ago. Although hypokalemic periodic paralysis is a rare disorder, clinicians should have a high index of suspicion for patients presenting with walking abnormalities, flaccid paralysis and hypokalemia. Early recognition of thyrotoxicosis and treatment by the emergency physician is necessary to prevent THPP related complications.

Key Words: Hypokalemic periodic paralysis, thyrotoxicosis, paraparesis *Nobel Med 2013; 9(1): 118-120*

PARAPAREZİ İLE BAŞVURAN TİROTOKSİK HİPOKALEMİK PERİYODİK PARALİZİ

ÖZET

Tirotoksik hipokalemik periyodik paralizi (THPP) nadir görülen ve sıklıkla Asya kökenli erkeklerde rastlanan, hipertiroidi ile ilişkili endokrinolojik bir bozukluktur. Hastalığın temel özellikleri hipertiroidi, hipokalemi, kas güçsüzlüğü ve akut paralizidir. Olgumuz, alt ekstremitelerde simetrik motor kayıp ile başvuran ve tirotoksik hipokalemik periyodik paralizi gelişmiş olan 24 yaşında bir erkek hastadır. Hasta sabah uyandığın-

da kollarında ve bacaklarında güçsüzlük hissetmiş ve yürüyemediğini farketmiştir. Hastanın özgeçmişinden birkaç ay öncesinde benzer semptomların geliştiği ve düzeldiği öğrenilmiştir. Hipokalemik periyodik paralizi nadir görülen bir bozukluk olmakla beraber; yürüme anormallikleri, flask paralizi ve hipokalemiyle başvuran hastalarda klinisyenlerin yüksek oranda şüphe duymaları gerekir. THPP ilişkili komplikasyonların önlenmesi için, acil hekimleri tarafından tirotoksikozun erken tanınması ve tedavisi gereklidir.

Anahtar Kelimeler: Hipokalemik periyodik paralizi, tirotoksikoz, paraparezi *Nobel Med 2013; 9(1): 118-120*

INTRODUCTION

Hypokalemic periodic paralysis is a rare but potentially fatal disorder that may accompany thyrotoxicosis.¹ Fatal episodes of muscle weakness occur through the involvement of the respiratory muscles, and life-threatening cardiac arrhythmias can be seen.² The main causes of the hypokalemic periodic paralysis include familial hypokalemic periodic paralysis, barium intoxication, and thyrotoxicosis.¹ In case of thyrotoxic hypokalemic periodic paralysis (THPP), thyroid hyperfunction alters glucose metabolism, insulin secretion and the Na⁺/K⁺ adenosine triphosphatase pump, which facilitates K⁺ influx into the cells resulting in relative hypokalemia.^{1,2}

Hypokalemic periodic paralyzes are characterized by recurrent episodes of muscle weakness occurring at irregular intervals, typically with complete recovery between attacks. Acute attacks are often precipitated by exercise, heavy physical activity, stress, cold, heat, vomiting or a high carbohydrate meal.²⁻⁴

We present a case of THPP, who was suddenly unable to walk owing to hypokalemia.

CASE REPORT

A 24-year-old male patient presented with proximal and distal muscle weakness in lower extremities since he woke up (for one hour). He suffered palpitation and sweating for the last week and marked weakness in his both thighs and also in both arms for two days. The patient was taking no medications, herbal products or illicit drugs. The patient reported having a similar episode a few months ago, however he did not seek any medical advice. He denied any family history of paralysis or any thyroid disorder.

In the emergency department, the patient was fully conscious, oriented and anxious. Vital signs were; blood pressure of 130/60 mm of Hg, temperature of 36.7°C, pulse of 120 beats/minute, respiratory rate of 22 breaths/minute and oxygen saturation of 97% in room air. His physical examination revealed tachycardia, marked sweating, reduced deep tendon reflexes and motor weakness in both lower extremities (4/5 strength). He also stated having weakness in his both arms, however there was no weakness on neurological examination (5/5 strength). Cranial nerves, sensory examination, and other examinations including cardiac, respiratory and skin were normal throughout.

Initial laboratory tests showed a significant hypokalemia (1.8 mEq/L; normal: 3.5-5.5 mEq/L). Otherwise, the complete blood count, electrolytes

including sodium and calcium, liver function tests, coagulation parameters were in normal ranges. His creatine kinase level was 35 IU/L (normal:38-174 IU/L). Thyroid-stimulating hormone was low (<0.01 mIU/L; normal:0.35-5 mIU/L) with high free T₃ (12.00 pg/mL; normal:2.3-4.2 pg/mL) and free T₄ (39.70 ng/mL; normal:0.9-1.8 ng/mL). A 12-lead electrocardiogram showed sinus tachycardia and unspecific ST-T changes.

The diagnosis of THPP was made based on physical examination findings and laboratory results. In the emergency department, the patient was treated with oral and intravenous potassium (30 mEq/L per hour, for three hours), also oral propylthiouracil 200 mg and oral propranolol was initiated. The patient was admitted to hospital ward. After 24 hours, his hypokalemia and thyroid function tests were corrected and the patient's strength returned to normal in lower extremities.

DISCUSSION

Thyrotoxic hypokalemic periodic paralysis occurs in approximately 2% of all thyrotoxic patients of Asian descent, with a male to female ratio of 20:1.⁴ Like the other studies, Manoukian et al. demonstrated in their American population based study that, THPP is most commonly seen in males with Asian heritage. Attacks of paralysis tend to occur during the night hours and proximal muscles of the lower extremities are more affected than other muscle groups.⁵ Therefore, historically the condition has been referred to in the literature as "nocturnal paralysis".⁶ Our patient was also a young Asian male, who recognized his weakness in morning hours.

Maurya et al. found that, primary (idiopathic) hypokalemic periodic paralysis occurred in 56.7% of their study population. Also, 43.3% of patients had a secondary cause for their condition, which included renal tubular acidosis, Gitelman syndrome, and thyrotoxicosis.⁷ Despite this finding, the literature about hypokalemic paralysis reveals conflicting results. Another study argue that, secondary causes for hypokalemic periodic paralysis are more common (68%) than the hereditary causes.⁸

The leading secondary causes include thyrotoxicosis, hyperaldosteronism, nephrotic syndrome, acute tubular necrosis, diabetic ketoacidosis, barium intoxication, diuretics, laxative or thyroid hormone abuse, diarrhea, vomiting and drugs (tocolytics, theophylline, amphotericin B, corticosteroids). Late onset hypokalemic periodic paralysis raises strong suspicion of a secondary rather than primary →

periodic paralysis.⁹ Herbal medicines and thyroxine-containing dietary supplements for weight loss, which are growing in popularity, should be also considered in hypokalemic paralysis.¹⁰ The trigger and important role of thyrotoxicosis was widely accepted. Our patient did not have any electrolyte imbalance except his hypokalemia and he denied taking any drugs including herbal remedies.

The clinical findings of thyrotoxicosis may be subtle in many THPP cases, therefore thyroid function tests should be performed in all patients with hypokalemic paralysis.¹¹ Thyrotoxicosis causes an increase in β -adrenergic hypersensitivity and increase in potassium influx into the cells via the sodium/potassium ATPase pump. Oral or intravenous propranolol and propylthiouracil can be initiated early to stop the cellular uptake of potassium. To prevent recurrences during the remainder of the thyrotoxic period, nonselective beta-blockers are considered first-line therapy.^{5,6} Potassium replacement was questioned in the literature. Because the total body potassium stores are normal in patients with THPP, fast and excessive repletion of potassium alone is controversial and rebound hyperkalemia is a potential hazard of potassium administration.³ There are no randomized controlled trials addressing

the value of potassium supplementation and one author suggests that, vigorous potassium replacement should be reserved in patients with life-threatening arrhythmias due to hypokalemia.¹² In our patient, we treated thyrotoxicosis and potassium “deficiency” simultaneously, no dysrhythmias or any other complications were observed.

Emergency physicians should be aware of the hypokalemic periodic paralysis associated with thyrotoxicosis and consider monitoring electrolytes in patients presenting with acute weakness. THPP is a rare complication of hyperthyroidism, but thyroid function tests should be carried out routinely in hypokalemic paralysis.¹ Although potassium supplements help the recovery of the patients, the definitive treatment for THPP is control of the hyperthyroid state.

CONCLUSION

Weakness is one of the common complaints in emergency departments. It is important that the emergency physician recognize this uncommon syndrome early and initiate the appropriate management for restoration of serum volume potassium and to treat the underlying thyrotoxicosis.

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