



Thyrotoxic Hypokalemic Periodic Paralysis: A Case Report

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Abstract

Hypokalemic periodic paralysis is a rare disorder characterized by reversible attacks of muscle weakness accompanied by episodic hypokalemia. The most common causes of hypokalemic periodic paralysis (HPP) are familial periodic paralysis, thyrotoxic periodic paralysis (TPP) and sporadic periodic paralysis, respectively. There are generally some precipitating factors such as stress, vigorous exercise and high carbohydrate food consumption which all ease the occurrence of attacks. The duration of attacks range from 2-36 hours and can be shortened by K⁺ supplementation in appropriate situations. 28 years old male, admitted to our clinic with severe weakness at his legs and arms, on laboratory examination severe hypocalcemia due to overt thyrotoxicosis detected. After antithyroid drug therapy his symptoms and hypocalcemia resolved. After three months of therapy he underwent total thyroidectomy because of incomppliance to medical therapy. Rapid recognition and management of the disorder were the key factors to avoid fatal complications.

Key Words: Hypokalemic periodic paralysis, thyrotoxicosis, graves disease

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Introduction

Thyrotoxic hypokalemic periodic paralysis (THPP) is characterized by thyrotoxicosis, hypokalemia and acute proximal muscular weakness. The incidence of TPP, which is a rare complication of hyperthyroidism, is 0.1-0.2% [1]. In many cases, the paraplegic presentation of the illness and clinical unfamiliarity with thyrotoxicosis can delay diagnosis and treatment. We report a case of TPP due to Graves disease in a Turkish male, who presented with periodic paralytic episodes for a long time, before the diagnosis was made.

Case report

A male patient aged 28 was admitted to the hospital, complaining of extreme fatigue and muscle weakness in his arms and legs. Medical history revealed prior use of antithyroid drugs for approximately one month, two years ago. The patient complaint of fatigue and muscle weakness in his legs once in every 2-3 months for the last 2 years, the attacks resolved spontaneously after 4-6 hours. It was revealed that the patient had come to the emergency room with this complaint more than once, upon which he had been diagnosed with hypopotassemia, received potassium (K⁺) replacement and had been discharged. Physical examination revealed a slightly enlarged thyroid gland without nodules, and mild exophthalmus. Neurological examination revealed bilateral muscle strength of 1/5 for the lower extremities and 2/5 for the upper extremities. Other system examination results were normal. Medical tests revealed K⁺ 2.2 mmol/l (3.5-5.1), free T3 19.7 pg/mL (2.0-4.4), free T4 4.57 ng/dL (0.93-1.7), TSH <0.005 μ IU/mL (0.27-4.2), and thyroid receptor antibody 20 IU/L (0-9). Liver and kidney functions and complete blood count parameters were normal. Ultrasonography revealed that thyroid parenchyma was enlarged and extremely heterogenic, with an increased blood flow in the doppler USG. Treatment with propranolol 120 mg/day and methimazole 40 mg/day was initiated. Additional K⁺ replacement was not deemed necessary since K⁺ serum levels following the 3 vials of KCL replacement were normal. No similar case of paralysis was present in the family history of the patient. The patient showed low compliance to drugs, therefore a total thyroidectomy was performed after the 3rd month of the antithyroid treatment. Pathology revealed adenomatous goiter. After operation, no periodic paralysis attacks was noted and laboratory evaluation revealed normal potassium level during the three years of the follow-up period.

Discussion

Periodic paralysis is a rare disorder that is characterized by recurring muscular weakness or episodes of paralysis. The severity of the attacks can vary, from muscle stiffness to extensive paralysis. Respiratory system muscle stiffness is rare but can be fatal [2]. The disorder is divided into three forms: Hypokalemia, hyperkalemia, and normokalemia. Hypokalemia can be inherited through autosomal dominant transmission and can also develop secondary to thyrotoxicosis [3]. Thyrotoxic hypokalemia periodic paralysis is more prevalent in Asian males but continues to increase in western societies [4]. TPP is characterized by painless paralysis following sudden, short, repetitive, strenuous bouts of exercise or extreme carbohydrate diets or long bouts of rests [5]. Respiratory muscles are usually protected [6]. Thyroid hormones increase Na-K-ATPase activity allowing K⁺ to enter the cells [7]. Therefore, hypokalemia can be cured by antithyroid or beta blocker treatment without K⁺ replacement. However, K⁺ replacement should not be avoided in case of life-threatening severe hypokalemia [8]. Proper treatment of thyrotoxicosis should be continued to prevent attacks after successful initial treatment of hypokalemic periodic paralysis. On the other hand, the patient should be advised to abstain from attack-inducing activities such as fasting, exercise, extremely high carbohydrate diets and alcohol intake. In conclusion, thyrotoxic hypokalemic periodic paralysis is a rare disorder, especially among Turkish population, and it should always be considered in patients with acute paralysis and hypokalemia.

No written consent was obtained from the patient since no visual content or ID information that could reveal the identity or violate the privacy of the patient was used in the article.

Conflict of Interest

No conflict of interest was declared by the authors.

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