

Thyrotoxic Hypokalemic Periodic Paralysis: A Case Report

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Abstract

Periodic Thyrotoxic Hypokalemic Paralysis (PTHP) is a rare endocrinological complication characterized by hypokalemia, episodes of muscle weakness and thyrotoxic symptoms. The following is a case report of PTHP in a black patient diagnosed with hyperthyroidism during an episode of sudden and progressive weakness associated with hypokalemia. PTHP is often underdiagnosed and should be remembered as a diagnosis of acute muscle weakness in young patients, especially males, regardless of ethnicity, although it is frequent in Asians.

Keywords: Periodic thyrotoxic hypokalemic paralysis • Hyperthyroidism • Neurological disorders

Introduction

Periodic Thyrotoxic Hypokalemic Paralysis (PTHP) is a rare potentially lethal endocrinological disorder characterized by hypokalemia, episodes of muscle weakness, and thyrotoxic symptoms. It mainly affects men, between the third and fourth decades of life, of Asian origin, hardly reported in an Africa descent population [1,2]. The pathophysiology of PTHP remains unclear, but it is known to occur secondary to rapid intracellular influx of potassium in the skeletal muscles during thyrotoxic crises [3]. Although it can be found in any cause of thyrotoxicosis, it is more commonly identified in patients with Graves' disease [4]. Due to being a rare condition and with manifestations often found in neurological disorders the aim of this study is to demonstrate the clinical manifestations, complications and therapeutic management of a 32-year-old African American male in the emergency department, with tetraplegia, acute respiratory failure and electrocardiographic changes related to hyperthyroidism, and to warn about the importance of considering PTHP as a diagnostic hypothesis in cases of paralysis associated with hypokalemia.

Case Report

A 32-year-old African American male, previously healthy, with no family history of similar clinical features, was admitted to the emergency medical service on his town because of the sudden symmetrical reduction of motor strength of the legs (MRC 3/5), afebrile, without signs of meningeal irritation, 15 points on the Glasgow coma scale, and goiter on examination and palpation of the thyroid (Figure 1). He reported drinking a few hours after the onset of symptoms, in addition to frequent diarrheal episodes that began two months before hospital admission. After 6 hours of admission to the emergency room,

he had upper limb involvement and worsening of limb strength 2/5 on the MRC scale. He presented tetra paresis in addition to reduction of thermal sensitivity, as well as pain and tactile sensitivity in all limbs. Also, loss of tendon reflexes in lower limbs.

The patient was referred to specialized clinical evaluation, manifested tachycardia, oxygen desaturation (70%), associated with significant respiratory effort. orotracheal intubation was performed, and mechanical ventilation was maintained. and then evolved with ventricular tachycardia and hemodynamic instability that was reversed after electrical cardioversion.

Laboratory tests revealed hyperthyroidism due to Graves' disease with TSH values: 0.01 IU/L (NR 0.38-5.33 mIU/L); Free T4: 4.8 ng/dL (NR 0.54-1.24 ng/dL); ANTI-TRAB: 40 U/ml (NR: <0.55); besides hypokalemia of 2.6 mEq/L



Figure 1. Thyrotoxic goiter.

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(NR: 3.5-5.1 mEq/L); and Anti-Nuclear Factor (ANF): non-reactive. Thyroid ultrasonography evidenced an increase in total volume, without nodules. Electroneuromyography showed signs of axonal demyelinating neuropathy in recovery phase. Computed Tomography and magnetic resonance imaging of the skull, thoracic and lumbar spine showed no apparent lesions justifying the clinical presentation. After potassium replacement, the patient presented significant clinical improvement and was withdrawn from mechanical ventilation. He remained with only a predominant deficit of quadriceps strength and reduced patellar reflex, both bilaterally. Neurological symptoms only achieved complete remission after antithyroid administration (methimazole) and the use of a non-selective beta-blocker (propranolol). Currently, the patient is undergoing ambulatory follow-up and maintains pharmacological treatment, with complete recovery of muscle strength 5/5 on the MRC scale and normalization of tendon reflexes.

Discussion

Characterized by the acute onset of severe hypokalemia and significant muscle weakness, PTHP is a rare complication found in thyrotoxicosis. It is known that the thyrotoxic crisis is more incident in women; however, when PTHP is treated, there is a significant inversion in this proportion, being about 17 to 76 times more common in men than in women [5]. Most of the PTHP reports have been described in the Asian population. Considering epidemiological data, it was found that the incidence in this population is 10 times higher when compared to white individuals, being sporadically observed in non-Asian, As white, black and Hispanic populations [6]. In acute cases of ascending muscle weakness, PPHT should always be suspected due to the lethality of the condition [7]. Often, the fall in serum potassium level during a crisis is not significant, remaining within normal parameters. In addition, the symptoms of thyrotoxicosis can be nonspecific and very subtle, such as diarrhea and vomiting, and go unnoticed during the initial condition [8]. These factors make the diagnosis difficult and can be confused with its two main differential diagnoses: familial hypokalemic periodic paralysis (similar clinical and laboratory presentation) and Guillain-Barré syndrome [9]. Therefore, the investigation of thyroid function is essential for this differentiation, as well as the presence of antibodies to Graves' disease, as was done in the case described. PPHT is associated with several triggering factors, such as a meal rich in carbohydrates, stress, trauma, infections, medications, rest after exercise and, as in the case reported, acute alcohol intake [10]. Despite having an indefinite pathophysiology, the relationship between the occurrence of episodes of paralysis and hypokalemia is clear, resulting from the high and rapid cellular influx of potassium, with serum values generally below 3.0 mmol/L being found. The mechanism through which hypokalemia leads to muscle weakness results from increased thyroid hormones, beta-2 adrenergic stimulation and hyperinsulinemia, which generate increased Na/K-ATPase activity in the skeletal muscle cell membranes resulting in greater potassium displacement to the intracellular medium. Such alteration is seen in those patients with genetic predisposition (up to 33% of cases), thyrotoxicosis, being the most common cause in Graves' disease, and in those exposed to precipitating factors [11].

The clinical presentation of PPHT is characterized by sudden, transient and recurrent episodes of flaccid paralysis in lower limbs and decreased tendon reflexes, with severity ranging from slight reduction in muscle strength to quadriplegia. It also has a generally ascending and asymmetric progression, affecting mainly the proximal muscles. In addition, hypokalemia may present subtle or even relevant thyrotoxic signs and symptoms, such as diarrhea, weight loss and tachycardia. This paralysis rarely involves the ventilatory and facial muscles, so the manifestation of acute respiratory failure is poorly described. In this case, the evolution of the condition with ventilatory disorder in the reported patient was observed, which demonstrates the severity and

relevance of the case. In addition, other potentially lethal complications were observed among the possible and frequent electrocardiographic changes in this condition, which are characterized by sinus tachycardia, prominent U wave, prolonged PR interval, increased amplitude of the P wave, enlarged QRS complexes, first degree of blockage atrioventricular and ventricular arrhythmias. The immediate treatment should be the correction of hypokalemia in order to avoid serious complications, such as cardiorespiratory changes. Potassium replacement should preferably be performed orally, since, as hypokalemia is caused by intracellular influx of potassium and not by loss, rebound hyperkalemia may occur. In addition, by blocking the sodium/potassium ATPase pump, non-selective beta-blockers are indicated to accelerate recovery and prevent new attacks of paralysis, giving preference to propranolol. For the definitive treatment, it is necessary to use of antithyroid medications, such as methimazole or propylthiouracil, radioactive iodine and even thyroidectomy, in order to establish control of hyperthyroidism and prevent recurrences.

Conclusion

From the report, it is concluded that PPHT is a rare condition with potential lethality, which is underdiagnosed several times. This disease should be remembered as a diagnosis of acute muscle weakness in young patients, especially males, regardless of ethnic origin, although it is more prevalent in patients of Asian origin. PPHT, when correctly diagnosed and treated, shows a good prognosis if corrections of hypokalemia and hyperthyroidism are performed, with consequent neuromuscular and cardiac improvement; in addition to helping, allowing the reduction of new crises.

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