

Case Report

A Case of Thyrotoxic Periodic Paralysis

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Abstract

Hypokalemic periodic paralysis is an uncommon manifestation of thyrotoxicosis and the condition is most commonly reported in young Asian men. Patient often presents with acute onset symmetrical flaccid muscle paralysis and proximal limb muscles may be more severely involved compared to distal muscles. Thyroid swelling as well as features of thyrotoxicosis may not be overt. A very high index of suspicion is often required for the diagnosis of thyrotoxic periodic paralysis. Treatment of thyrotoxicosis prevents further episodes of paralysis. We report a case of thyrotoxic periodic paralysis in a young male.

Key words: hypokalemic periodic paralysis, thyrotoxic periodic paralysis, thyrotoxicosis, periodic paralysis

Introduction

Periodic paralyses refer to a heterogeneous group of muscle diseases characterized by episodes of flaccid muscle weakness occurring at irregular intervals. Thyrotoxic periodic paralysis an uncommon manifestation of thyrotoxicosis is an acquired form of periodic paralyses. The condition is most common in young males of Asian origin. We here report a case of thyrotoxic periodic paralysis in a young male.

Case Report

29 year old male patient presented to casualty with weakness of limbs more involving the lower limb of 6 hours duration. There were no associated involuntary movements or sensory symptoms or any history suggestive of cranial nerve involvement. There was no history of antecedent fever or diarrhoea or vomiting or myalgia or trauma or vaccination. He gave history of having a feast on the same day and also similar episodes in the past often subsiding in less than a day. He had no similar illness among his family members. On examination his normal general physical examination and had no thyroid swelling. Vitals were stable except for tachycardia (Heart rate: 124/min). His higher mental functions, cranial nerves and sensory system were within normal limits. He had flaccid weakness more in the lower limb (Grade 2/5 in

upper limbs and Grade 3/5 in upper limbs). Reflexes were diminished all over and he had a flexor plantar response. Other systems were within normal limits.

Routine hemogram, urine examination and random blood sugar were within normal limits. Serum electrolytes were as follows; sodium 142mEq/L, potassium 2.3mEq/L, calcium 9.1mg/dl and magnesium 2.1mg/dl. Urine spot potassium: creatinine ratio was 0.9 and electrocardiogram revealed sinus tachycardia and features of hypokalemia. Thyroid function tests were done and the results were; T3 0.01?IU/L and total T4 of 48.7?g/L and total T3 of 658 ng/L. Arterial blood gas analysis revealed no hypoxemia and he had a normal acid base status.

Patient was managed with potassium supplements and he responded promptly. Patient was initiated on Carbimazole and Propranolol also for control of thyrotoxicosis. Patient was discharged in a stable state and he had no episodes of weakness after a follow up period of 6 months by now.

Discussion

Hypokalemic periodic paralysis is one of the rare presentations of thyrotoxicosis. Even though the condition has been reported worldwide, it is com-

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mon in Asians. In Chinese and Japanese people with hyperthyroidism, 1.8–1.9% experience TPP in contrast to North America, where studies report a rate of 0.1–0.2%.⁽¹⁾ The condition typically affects males and the age of onset is typically between 20 - 40years.⁽¹⁾ Various genetic mutations have been demonstrated in patients with thyrotoxic periodic paralysis. Mutations have been reported in the genes coding for potassium voltage-gated channel and sodium channel protein type 4 subunit alpha¹. Studies have demonstrated mutations in *KCNJ18*, an inward-rectifier potassium ion channel in as high as 33% of patients with TPP². The thyroid disease most commonly associated with periodic paralysis is Graves' disease while cases have been reported with toxic nodular goiter, toxic adenoma, TSH-producing pituitary adenoma and Amiodarone induced hyperthyroidism³.

The muscle weakness in thyrotoxic periodic paralysis is considered due to moderate to severe hypokalemia. Hypokalemia in thyrotoxicosis neither results from reduced intake nor increased loss but is due to transcellular shift due to increased Na⁺/K⁺ATPase activity. The mechanism of the increased Na⁺/K⁺ATPase activity is still poorly understood¹. An attack typically begins as muscle pain or cramps followed by rapidly developing paralysis. The flaccid paralysis is often symmetrical and often starts in lower limbs. Proximal muscles tend to be more affected than distal muscles. Muscles supplied by cranial nerves are often spared and respiratory muscle involvement is rare.

Attacks typically resolve in a few hours, even in the absence of treatment^{1,3,4}. Attacks may be precipitated by physical exertion, alcohol consumption or high carbohydrate meals. Symptoms of hyperthyroidism may be present but occur in only half of all cases⁴. A medline based review of thyrotoxic periodic paralysis by Kung et al had found that the features of hyperthyroidism are often subtle in patients with thyrotoxic periodic paralysis¹.

Definitive treatment of thyrotoxic periodic paralysis consists of the management of the underlying thyroid disease. An acute attack is treated with potassium supplementation; however, caution needs to be exercised as rebound hyperkalemia can result as potassium shifts to the extracellular space⁵. β -adrenergic blockers like propranolol may be used to prevent attacks until euthyroid state is achieved. Subsequent attacks may be prevented by avoiding known precipitants but use of potassium supplements is not useful for prophylaxis against further paralytic attacks¹.

Our patient was a typical case with respect to the age, sex and ethnicity. The patient had recurrent episodes of flaccid paralysis making him a typical case of periodic paralysis. He had hypokalemia as well as thyrotoxicosis and the current episode might have been precipitated by the feast he had on the same day. By this case report we would like to emphasize that the possibility of thyrotoxic periodic paralysis should always be kept in mind while dealing with a case of flaccid paralysis especially in young Asian men even in the absence of overt features of hyperthyroidism.

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