

## Thyrotoxic periodic paralysis

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### ABSTRACT

This article aims at highlighting the importance of suspecting thyrotoxicosis in cases of recurrent periodic flaccid paralysis; especially in Asian men to facilitate early diagnosis of the former condition. A case report of a 28 year old male patient with recurrent periodic flaccid paralysis has been presented. Hypokalemia secondary to thyrotoxicosis was diagnosed as the cause of the paralysis. The patient was given oral potassium intervention over 24 hours. The patient showed complete recovery after the medical intervention and was discharged after 24 hours with no residual paralysis. Thyrotoxic periodic paralysis (TPP) is a complication of thyrotoxicosis, more common amongst males in Asia. It presents as acute flaccid paralysis in a case of hyperthyroidism with associated hypokalemia. The features of thyrotoxicosis may be subtle or absent. Thus, in cases of recurrent or acute flaccid muscle paralysis, it is important to consider thyrotoxicosis as one of the possible causes, and take measures accordingly.

**Key words:** Acute flaccid periodic paralysis, genetic predisposition, hypokalemia, oral potassium, thyrotoxicosis

### INTRODUCTION

Periodic paralysis is an uncommon manifestation of thyrotoxicosis.<sup>[1]</sup> It is almost ten times more common amongst Asian males as compared to North Americans. In the latter, the incidence ranges from 0.1 to 0.2%.<sup>[2]</sup> The patient may not present with classical symptoms of thyrotoxicosis, thus timely diagnosis may be hampered. We report such a case of recurrent flaccid paralysis which was caused by a silent thyrotoxicosis.

### CASE REPORT

A 28 year old male presented at our institute with weakness of all four limbs, which started on the day of reporting. He first noticed weakness while getting up from the bed in the morning. Patient recollected presence of muscle cramps the previous night. He had no history suggestive of sensory, cerebellar or cranial nerve involvement. There

was no history of trauma, fever, recent vaccination or high carbohydrate ingestion. He had history of similar episodes in the past one year; each episode recovering within a day. No family member had similar symptoms.

On examination, his vital signs were normal. He had flaccid weakness of all four limbs with power of 2/5 in all the limbs. His reflexes were diminished, and the plantar response was flexion. Rest of the neurological and systemic examination was otherwise normal. Recurrent episodes of flaccid paralysis with quick recovery in the hospital were suggestive of a periodic paralysis.

### Investigations

Laboratory investigations including complete hemogram, urine routine examination and blood sugar, which showed normal results. Serum potassium was 2.3 mEq/L, sodium was 134 mEq/L and serum calcium was 8.2mg/dl, and these findings were consistent with hypokalemic periodic paralysis. Further investigation was aimed at establishing the cause of hypokalemia. From the history, familial periodic paralysis, gastrointestinal disorders causing potassium loss and chronic diuretic abuse were excluded. Urine electrolytes' test was done to rule out renal tubular acidosis. Chest radiograph and ultrasound abdomen were normal. Electrocardiogram showed sinus tachycardia.

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Even though clinically no features of thyrotoxicosis except for tachycardia were present, we proceeded with a thyroid function test which showed a thyroid-stimulating hormone (TSH) level of 0.02  $\mu$ IU/L, total T4 of 68 $\mu$ g/L and total T3 of 700ng/L, suggestive of thyrotoxicosis. Thus, thyrotoxicosis was established as the cause of recurrent hypokalemic paralysis in this patient.

### Treatment and intervention

Patient was treated with oral potassium; about 60 meq over 24 hours. He showed complete recovery with this intervention, and there was no residual paralysis at discharge. His serum potassium was 3.8 meq/L at discharge after 24 hours. He was started on carbimazole 10 mg three times a day and propranolol 40 mg three times a day for the thyrotoxicosis.

## DISCUSSION

Periodic paralysis associated with the thyrotoxic state is a rare and peculiar disorder that affects only the skeletal muscles. The degree of paralysis varies in different attacks. Severe episodes can cause paralysis of all skeletal muscles. Smooth muscles are seldom affected; however, in severe attacks, muscles controlling respiration may get paralysed. Cardiovascular dysfunction is not observed in this disorder. The cardinal feature of a typical attack is muscle weakness and symmetrical paralysis beginning in the proximal muscles of the legs. Hypokalemia (potassium level <3.0 mmol/L) associated with hyperthyroidism is also common.<sup>[3]</sup>

A typical attack of thyrotoxic periodic paralysis lasts from a few hours to several days. Most attacks occur in the morning or evening.<sup>[3]</sup> In this patient, the first two attacks lasted for few hours, and he recovered from the weakness spontaneously; however, the current attack lasted for about 24 hours and required treatment with oral potassium. All the three attacks occurred in the early morning hours. In contrast to familial periodic paralysis, in which the first attack occurs before the age of 10 years, patients with thyrotoxic periodic paralysis are usually in the age group of 20 to 40 years.<sup>[3]</sup>

Asians are more affected by this condition, with one study showing that Polynesians were at 159-fold higher risk compared to white Europeans.<sup>[4]</sup> There is an overwhelming male predominance, with a male-to-female ratio of 77:20.<sup>[5]</sup> The condition has been reported worldwide, including United Kingdom, United States (in whites and African Americans), and South America.<sup>[6-8]</sup> A low serum potassium level has been noted in the majority of patients with thyrotoxic periodic paralysis. Thyroxine is involved in the regulation and maintenance of

normal body and cellular levels of sodium and potassium. Administration of thyroid hormone has been shown to increase the fractional excretion of potassium in various studies.<sup>[9]</sup> Compared with other tissues in the body, human renal tissue contains a higher concentration of messenger RNA for the thyroid receptors alpha-1, alpha-2, and beta. As the thyroid hormone levels in the serum increase, there may be an alteration of the thyroid-receptor expression, which in turn may influence the effects of the hormone on that particular tissue.<sup>[10]</sup>

Asian and Hispanic patients with thyrotoxicosis are particularly susceptible to periodic paralysis. It is also important to remember that, although hypokalemia is common during the acute paralytic episode, the between-attacks' levels of plasma potassium and total body potassium remain within normal limits.<sup>[11]</sup> Precipitating factors of hypokalemic thyrotoxic periodic paralysis include strenuous exercise followed by rest, excessive ingestion of carbohydrate-rich food, administration of insulin or epinephrine, trauma, exposure to cold temperatures, infection, menstruation, and emotional stress.<sup>[3]</sup> The genetic basis of thyrotoxic periodic paralysis also has been intensely studied. An association of TPP with HLA-DRw8 gene in Japanese patients and A2BW22, AW19B17 genes in Chinese patients have suggested the possibility that these haplotypes may serve as genetic markers.<sup>[12,13]</sup> Some other human leukocyte antigen subtypes (B5, BW46) and genetic mutations (KCNE3) also have been seen to be associated with TPP.<sup>[14]</sup> The high incidence of this disorder in Asians suggests that though the basic defect may be genetically determined, it manifests itself only when challenged by thyrotoxicosis.

Definitive treatment of thyrotoxic periodic paralysis consists of the management of thyrotoxicosis by medical, surgical or radioactive iodine therapy. Treatment for an acute attack is potassium administration; however, caution needs to be exercised as excessive doses of potassium can lead to hyperkalemia once potassium shifts to the extracellular space.<sup>[15]</sup> No correlation was observed between the administration of potassium dose and the recovery time.  $\beta$ -adrenergic blockers like propranolol can be used to prevent attacks until euthyroid state is achieved. Other effective preventive measures include a low-carbohydrate diet and using potassium-sparing diuretics.<sup>[16]</sup> Use of potassium supplements is not useful for prophylaxis against further paralytic attacks, and it should not be given to patients between episodes.

A medline based review of TPP over 40 years, which analysed 281 primary articles and 168 references found that the features of hyperthyroidism are often subtle in

patients with thyrotoxic periodic paralysis. The review also notes that the recurrent episodes of paralysis remit with definitive control of hyperthyroidism.<sup>[17]</sup>

In this patient, the diagnosis was delayed due to absence of goitre or any of the other florid features of thyrotoxicosis. Thus this article highlights the importance of suspecting thyrotoxicosis in cases of recurrent periodic flaccid paralysis; especially in Asian men to facilitate early diagnosis. Since the thyrotoxic symptoms may be subtle or absent, and TPP is much commoner, the authors suggest that thyroid function test should form a routine part of assessment of recurrent hypokalemic periodic paralysis in Asian men.

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