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Case Report

Thyrotoxic periodic paralysis with normokalemia: a rare case report

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare etiology for muscle paralysis. It is frequently seen in Asian men. Apart from paralysis, most of them are asymptomatic and are devoid of other clinical features of hyperthyroidism. Due to the rarity of this condition, it is often missed and hence fatal. The metabolic derangement seen in TPP is hypokalemia and is thought to be the pathogenesis of the muscle paralysis. Although in rare clinical encounters, patients with TPP may present with normokalemia. The disorder usually presents in the 3rd decade. The cause for mortality in patients with TPP may be due to respiratory muscle paralysis leading to compromise of the respiratory function or refractory arrhythmias due to hypokalemia. Here we report a rare case of TPP with normokalemia.

Keywords: Periodic paralysis, Thyrotoxicosis, Normokalemia

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a rare etiology for muscle paralysis. It is frequently seen in Asian men. Apart from paralysis, most of them are asymptomatic and are devoid of other clinical features of hyperthyroidism. Due to the rarity of this condition, it is often missed and hence fatal. The metabolic derangement seen in TPP is hypokalemia and is thought to be the pathogenesis of the muscle paralysis. Although in rare clinical encounters, patients with TPP may present with normokalemia. The disorder usually presents in the 3rd decade. The cause for mortality in patients with TPP may be due to respiratory muscle paralysis leading to compromise of the respiratory function or refractory arrhythmias due to hypokalemia.[1] Here we report a rare case of TPP with normokalemia.

CASE REPORT

A 35 year old male presented with gradual onset weakness of both lower limbs for the past two days. There was no history of fever, upper respiratory infection or seizures There were no history suggestive of cranial nerve or sensory or bladder or bowel disturbances. However he had palpitations and heat intolerance for the past few weeks. There was no similar episodes of weakness in the past. Family history of similar illness were negative.

On examination he was conscious and had normal vital signs. There was no goitre or eye signs of hyperthyroidism. Central nervous system examination revealed symmetrical weakness of both lower limbs with a power of 3/5. Mild hypotonia with diminished deep tendon jerks were seen in both lower limbs. Examination of upper limbs were found to be normal. Cranial nerve, fundus, sensory system, cerebellum and autonomic nervous system examination were normal. Other systems were clinically normal.

Laboratory tests revealed normal complete hemogram, blood sugar, lipid profile, renal and liver function tests. Serial measurements of serum potassium levels were found to be normal. Serum magnesium and phosphate levels were within normal limits. Thyroid profile done revealed FT3-10.9 pg/mL (Normal range - 2.3 - 4.1 pg/mL), FT4-1.83 ng/dL (Normal range 0.9 - 1.7 ng/dL), TSH -<0.015 mIU/mL (Normal range - 0.40 - 4.50

mIU/mL) suggestive of hyperthyroidism. Magnetic resonance imaging (MRI) of the brain and nerve conduction study (NCS) were within normal limits. Anti TPO was elevated.

Patient was started on tablet carbimazole 10 mg twice daily, tablet propranalol 10 mg twice daily. Muscle tone and power improved over a week and was able to walk without support.

DISCUSSION

Thyrotoxic periodic paralysis (TPP) is a recognized disorder in Asian residents. The incidence of TPP is approximately 2% in patients with thyrotoxicosis.[2] Though Thyrotoxic periodic paralysis is a curable disorder, can be fatal if misdiagnosed especially when associated normokalemia and is frequently diagnosed as Guillain-Barré syndrome, hypomagnesemia or hypophosphatemia or functional disorder.[3,4]

The clinical features include acute flaccid symmetrical weakness that usually involving the lower limbs. The most frequent precipitating factors are strenuous exercise, excessive alcohol intake and carbohydrate rich meal. Patients with TPP usually exhibit the weakness involving the lower limbs in the early hours of the day, that may be or not be preceded by muscle cramps. The reflexes are usually diminished but can be normal at times with no sensory and bladder involvement

The pathogenesis of normokalmic TPP is not well understood. Patients with excessive secretion of thyroid hormones cause overstimulation of Na +/k+ ATPase pump which is present in the in cell membranes of skeletal muscles, increased insulin levels and stimulation of the adrenergic receptors that leads to potassium shift into the intracellular compartment, thereby causing hypokalemia and flaccid muscle paralysis that may last upto 36 hours.[4,5]

Previous studies report that the potassium levels can be normal as seen in our case, or even higher in patients with TPP, making it a challenge for the clinicians.[3,5,6] Wu et al reported two patients with normokalemic TPP with potassium of 3.8 and 4.7, respectively at the time of flaccid paralysis.[3] González-Treviño and Rosas-Guzman reported a patient with recurrent normokalemic flaccid paralysis eventually diagnosed with Graves' disease.[7]

Management of patients with TPP includes anti thyroid drugs, beta blockers and supplementation of potassium especially in the background of hypokalemia. Betablockers directly acts on Na + K + ATPase. Potassium correction must be cautious as patients can land up in hyperkalemia.[8] Non selective beta blockers such as propranolol is used to ameliorate acute attacks and is used for secondary prophylaxis.[5] However the mainstay of treatment in patients with TPP is anti thyroid drugs like carbimazole, radioiodine ablation or surgery.[4] Review of the literature shows that few authors have treated patients with acetazolamide, spirinolactone, and prophylactic low dose potassium supplementation.[9]

Serial monitoring of potassium levels is mandatory as patients presenting with normokalemia can develop hypokalemia later leading to worsening of the weakness. A case report published by Kufs et al showed that a patient with TPP who presented with normokalemia developed worsening paralysis few hours later due to hypokalemia.[10]

Patients must be educated about the precipitating factors and advice for alcohol abstinence, avoid high carbohydrate diet, stress, strenuous exercise as these can lead to acute attacks.[4]

CONCLUSION

Clinicians must keep TPP as a differential diagnosis for patients presenting with acute flaccid paralysis. Patients with TPP usually present with hypokalemia at the time of paralysis. But in rare situation, normokalemia can be seen during presentation. Serial monitoring of potassium levels is warranted in those patients. TPP associated with normokalemia is often missed and can be life threatening. We report this rare disorder as early identification and appropriate management of this treatable endocrine disorder is the cornerstone.

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