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THYROTOXIC PERIODIC PARALYSIS: A CASE REPORT

Endocrinology

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare complication of hyperthyroidism which presents with hypokalemia and muscle weakness, more commonly seen in Asian men than women. The presence of elevated level of triiodothyronine (T3) and thyroxine (T4) and hypokalemia are the guiding signs towards the diagnosis. Prompt diagnosis and treatment are very important for preventing further damage. Treatment protocol includes correction of hypokalemia and achieving as well as maintaining the euthyroid state. We report the first such case from our hospital.

KEYWORDS

Thyrotoxic Periodic Paralysis, Hypokalemia, Thyrotoxicosis

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a transient but potentially serious disorder characterized by triad of thyrotoxicosis, muscle paralysis and acute hypokalemia. TPP is most commonly seen in Asian population, with an incidence of approximately 2% in patients of thyrotoxicosis and more in males than females.[1] Correct and quick diagnosis of TPP is necessary not only in managing the case with non-selective beta blockers and correction of hyperthyroidism, but also prevents the risk of rebound hyperkalemia due to excessive potassium replacement. [2]

CASE REPORT

A 30 year old male presented in our emergency department with the symptoms of generalized weakness that was more in both the lower extremity. These symptoms started in the evening as he noticed difficulty in walking till the bathroom. He also gives similar history one year back but did not get the previous reports and prescription and had taken treatment from some other hospital. There was no history of fever, diabetes, hypertension or seizures. Also his bowel and bladder were not involved.

On physical examination, there was a swelling in the neck which indicated enlargement of thyroid with firm consistency and without any nodule. The muscle power in lower limb was 3/5 and tendon reflexes were 0/4. The patient also exhibited fine tremors. There was no sign of sensory or cranial nerve deficits and no exophthalmos.

On admission, his serum potassium was 1.9 mEq/L (normal range, 3.6-5.0 mEq/L), total thyroxine (T4) was >24.86 (5.1-14.10 µg/dL), total triiodothyronine (T3) was >6.51 (0.80-2.0 ng/ml), TSH level 0.24 uIU/mL (0.35-94) and serum phosphorus was 4.2 mg/dL (normal range, 2.6-4.5 mg/dL). His serum magnesium was 1.6 mg/dL (normal range, 1.7-2.6 mEq/L). Urine potassium per 24 hours was 92 mmol/day (reference range, 25–125 mmol/day). Heart rate was 100 beats per minute. Urgent electromyography and nerve conduction studies were normal. The following investigations showed normal results: complete blood count, hemoglobin, C-reactive protein, erythrocyte sedimentation rate, serum electrolyte, chest X-ray and liver function test. Lactate dehydrogenase (LDH), serum creatine phosphokinase (CPK) and rheumatoid factor were also normal.

The patient was diagnosed as hypokalemic paralysis and was given 40 mEq of intravenous potassium chloride in the emergency department and was then started on a normal saline infusion with 20 mEq/L of potassium. He was also placed on an oral potassium replacement, which resulted in resolution of his lower-extremity paralysis.

On the second day of admission, his serum potassium level increased to 4.2 mEq/L. The thyroid-stimulating hormone level was <0.02 mU/L (reference range, 0.50–6.80 mU/L), and the free thyroxine (T_4) level was 4.56 ng/dL (reference range, 0.89–1.76 ng/dL). Thyrotoxicosis was diagnosed, and the patient was given propranolol 40 mg three times a day. The patient's thyroid-stimulating immunoglobulin level was elevated, and he was started on carbimazole 20 mg per day. The weakness in lower limbs resolved and the patient was discharged on the fourth day.

DISCUSSION

The high incidence of TPP in Asians is genetic and is associated with the presence of HLA-DRw8 gene, but the precise pathogenesis of TPP still remains unclear.[3] Hypokalemia in TPP may be attributed to the intracellular shift of potassium and not the total body depletion. In patients of TPP the Na+/K+ ATPase activity is significantly high in muscles and platelets.[4] Hyperthyroidism may cause release of adrenalin which may cause Na+/K+ ATPase pump activation leading to increase cellular uptake of potassium.[5] Treatment of hyperthyroid state and correction of hypokalemia are the mainstay for managing TPP. However, there is a risk of rebound hyperkalemia due to release of potassium from the cells after recovery. There is an evident association between the dose of potassium administered and the degree of rebound hyperkalemia.[6] The concomitant use of non-selective beta blocker like propranolol in treating TPP prevents the intracellular shift of potassium by inhibiting the adrenergic stimulation of Na+/K+ATPase. Antithyroid drugs, thyroidectomy or radioiodine therapy may be chosen for achieving and maintaining the euthyroid state.

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