Case Report

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Thyrotoxic Periodic Paralysis

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ABSTRACT
Thyrotoxic periodic paralysis (TPP) is an alarming and potentially lethal complication of hyperthyroidism characterised by muscle paralysis and hypokalaemia. It is often not recognised when first seen because of lack of familiarity with the disorder and partly due to the subtleness of thyrotoxicosis. Early diagnosis and treatment can prevent severe cardiopulmonary complications. We hereby report a male patient who was evaluated and diagnosed to have TPP.
KEYWORDS
Thyrotoxic Periodic Paralysis, hypokalaemia, thyrotoxicosis

Introduction

Thyrotoxic periodic paralysis (TPP) is an uncommon disorder characterised by simultaneous thyrotoxicosis, hypokalaemia, and paralysis that occurs primarily in males of South Asian descent.[1] Many affected patients do not have obvious symptoms and signs of hyperthyroidism and hence may be misdiagnosed or overlooked on presentation.[2] We hereby report a male patient who presented to us with weakness of all four limbs. The patient was evaluated and diagnosed to be having TPP.

Case History

A 30-year-old male patient, who was an agriculturist by profession, presented with weakness of all four limbs of one-day duration. The weakness first appeared in his lower limbs and then in the upper limbs. There were no sensory symptoms or bladder involvement. He was not a known hypertensive, diabetic or thyrotoxic patient. He was not on any medication for any significant illness.

On general physical examination, there was no pallor, icterus, cyanosis, clubbing, lymphadenopathy or pedal oedema. Multinodular goitre was noted on thyroid examination. There was no exopthalmos, lid lag, pretibial myxoedema or other signs of thyrotoxicosis. Thyroid bruit was absent. Pulse rate was 96/minute, blood pressure of 140/80mmHg, and respiratory rate 18/minute. On central nervous system examination, the higher mental functions and cranial nerve examination were within normal limits. Motor system examination showed the presence of flaccid quadriparesis with areflexia. Sensory system examination was within normal limits. Cardiovascular and respiratory system examination were normal.

Investigations revealed: haemoglobin (Hb) -13.1 gm%, total count (TC) - 11,400/cmm, platelet count - 49,000/cmm, random blood sugar (RBS) - 110mg/dl, blood urea - 29 mg/dl, serum creatinine - 0.8 mg/dl. Serum electrolyte profile showed sodium - 143 mEq/L, potassium - 2.2mEq/L, chloride - 112mEq/L. Serum calcium and magnesium levels were within normal limits. Electrocardiogram (ECG) was normal. Human Immunodeficiency Virus (HIV) ELISA was non reactive. Bone marrow biopsy and ultrasonography of abdomen were normal. Fine Needle Aspiration Cytology (FNAC) of thyroid showed features of hyperplastic colloid goitre. Ultrasonography of thyroid showed hyperechoic small nodules in both lobes as well as isthmus suggestive of multinodular goitre. Thyroid profile was: total T3 - 2.34 (normal: 0.60 - 1.81ng/ml), total T4 - 13.9 (normal: 4.5 - 10.9 mcg/dl), thyroid-stimulating hormone (TSH) - 0.01 (normal: 0.35 - 5.5IU/ml). Antithyroid antibodies and antiplatelet antibodies were negative. Nerve conduction study was normal. A final diagnosis of TPP with idiopathic thrombocytopenia was made.

The patient was administered 40mmol potassium chloride intravenously. He was treated with tablet carbimazole 10mg three times a day and tablet propanolol 10mg twice a day. The patient's weakness in all four limbs improved dramatically within an hour after potassium chloride administration. As he had persistent thrombocytopenia during his stay in hospital, he was commenced on tablet prednisolone (1mg/kg body weight). His platelet count normalized in one month after which the steroid dose was tapered and stopped.

Discussion

TPP is an uncommon disorder characterised by simultaneous thyrotoxicosis, hypokalaemia and paralysis that occurs primarily in males of South Asian descent. The overall incidence of TPP in Chinese and Japanese thyrotoxic patients is 1.8% and 1.9% respectively.[3, 4] Sporadic cases have been reported in non-Asian populations such as Caucasians, Afro-Americans, American Indians and Hispanics. With population mobility and admixture, TPP is becoming more common in Western countries. Many affected patients are in the age group of 20 - 40 years and do not have obvious symptoms and signs of

hyperthyroidism.[5] The attack is characterised by recurrent, transient episodes of muscle weakness that range from mild weakness to complete flaccid paralysis. The proximal muscles are affected more severely than distal muscles. Attacks usually first involve the lower limbs, and progress to the girdle muscles and subsequently the upper limbs. Sensory function is not affected. Although patients can present with quadriparesis that resembles Guillain-Barre Syndrome or transverse myelitis, the bladder and bowel functions are never affected. Patient may experience recurrent episodes of weakness that last from a few hours up to 72 hours with complete recovery between the attacks. In the majority of patients, deep tendon jerks are markedly diminished or absent although some patients may have normal jerks.

Patients with TPP usually experience the attacks a few hours after a heavy meal or in the early morning hours upon waking. More than two-thirds present to the emergency department between 2100 and 0900 hours; hence it was initially described as nocturnal palsy or night palsy.[6] It has been shown that plasma glucose and insulin responses to meals are markedly higher in the evening than in the morning in control subjects. Such a phenomenon suggests a possible mechanism for the nocturnal preponderance of TPP. Another explanation could be the circadian rhythmicity of many hormones reaching their peak levels during sleep. Hypokalaemia is considered to be the most consistent electrolyte abnormality in TPP and a hallmark of the syndrome along with hyperthyroidism. It has been demonstrated that hypokalaemia is a result of potassium shift into cells and that it is not caused by total body potassium depletion.[7]Patients with thyrotoxic periodic paralysis have an underlying predisposition for activation of Na+/K+-ATPase activity either directly by thyroid hormones or indirectly via adrenergic stimulation, insulin or exercise. Increased Na+-K+ ATPase activity is postulated to contribute to hypokalaemia.[8]

The majority of cases of hyperthyroidism associated with thyrotoxic periodic paralysis are due to Graves disease although other conditions including thyroiditis, toxic multinodular goitre, toxic adenoma, TSH secreting pituitary tumour, ingestion of T4 and inadvertent iodine excess have also been implicated.[9] Assaying of thyroid function in patients with hypokalaemic paralysis distinguishes thyrotoxic periodic paralysis from other forms of hypokalaemic periodic paralysis. Thyrotoxic periodic paralysis occurs only in the presence of hyperthyroidism and is abolished when thyroid hormones are normalised.

Immediate therapy with potassium supplementation and beta-adrenergic blockers can prevent serious cardiopulmonary complications and may hasten recovery of periodic paralysis.[10] Potassium chloride is given intravenously and/or orally. Regular potassium supplementation as prophylaxis against further paralysis when the patient has normal serum potassium level is ineffective. Effective control of hyperthyroidism is indicated to prevent recurrence of paralysis.

Conclusion

To conclude, although the association of thyrotoxicosis and periodic paralysis has been well known, TPP is often not recognised when first seen because of lack of familiarity with the disorder and partly because of the subtleness of thyrotoxicosis. When a young male of South Asian descent is initially seen with severe lower limb weakness or paralysis, TPP should be considered in the differential diagnosis and investigated for its presence since it is a curable disorder that resolves when euthyroid state is achieved.

Competing Interests
None declared
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