

CASE OF THYROTOXIC PERIODIC PARALYSIS

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is an uncommon disorder characterized by simultaneous thyrotoxicosis, hypokalaemia, and paralysis. The definitive therapy for TPP includes treatment of hyperthyroidism with antithyroid medications, surgical thyroidectomy, or radioiodine therapy. After initiation of definitive therapy, patients should avoid precipitating factors and continue propranolol until a euthyroid state is achieved to prevent recurrence. The present case report will focus on thyrotoxic periodic paralysis.

KEYWORDS: Paralysis, Treatment, Antithyroid medications.

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is an uncommon disorder characterized by simultaneous thyrotoxicosis, hypokalaemia, and paralysis that occurs primarily in males of South Asian descent.^[1] Patients are usually not showing any signs or symptoms of hyperthyroidism and hence maybe misdiagnosed or overlooked but time is of the essence in such cases. Hence when a young male presented with acute weakness of all limbs, he was immediately evaluated for Thyrotoxic periodic paralysis.

CASE REPORT

A 26 year old male was apparently well 2 days back when he presented with complaints of pain in groin and inner thighs along with malaise since 2 days, the patient then developed difficulty in walking since 3am last night before he went to bed and ignored it which progressed to inability to move all limbs the next day at 7am when he woke up. He was carried from home by his family members to the emergency department of Subharti hospital at 9am.

The pain in the thighs was dull in intensity, non-progressing, non-referring which he ignored as he is a labourer and often gets aches due to work. The associated malaise was also thought to be a part of being overworked. Until he developed difficulty in walking which he noticed while going to bed at 3am after returning from a wedding where he had a heavy meal

and some alcohol intake the night before and for the pain he took he took an NSAID which he thought would be relieved on rest but when he woke up the other day he was unable to move his limbs at all.

On examination in the emergency room his vitals were stable except patient had tachycardia of 110 bpm. No pallor, icterus, lymphadenopathy, cyanosis, clubbing, JVP and hydration was normal. CNS examination revealed that the patient was conscious oriented to time, place, person, B/L pupils were NSNR and B/L plantars were mute. No neck rigidity and kernigs sign was absent. Urinary and bowel control was present. Power was 0/5 in all limbs and all DTRs were absent. Tone was decreased in all limbs. Sensory system was intact. Cranial nerves were intact. Respiratory, cardiac and abdomen examination was unremarkable.

No history of recent fever, vomiting, loose stools, any trauma, abnormal body movements, loss of consciousness or any similar episode in the past. No history of any chronic illness. Patient was a non-smoker, occasional-alcoholic with normal sleep, bowel, bladder and appetite.

Investigations revealed ABG was normal except his potassium levels were 2.2 mmol/l. CBC revealed hb of 14.9 g/dl and WBC count of 17.5×10^3 mm with neutrophils of 88%. LFT was within normal limits except ALP was mildly raised 124U/L. KFT showed urea 42

mg/dl, creatinine 0.9 mg/dl, sodium 140mmol/l where as potassium was 1.9 mmol/L. ESR was 65. Serum calcium, magnesium and phosphorus were within normal range. Radiological investigations, chest xray, NCCT head, cervical spine x-ray and usg abdomen were normal. ECG revealed sinus tachycardia with a prolonged PR interval and benign PVCs.

The alarming level of potassium 1.9 mmol/L drew our suspicion to Hypokalemic Periodic Paralysis. Supplementing IV potassium as well as oral supplementation was started following which the limb weakness started to improve in the next 24 hours. TSH levels were sent which were 0.02 microIU/mL (normal 0.35-5.5), free T4 was 39.63 pmol/L(normal 11.5-22.7) and free T3 was 6.41pmol/L (3.5-6.5). His anti-TPO levels were 246U/mL (normal <60). This confirmed our diagnosis of Hypokalemic periodic paralysis due to Graves Disease (thyrotoxicosis). Patient was immediately started on Neomercazole 10mg TDS the 2nd day and propranolol 20mg/day TDS. With physiotherapy and reassurance along with maintaining hydration and normal levels of potassium, the patient drastically improved and power was now 4/5 in all limbs. He was able to walk on the 5th day of the treatment and was discharged with stable vitals. 1 week as well as a 4 week follow up revealed complete resolution of his symptoms. Patient was continued on anti- thyroid as well as beta blockers with dose alteration in the 6 month follow up.

DISCUSSION

This case was one of the typical ways TPP presents itself. Many affected patients are in the age group of 20 - 40 years and do not have obvious symptoms and signs of hyperthyroidism.^[2] Patients present with acute onset of proximal symmetrical ascending lower-extremity muscle weakness during the early morning hours or while resting after strenuous exertion and a high-carbohydrate meal. Acute episodes may be preceded by muscle aches, cramps, and muscle stiffness.^[3] Other precipitating factors include trauma, exposure to cold, emotional stress, infection, alcohol ingestion, menses, and drugs like diuretics, insulin, or steroids. This patient had a history of strenuous activity as a labourer having muscle cramps and a high carbohydrate meal and alcohol intake at night after which he developed the paralysis the next day.

Not only the clinical presentation but also the investigations were crucial for the early diagnosis and treatment of the patient. The extremely low levels of potassium made the differential diagnosis stem from familial hypokalemic periodic paralysis, paramyotonia congenita, potassium-aggravated myotonia, myotonia congenita (MC), both recessive and dominant MC, hyperaldosteronism and physiologically similar states, diuretic abuse, and myasthenia gravis. The response to the correction of potassium with following report of thyrotoxicosis narrowed the diagnosis to TPP.

Electrocardiographic findings may be characteristic of hypokalemia, with increased P-wave amplitude, prolonged PR interval, widened QRS complexes, decreased T-wave amplitude, and U waves. Unlike hypokalemia from other causes, sinus tachycardia predominates in patients with TPP.^[4] Other electrocardiographic abnormalities include atrioventricular block, atrial fibrillation, ventricular fibrillation, and asystole.^[5] The patient had a prolonged PR interval and benign PVCs along with sinus tachycardia.

The definitive therapy for TPP includes treatment of hyperthyroidism with antithyroid medications, surgical thyroidectomy, or radioiodine therapy. After initiation of definitive therapy, patients should avoid precipitating factors and continue propranolol until a euthyroid state is achieved to prevent recurrence.^[6,7] TPP is curable once a euthyroid state is achieved. In the setting of TPP, propranolol, a nonselective beta-blocker, prevents the intracellular shift of potassium and phosphate by blunting the hyperadrenergic stimulation of Na⁺/K⁺-ATPase. It has been observed that propranolol given alone either orally (3 mg/kg) or intravenously normalizes serum potassium levels on an average of 120 minutes; hence, initial therapy for stable TPP should include propranolol.^[8] This was included in his initial management and he was given antithyroid drugs for thyrotoxicosis.

The patient completely recovered with the correction of potassium and treatment of the thyrotoxic state. A proper follow up of his thyroid functions with knowledge of the triggering factors, such episodes can be prevented in future.

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