Case Report

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A rare complication of a relatively common disease: periodic paralysis caused by thyrotoxicosis

Abhishek Chanda, Mainak Mandal, Suman Sarkar*, Nirmalya Roy

Department of Medicine, KPC Medical College and Hospital, Kolkata, West Bengal, India

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*Correspondence: Dr. Suman Sarkar.

E-mail: sumansarkar32@gmail.com

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ABSTRACT

Thyrotoxic periodic paralysis is a sporadic form of hypo-kalemic periodic paralysis and it is most commonly presents as sudden onset weakness in the proximal muscles. It is a disorder most commonly seen in Asian men which is characterised by abrupt onset of hypo-kalemia and paralysis and it is primarily affects lower extremities and is secondary to thyrotoxicosis. It is due to an intracellular shift of potassium induced by thyroid hormone sensitisation of Na/K ATPase rather than depletion of total potassium. The absence of a family history, male predilection and, presentation in second to fourth decade of life and signs of thyrotoxicosis and sinus tachycardia help in the diagnosis of the disorder. The age of onset is later and corresponded with age of incidence of thyrotoxicosis. It is very essential to diagnose TPP early and treat appropriately.

Keywords: Thyrotoxicosis, Periodic paralysis, Hypo-kalemia

INTRODUCTION

Thyrotoxic periodic paralysis is a disease state characterised by acute onset profound proximal muscle weakness and severe hypokalemia in patients with thyrotoxicosis. It is characterised by the triad consist of episodic muscle paralysis, thyrotoxicosis and hypokalemia without total body potassium deficit. Common triggering factors are carbohydrate rich food, estrogen, epinephrine, high salt intake, stress and drugs.² It mainly affects Asian people, later age of onset with incidence of approximately 2% people in patients with thyrotoxicosis.^{3,10} It is commonly misdiagnosed with familial periodic paralysis in western countries due to its similarities. Familial periodic paralysis is an autosomal dominant disorder caused by a defect in the gene coding for L type of calcium channel 1 subunit (CACNA1S). Some evidences showing the role of a genetic mutation in channel Kir2.6 leading to pathogenesis of this disease.4 The neuromuscular presentation of TPP and FPP are similar so to enhance diagnosis of TPP doctors need to look for small signs of hyperthyroidism and treat accordingly.

CASE REPORT

We present an interesting case of a 57-year-old gentleman who presented to us in the outpatient department with chief complaint of multiple aches and pains associated with generalised weakness for last 2-3 years. On detailed history taking he reports that he had frequent attacks of severe weakness following diet rich in sweets. He also says severe aching pain over upper torso very often, weakness associated with inability to raise his arms over the shoulder or to raise from squatting position and weakness also present in both upper and lower limbs. The weakness lasted for short periods and often he had to go to hospital and had to be given IV fluids but no other details available. He often complains of soreness of back, arms and thighs after recovery. Episodes were particularly frequent during fasting and became more frequent recently. He denied any history of weakness of facial muscles, diurnal variation of weakness, weakness of forearms, fingers, legs and toes. There is no evidence of tingling and numbness or evidence of burns or trophic ulcers. He has no known systemic comorbidities. On examination he was alert conscious cooperative, pulse-98/min, Bp-120/70 mm of hg, normal temperature, SpO₂ of 98% in room air. His BMI is 22.1 kg/m² but there is no icterus, no pallor, no cyanosis, no koilonychia, no lymphadenopathy or prominent neck veins. There was grade 2 goiter present-both visible and palpable (Figure 1). On systemic examination normal S1 and S2 heard, bilateral vesicular breath sound, per abdomen was soft and non-tender, all higher mental functions were intact.

Table 1: Showing all biochemical parameters.

Parameters	Values
Haemoglobin	11.1 g/dl
Total leukocyte count	6030
Platelets	2.1 lakh
ESR	30 in 1 st hr
Sodium	138 mg/dl
Potassium	2.5 mg/dl
Magnesium	2.1
Urea	22 g/dl
Creatinine	1.2 g/dl
TSH	<0.01 uIU/dl
fT4	5.73(N-0.8-2) pg/dl
Total T3	(N-0.52-1.85) ng/ml
TSH receptor antibody	19.63.
Urine spot sodium	43.3 mmol/l
Urine spot potassium	9.7 mmol/l
Serum LDH	186 U/l
ABG PH	7.522
ABG pO ₂	98
ABG pCO ₂	42.2
ABG HCO ₃	33.5
Fasting blood sugar	158 mg/dl

His liver function test and urine routine examination were normal with no growth in urine culture and sensitivity.

ECG was normal, nerve conduction test-reduced amplitude of action potential. EMG-Electrically silent. USG whole abdomen and echo 2 D both are normal

Thyroid scintigraphy: Avid trapping function, possibility of subclinical graves (Figure 2).



Figure 1: The gentleman with grade 2 goiter.

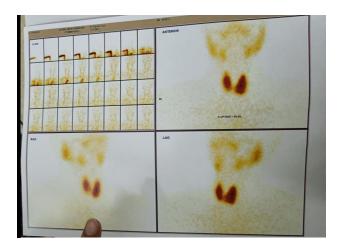


Figure 2: Thyroid scintigraphy showing avid trapping function possibility of subclinical graves.

He was initially treated with IV and oral potassium replacement along with spironolactone and propranolol. Then after confirming the diagnosis with thyroid scintigraphy report we started him carbimazole 10 mf thrice daily. His weakness gradually recovered and discharged with medication and instructed him for regular follow up.

DISCUSSION

Thyrotoxic periodic paralysis is a life-threatening condition, though it's a complication of thyrotoxicosis but it is not related to the etiology, severity and duration of thyrotoxicosis.⁵ The family history is usually absent. The events that lead to this condition in thyrotoxicosis is complex. They include hyperthyroidism, genetic predisposition and exaggerated insulin response and hyperadrenergic state. TPP has been gradually documented increasingly in Asian population and Hispanic population in United States. Thyrotoxic periodic paralysis has a incidence of 2% in asians compared to only 0.1-0.2% in non-Asians with male predominance which is quite rare in thyroid disease presentations. 6-8 The initial presentation is often delayed due to subtleness of the clinical features of thyrotoxicosis and similarities of the paralysis with more common conditions. 9 Diagnosis of this thyrotoxic periodic paralysis should be suspected in those patients presented with paralysis associated with hyperthyroidism and hypokalemia and must be distinguished from other causes of acute paralysis like myasthenia crisis, GB syndrome, botulism etc. 10 Increased awareness among physicians about this will result in high chance early diagnosis, correct treatment and prevention of recurrent episodes.

CONCLUSION

An alarming and serious complication of thyrotoxicosis and it is increasingly common in Asian as well as in western countries and can be related to genetic predisposition and exaggerated insulin response and

hyperadrenergic state. While evaluating acute paralysis with hypokalemia thyrotoxicosis has to be kept in mind because subtleness of underlying thyrotoxicosis can delay diagnosis. Increased awareness among physicians about this will result in high chance early diagnosis, correct treatment and prevention of recurrent episodes.

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