Original Research Paper

Volume - 15 | Issue - 08 | August - 2025 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

General Medicine

RAPID REVERSAL OF PARALYSIS

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KEYWORDS :

INTRODUCTION:

Hyperthyroidism is associated with several muscular disorders, including thyrotoxic myopathies, myasthenia gravis, Thyroid ophthalmoplegia, and periodic paralysis. Thyrotoxic periodic paralysis (TPP) is a rare endocrine disorder, it includes sudden onset of muscle weakness, hypokalemia, and hyperthyroidism. The absence of a family history of paralysis, male sex, presentation in the second to fourth decades of life, systolic hypertension, and signs of thyrotoxicosis help in the diagnosis of TPP. We report a case of a young hypertensive male presenting to the emergency department with a sudden onset of painless muscle weakness in all 4 limbs on waking from sleep.

CASE REPORT:

A 34-year male office clerk presented to the emergency department with a sudden, painless weakness involving all four limbs. The weakness began in the lower limbs around 6 a.m, as he attempted to get out of bed and progressed rapidly to the upper limbs within a minute. He was brought to the hospital on a stretcher. There was no history of trauma, recent immunization, strenuous physical activity, fever, heavy carbohydrate intake, or emotional stress. He admitted to alcohol consumption the night before.

The patient had been diagnosed with hypertension two years prior and was on a daily dose of Telmisartan 40 mg. He also had a history of hyperthyroidism diagnosed two years ago and was initially prescribed Carbimazole 10 mg twice daily. After one year, patient got his Thyroid functions self evaluated,that showed elevated TSH which probed him to consult an local practioner who is an unqualified practitioner due to elevated TSH levels (exact values unavailable) and was switched to Thyronorm 75 mcg daily, which he had been taking for the past year. He denied any prior similar episodes or a family history of paralysis. Classical signs of hyperthyroidism were not evident upon presentation.

On examination, the patient appeared well-nourished. His pulse was 131 beats per minute, and blood pressure was 150/80 mmHg. Neurologically, he demonstrated decreased muscle tone and strength graded at 1/5 in all limbs, with diminished deep tendon reflexes. However, cranial nerve examination and sensory testing were unremarkable.

An ECG revealed sinus tachycardia, ST segment depression, T-wave flattening, and prominent U-waves. Laboratory investigations showed significant hypokalemia (serum potassium 2.2 mEq/L), low TSH (<0.01 µU/mL), elevated FT3 (7.28 pg/mL) and FT4 (5.45 ng/dL), and reduced urinary potassium excretion (9.29 mmol/L). Other parameters, including hemoglobin, renal function, liver enzymes, and ABG, were within normal limits (see Table 1).

The patient was promptly treated with intravenous potassium replacement. Within 4 hours, his muscle power began to improve, and complete recovery was observed by 12 hours. He was subsequently started on propranolol 40 mg two times daily and carbimazole 10 mg three times daily to manage the underlying thyrotoxic state.

Table 1. Laboratory Assessment

Day of illness	Day 0	4 hrs after admission	16 hrs after admission
Hemoglobin (gm%)	13.3		
Total counts(cells/uI)	7200		
Platelets (lack/uI)	2.65		
Total bilirubin (mg/dl)	0.6		
Direct bilirubin(mg/dl)	0.3		
Indirect bilirubin (mg/dl)	0.3		
Aspartate aminotransferase (SGOT)(U/I)	52		
Alanine aminotransferase (SGPT)(U/I)	29		
alkaline phosphatase (U/L)	78		
Total protein (g/dl)	7.6		
Albumin (g/dl)	4.2		
Serum urea (mg/dl)	27		
Serum creatinine (mg/dl)	0.7		
Urine			
Bilirubin	-ve		
Urobilinogen	-ve		
Blood	-ve		
Glucose	-ve		
Ketones	-ve		
Leucocytes	-ve		
Protein	-ve		
Ph	6.5		
TSH µU/mL	<0.01		
FT3pg/ml	7.28		
FT4ng/dl	5.45		
Sodium meq/L	141		
Potassium meq/L	2.2	3.7	4.6
Chloride meq/L	101		
Urine potassium mmol/L	9.29		
pH	7.405		

pCO2 mmHg	39		
pO2 mmHg	88		
$HCO3^-$ meq/L	23		
SaO2	98%		

DISCUSSION:

Rosenfeld demonstrated the relationship between thyrotoxicosis and recurrent paralysis in 1902.Approximately 2% of patients with hyperthyroidism have TPP, which is more common in Asian and Hispanic people. (1). Non-Hispanic Caucasians are estimated to have a 0.1–0.2% incidence of TPP. Despite being more prevalent in women, TPP is 20 times more common in males than in women (2).

Graves' disease remains the most frequently associated cause of TPP. Other etiologies include toxic multinodular goiter, excessive thyroid hormone intake, iodine-induced thyrotoxicosis, solitary toxic adenoma, lymphocytic thyroiditis, TSH-secreting pituitary tumors, and amiodarone-induced hyperthyroidism.(3)

Genetic predispositions have been proposed to explain the higher incidence of TPP in Asian populations. Certain HLA haplotypes, such as HLA-DRw8 in Japanese and HLA-A2, B-17, Bw22, and Aw19 in Chinese and Singaporean individuals, are associated with increased risk(4). Additionally, mutations affecting potassium channels-especially Kir2.6 (encoded by the KCNJ18 gene)-have been found in a significant portion of patients with TPP. These mutations disrupt potassium handling within muscle cells, which can trigger paralysis during thyrotoxic states.(5)

When comparing individuals with thyrotoxicosis with periodic paralysis to those without paralysis, the skeletal muscle cells and the Na+, K+-ATPase pump activity were higher in the former group. The biggest reservoir of potassium in the body is skeletal muscle, which is also crucial for maintaining extracellular potassium homeostasis. It is made possible via inward rectifying potassium channels and Na-K ATPase.(6)

Additionally, thyroid hormone directly activates the Na+ K+-ATPase pump and increases the number and sensitivity of β-receptors, which in turn enhances catecholamine-mediated potassium absorption.(7) Pump acivity is further amplified by elevated testosterone levels,this explains why prevalence of TPP is more in men than women.Insulin and catecholamines also play a role by promoting potassium movement into cells. These mechanisms contribute to a significant drop in serum potassium during acute episodes, although total body potassium remains unchanged.

KCNJ18, which codes for the inwardly rectifying K+ channel Kir2.6 unique to skeletal muscle, has been shown to have mutations that cause Kir2.6 normal amino acid sequence to be substituted with the amino acids V168M, R43C, and A200P. The efflux of potassium is inhibited by mutations in catecholamines, insulin, and Kir2.6.

Most patients with TPP are males aged 20–40 years, and episodes typically occur at night or early morning. Precipitating factors include alcohol intake, carbohydrate-rich meals, stress, infections, strenuous physical activity, and even menstruation. Muscle weakness typically begins proximally in the lower limbs and can progress to flaccid quadriplegia. Sensory functions, cranial nerves, and mental status usually remain intact. Severe cases may be associated with urinary retention and gastrointestinal symptoms due to smooth muscle involvement. At the time of presentation typical features of hyperthyroidism may or may not be present.

Electrocardiographic findings often reflect hypokalemia, including sinus tachycardia, low-amplitude or flattened T waves, ST segment depression, and prominent U waves. Other possible findings include prolonged PR intervals and patterns consistent with left ventricular hypertrophy.

Thyroid functional tests help in the definitive diagnosis of TPP, which helps in differentiating TPP from HPP (Hypokalemic periodic paralysis) which is a diagnosis of exclusion. Laboratory tests usually show low serum potassium and phosphate, with minimal urinary excretion of potassium, suggesting an intracellular shift rather than renal loss. ABG and renal function are typically normal. Hypophosphatemia can further exacerbate muscular weakness during an attack, while phosphate levels may rise between episodes.(3)

Management includes cautious potassium supplementation to correct hypokalemia while avoiding rebound hyperkalemia. High-dose non-selective beta-blockers like propranolol (3–4 mg/kg) can rapidly reverse symptoms, especially when used in combination with potassium replacement. The cornerstone of long-term management is restoring and maintaining a euthyroid state, which prevents recurrence.(8)

CONCLUSION:

TPP is a reversible but potentially dangerous complication of untreated or inadequately managed hyperthyroidism. Although rare, it should be suspected in any patient—especially males—who present with acute flaccid paralysis and hypokalemia, even in the absence of overt hyperthyroid symptoms. Prompt recognition and appropriate management, including potassium replacement and beta-blockade, can lead to full recovery. However, long-term prevention hinges on achieving and sustaining a euthyroid state. Early diagnosis and correct treatment are essential to avoid life-threatening complications and recurrence.

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