Case Report

Hypokalemic Paralysis Revealing Subclinical Hyperthyroidism: A Case Report of Thyrotoxic Periodic Paralysis

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ABSTRACT

Introduction: Thyrotoxic periodic paralysis is a rare, potentially life-threatening hyperthyroid complication causing transient muscle weakness due to intracellular potassium shift and hypokalemia.
Case Presentation: A 41-year-old man presented with a sudden onset of symmetrical weakness in all limbs. Laboratory evaluation revealed significant hypokalemia (serum potassium 2.3 mmol/L) and suppressed TSH, with low-normal thyroid hormone levels, consistent with thyrotoxicosis. The patient also exhibited clinical signs of hyperthyroidism, including heat intolerance, tremors, diaphoresis, and sinus tachycardia.
Management: Intravenous potassium replacement led to rapid resolution of symptoms. He was subsequently treated with propranolol and carbimazole. Follow-up thyroid tests at 4 weeks after carbimazole and propranolol showed reduced Total T4 and Free T3 with TSH 0.41 µIU/mL, suggesting early biochemical hypothyroidism; however, the patient remained clinically euthyroid with full recovery of muscle strength and normal serum potassium.
Conclusion: This case highlights the importance of considering TPP in patients with hypokalemic paralysis, even in the absence of classic hyperthyroid features or common triggers. Early diagnosis and appropriate treatment can reverse paralysis and prevent serious complications like cardiac arrhythmias.

KEYWORDS: Thyrotoxic Periodic Paralysis; Hypokalemia; Subclinical Hyperthyroidism; Muscle Weakness; Endocrine Emergency.

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INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a rare, reversible neuromuscular disorder associated with hyperthyroidism, marked by episodic muscle weakness and hypokalemia.[1] Though initially described in Asian populations, particularly among

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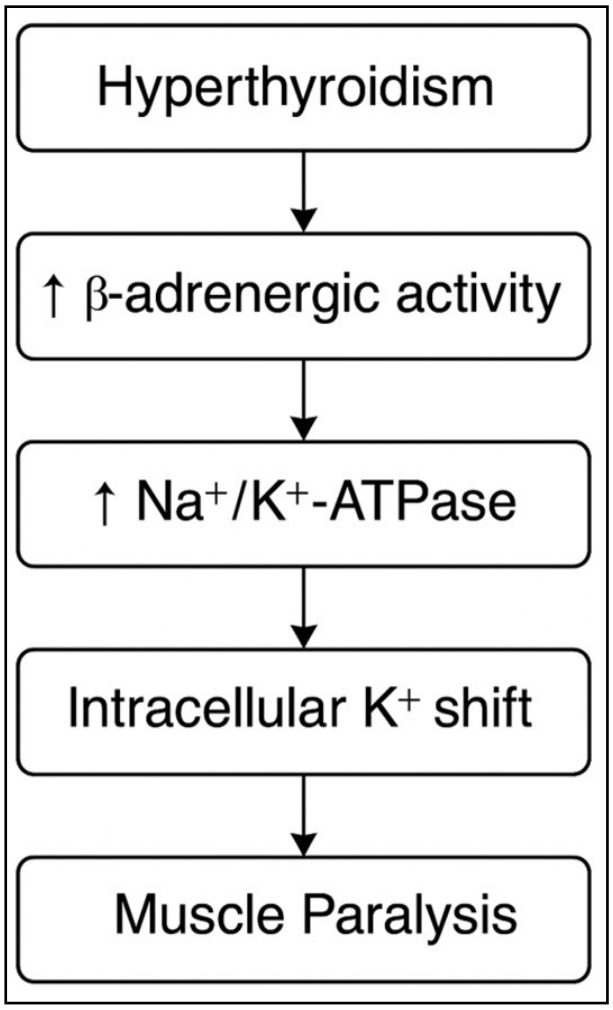

Fig.1: Pathophysiology of Thyrotoxic Periodic Paralysis.

Chinese, Japanese, and Thai males, TPP is increasingly recognized across other ethnic groups, emphasizing its global relevance.[2]

The underlying pathophysiology involves a sudden intracellular shift of potassium, primarily driven by thyroid hormone-induced stimulation of Na^+/K^+-ATPase activity in skeletal muscles. This process is often triggered by factors such as high carbohydrate intake, rest after strenuous exercise, or emotional stress (Fig.1), though episodes may also occur without identifiable provocations.[3]

Clinically, TPP may be the first indicator of thyrotoxicosis, especially in patients with otherwise subtle or subclinical features. The presentation typically involves acute, painless muscle weakness predominantly affecting the lower limbs, and in severe cases, may be accompanied by life-threatening cardiac arrhythmias due to profound hypokalemia.[4]

Despite its dramatic clinical course, TPP remains underrecognized, particularly in non-endemic regions, often resulting in delayed diagnosis and increased morbidity. Early identification and treatment of the underlying thyrotoxic state are essential to prevent recurrence and ensure full recovery.[5] Because hypokalemic paralysis has several possible causes—including familial (hereditary) periodic paralysis, renal tubular acidosis, and gastrointestinal or diuretic-induced potassium losses these alternative etiologies must be systematically excluded before attributing paralysis to thyrotoxicosis.

This case details a patient whose first indication of subclinical hyperthyroidism was an episode of hypokalemic paralysis. The report highlights the diagnostic approach, clinical management, and the importance of considering thyroid dysfunction in

Table-I: Clinical and Laboratory Parameters at Baseline and Post-Treatment.

Parameter	*At Presentation*	*Follow-up (4 Weeks Later)*	*Reference Range*
Serum Potassium (mEq/L)	2.3	3.6	3.5 – 5.5
Serum magnesium (mg/dL)	1.5	–	1.5 – 2.3
Serum creatinine (mg/dL)	0.7	–	0.5 – 1.4
Urine Potassium (mmol/L)	32	–	20 – 80
TSH (μIU/mL)	0.01	0.41	0.4 –4.0
Free T4 (pmol/L)	12.8	–	12 – 22
ECG Findings	Sinus tachycardia	Normal rhythm	–
Muscle Strength	Marked weakness (LL > UL)	Full recovery	Normal

LL: Lower limbs; UL: Upper limbs.

patients with unexplained muscle weakness and electrolyte abnormalities.

CASE PRESENTATION

A 41-year-old male who worked as a fruit hawker presented to the emergency department with sudden onset of symmetrical limb weakness. The weakness progressed rapidly, rendering him unable to walk or raise himself from a seated position. He also experienced difficulty lifting his arms above shoulder level. The episode followed a period of intense physical activity. The patient reported similar, milder episodes over the past three months, which had resolved spontaneously. The patient reported no relevant past medical history, was not taking any medications, and stated that he did not consume alcohol.

He also described associated symptoms such as excessive sweating, heat intolerance, and intermittent palpitations. Clinical evaluation showed that he was conscious and fully oriented, with vital signs indicating a heart rate of 105 beats per minute and a blood pressure reading of 120/80 mmHg. Neurological examination showed diminished muscle strength, graded 4/5 in the upper limbs and 3/5 in the lower limbs, with reduced deep tendon reflexes. Sensory examination was unremarkable.

Table-I shows that initial laboratory investigations revealed severe hypokalemia (serum potassium: 2.3 mEq/L), borderline-low serum magnesium (1.5 mg/dL), and normal renal function (serum creatinine: 0.7 mg/dL). Spot urine potassium was 32 mmol/L, and arterial pH and bicarbonate levels were within normal limits. These findings supported a diagnosis of hypokalemic periodic paralysis due to a transcellular potassium shift rather than renal loss. The patient was taking no medications that could cause renal or gastrointestinal potassium wasting, further supporting a transcellular mechanism over primary renal or gastrointestinal loss.

An electrocardiogram (ECG) showed sinus tachycardia without arrhythmias. The patient was managed with intravenous potassium chloride (40 mEq in normal saline) and oral potassium supplementation, resulting in rapid clinical improvement.

Thyroid function tests subsequently revealed suppressed TSH (0.01 µIU/mL) with free T4 (12.8 pmol/L) within the low-normal range, consistent with subclinical hyperthyroidism. Despite low-normal hormone levels, the patient exhibited mild hyperthyroid symptoms, including palpitations, tremors, and heat intolerance. A Technetium-99m thyroid uptake scan demonstrated diffusely increased tracer uptake with a mildly enlarged thyroid gland, consistent with Graves' disease. The overall uptake was within the normal range (4.2%). The diffuse pattern of uptake, rather than the absolute quantitative value, was considered diagnostic of Graves' disease; normal-range quantitative uptake is well described in early or subclinical thyrotoxicosis and does not exclude this diagnosis. A diagnosis of TPP due to subclinical hyperthyroidism was confirmed. Treatment with propranolol and carbimazole was initiated. Follow-up thyroid tests at 4 weeks after carbimazole and propranolol showed reduced free T4 with TSH rising to 0.41 µIU/mL (within the normal reference range), consistent with a favorable early biochemical response to antithyroid therapy; however, the patient remained clinically euthyroid with full recovery of muscle strength and normal serum potassium.

DISCUSSION

TPP is a rare, reversible complication of hyperthyroidism characterized by acute muscle weakness and hypokalemia. It belongs to a broader group of disorders known as periodic paralyses, which can be hereditary or acquired. TPP is an acquired form, triggered by excess thyroid hormone activity that leads to a transcellular shift of potassium into skeletal muscle cells rather than true potassium loss.[6]

In our case, a previously healthy middle-aged male presented with acute flaccid paralysis predominantly affecting the lower limbs. Although the episode followed physical activity, no other common precipitating factors such as carbohydrate loading or alcohol intake were identified. Initial laboratory workup confirmed significant hypokalemia with normal renal function and urinary potassium levels, supporting a diagnosis of redistributional hypokalemia rather than renal potassium loss. Magnesium was borderline low, which may have exacerbated the neuromuscular symptoms. Other causes of acute hypokalemic paralysis were also considered. Familial hypokalemic periodic paralysis was made unlikely by the absence of a history of recurrent attacks since childhood; renal tubular acidosis and diuretic-induced or gastrointestinal potassium loss were made unlikely by the normal renal function, absence of any diuretic use, and urinary potassium excretion that was appropriately low relative to the degree of hypokalemia. Together with the suppressed TSH, these findings pointed specifically to a thyrotoxic mechanism.

Importantly, this episode of muscle paralysis was the first clinical indication of underlying thyrotoxicosis—a pattern observed in up to 60–80% of TPP cases.[7] Thyroid function tests revealed suppressed TSH with borderline or mildly elevated thyroid hormone levels, indicative of biochemical thyrotoxicosis. Follow-up testing after treatment demonstrated biochemical suppression of thyroid hormone levels with sustained clinical recovery, supporting the effectiveness of therapy while suggesting early treatment-related hypothyroidism.

Electrocardiography revealed sinus tachycardia, a common finding in both thyrotoxicosis and hypokalemia. The normalization of rhythm following potassium replacement and beta-blocker initiation

Table-II: Summary of case reports of TPP with clinical highlights.

S. No.	*Author (Ref)*	*Year*	*Clinical Presentation*	*Outcome*
1.	Nilachandra et al.[13]	2004	Described two female patients diagnosed with TPP an unusual demographic finding	Responded to therapy
2.	Satam N et al.[15]	2007	Pediatric case: A 10-year-old presented with breathlessness and weakness, found to have thyrotoxicosis with normal K^+	Fatal outcome
3.	Balakrishnan RK et al.[9]	2011	Patient experienced recurrent flaccid paralysis secondary to undiagnosed (silent) thyrotoxicosis	Full recovery
4.	Sanyal D et al.[14]	2013	Case linked to thyroiditis, presenting with features of thyrotoxicosis and TPP	Recovered
5.	Chakrabarti S et al.[10]	2015	TPP triggered by intravenous hydrocortisone in an untreated Graves' disease patient	Recovered
6.	Chakrabarti S et al.[11]	2015	Young male with untreated Graves' disease developed TPP despite normal potassium levels during acute phase	Recovered
7.	Swati Hegde et al.[12]	2016	Middle-aged male with subclinical hyperthyroidism and Graves' disease presented with acute limb paralysis and hypokalemia	Improved

(propranolol) demonstrated the reversible nature of these manifestations. Clinically, the patient's presentation with proximal muscle weakness and rapid response to potassium supplementation aligns with typical features of TPP. Full neuromuscular recovery was achieved once thyroid hormone levels were brought under control.

Although TPP is more prevalent in young Asian males, reports from diverse ethnic populations, including cases with subclinical or silent presentations, are increasing.[8] A review of previously reported cases from India (Table-II) also demonstrates the clinical variability of TPP, including presentations in females, normokalaemic states, and pediatric or steroid-triggered scenarios, further emphasizing its diagnostic complexity.[9-15]

In our case, the absence of classic precipitating factors such as high-carbohydrate meals, alcohol intake, or physical exertion reinforces the need for a high index of suspicion, even when triggers are absent.[2,16] This emphasizes the importance of maintaining clinical vigilance, even when traditional precipitating factors are not present.

From a mechanistic perspective, excess thyroid hormone enhances adrenergic tone and upregulates Na^+/K^+-ATPase activity in skeletal muscle, driving potassium into cells. This explains the profound hypokalemia despite normal renal excretion. Non-selective beta-blockers, particularly propranolol, help mitigate these effects by reducing adrenergic stimulation and Na^+/K^+-ATPase activity. When combined with antithyroid therapy, they provide rapid clinical stabilization and prevent recurrence.[17]

Recurrence of paralysis is rare once euthyroidism is achieved. In our case, the patient remained asymptomatic on follow-up, with normal serum potassium and thyroid function, confirming sustained remission.[6]

This case reinforces key clinical principles: TPP can be the initial manifestation of thyrotoxicosis, may occur without evident triggers, and requires timely recognition and treatment to prevent serious complications such as cardiac arrhythmias. Early diagnosis and comprehensive management targeting both hypokalemia and the hyperthyroid state are essential for full recovery and recurrence prevention. This case report has certain limitations including the absence of thyroid scan image, ECG strip, and TRAb results, and lack of a detailed neuromuscular examination. Despite this, diagnosis and management were supported by clinical presentation, labs, and patient response.

CONCLUSION

TPP is a treatable yet potentially serious condition that can lead to sudden muscle weakness and low potassium levels. This case emphasizes the need to include TPP in the differential diagnosis of hypokalemic

paralysis, especially when patients present with mild or subclinical features of thyrotoxicosis. Early recognition and targeted management including potassium repletion, beta-blockade, and antithyroid therapy not only resolve acute symptoms but also prevent serious complications such as cardiac arrhythmias. Achieving and maintaining a euthyroid state is essential to prevent recurrence. Increased awareness of TPP, even in non-endemic populations and in the absence of classic triggers, is vital for timely diagnosis and optimal outcomes. Confirmatory TRAb testing and continued endocrinology follow-up are recommended to guide definitive long-term management of the underlying Graves' disease.

Abbreviations:

TPP: Thyrotoxic periodic paralysis
TSH: Thyroid stimulating hormone

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