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Thyrotoxic Periodic Paralysis Associated with Post-Fasting Refeeding during Ramadan in Graves' disease

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ABSTRACT

Introduction: Thyrotoxic periodic paralysis (TPP) is a rare, potentially life-threatening complication of thyrotoxicosis characterized by acute hypokalaemia and flaccid muscle weakness due to transcellular potassium shifts. We describe a case temporally associated with post-fasting refeeding during Ramadan.

Case Presentation: A 33-year-old Southeast Asian male presented with recurrent acute lower-limb flaccid weakness. Serum potassium level was 1.6 mmol/L during the first episode and 2.3 mmol/L upon re-presentation. Thyroid function tests showed TSH <0.01 mIU/L and free T4 of 41 pmol/L. TSH-receptor antibody level was elevated at 12.5 IU/L, confirming Graves' disease. Symptoms began about two hours after iftar, which consisted of a high-carbohydrate, rice-based meal and dessert. Serum magnesium, creatinine, bicarbonate, and urinary potassium levels were within normal ranges, supporting an intracellular shift rather than total-body potassium depletion.

Methods and Results: Cautious potassium replacement (80 mmol over 24 hours), oral propranolol 40 mg three times daily, and carbimazole 20 mg twice daily led to rapid resolution of weakness and normalization of serum potassium without rebound hyperkalemia.

Conclusion: This case highlights the importance of considering TPP in cases of unexplained hypokalaemic paralysis, particularly in Asian males, and draws attention to post-fasting refeeding during Ramadan as a potential precipitating factor mediated by insulin-driven potassium shifts.

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Introduction

Thyrotoxic periodic paralysis (TPP) is a rare but potentially life-threatening complication of thyrotoxicosis, characterized by acute hypokalaemia and flaccid muscle weakness caused by intracellular potassium shifts rather than total body depletion [1]. It predominantly affects males of Asian descent and is often triggered by high carbohydrate intake, rest after exertion, and adrenergic stimulation [2]. The underlying mechanism involves increased Na^+/K^+-ATPase activity induced by thyroid hormone, insulin, and catecholamines [3]. Although carbohydrate-rich meals are well-recognized triggers, the role of prolonged fasting followed by refeeding, particularly during Ramadan, remains underreported despite its potential impact on electrolyte balance [4].

Case Report

A 33-year-old Southeast Asian male with no known comorbidities presented to a South African regional hospital with acute-onset lower-limb weakness. The weakness was flaccid, predominantly proximal, and progressed over several hours, resulting in significant functional impairment. There were no sensory deficits, cranial nerve involvement, or sphincter disturbances. He reported progressive unintentional weight loss, palpitations, heat intolerance, and anxiety over the preceding months.

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He had experienced a similar episode shortly before this presentation, during which his serum potassium level was 1.6 mmol/L. Intravenous potassium replacement resulted in clinical improvement, and he was subsequently discharged.

Upon re-presentation, serum potassium was 2.3 mmol/L (reference range: 3.5–5.1 mmol/L). Thyroid function tests revealed TSH <0.01 mIU/L (0.27–4.20) and free T4 41 pmol/L (12–22). TSH-receptor antibody (TRAb) was positive at 12.5 IU/L (reference <1.75). Serum magnesium was 0.85 mmol/L (0.70–1.00), creatinine was 78 µmol/L (60–110), and bicarbonate was 24 mmol/L (22–28). Spot urinary potassium was 12 mmol/L, consistent with appropriate renal conservation and an intracellular shift rather than renal potassium wasting.

Further history revealed that the patient was observing Ramadan. Both episodes of weakness began approximately two hours after iftar. The evening meal consisted of a substantial rice-based dish with dessert, providing a large carbohydrate load.

A diagnosis of thyrotoxic periodic paralysis secondary to previously undiagnosed Graves' disease was made. Differential considerations included familial hypokalaemic periodic paralysis; however, the patient's age, Southeast Asian ethnicity, clear clinical and biochemical evidence of thyrotoxicosis, positive TRAb, and absence of a family history made TPP far more likely. Other secondary causes of hypokalaemic paralysis (gastrointestinal or renal losses, drugs, barium poisoning) were excluded based on history and the normal acid–base status, magnesium, creatinine, and low urinary potassium levels.

Acute management included cautious potassium replacement (40 mmol potassium chloride administered twice; serum potassium was 2.8 mmol/L after the first replacement and 4.2 mmol/L after the second) under cardiac monitoring, oral propranolol 40 mg three times daily, and carbimazole 20 mg twice daily. Muscle strength recovered rapidly, and serum potassium normalized within 24 hours without rebound hyperkalemia. The patient was discharged with endocrine follow-up.

A later outpatient thyroid ultrasound showed a diffusely enlarged, heterogeneous thyroid with markedly increased vascularity and no discrete nodules.

Discussion

TPP is characterized by the abrupt onset of hypokalaemia and flaccid paralysis, predominantly affecting the lower extremities, secondary to thyrotoxicosis [1]. Hypokalaemia results from an intracellular shift of potassium induced by thyroid hormone-mediated sensitization of the Na^+/K^+-ATPase rather than total-body potassium depletion [5]. Hyperthyroidism induces a hyperadrenergic state that further enhances pump activity [5].

Classic precipitants include strenuous exercise, high-carbohydrate meals, and other stimuli that cause surges in insulin or catecholamine levels. In the present case, the temporal relationship is particularly notable: both attacks occurred approximately two hours after iftar, which included rice and dessert. Prolonged daytime fasting itself does not cause hypokalaemia and may even result in a mild increase in serum potassium levels [4,6]. We therefore hypothesize that the large post-fast carbohydrate load induced significant hyperinsulinemia; in the context of upregulated Na^+/K^+-ATPase activity secondary to untreated thyrotoxicosis, this insulin surge triggered the intracellular potassium shift and subsequent paralysis. Direct insulin measurements were not obtained; therefore, this mechanism remains a physiologically plausible hypothesis rather than a proven fact.

Management must balance the rapid restoration of muscle strength against the risk of rebound hyperkalemia. Because total-body potassium levels are usually normal, aggressive potassium replacement may result in dangerous extracellular shifts once the attack resolves. Contemporary guidance therefore favors modest potassium supplementation (commonly limited to ≤50–90 mmol within the first 24 hours unless life-threatening arrhythmia or respiratory compromise is present), combined with non-selective β-blockade [7,8]. Propranolol reduces hyperadrenergic stimulation of the Na^+/K^+-ATPase and may modestly inhibit insulin release.

Table 1. Clinical timeline of the patient's presentation, investigations and management.

Time point	Clinical event	Key findings / Intervention
Preceding months	Progressive thyrotoxic symptoms	Unintentional weight loss, palpitations, heat intolerance, anxiety
Index presentation	Acute proximal flaccid lower-limb weakness ~2 h after iftar	K^+ 1.6 mmol/L; Potassium corrected and patient discharged without any further work up
Second presentation (11 days later)	Acute proximal flaccid lower-limb weakness ~2 h after iftar	K^+ 2.3 mmol/L; Potassium corrected. Carbimazole and Propranolol started as further investigations done on this admission were suggestive of Graves disease.

In this patient, 80 mmol potassium supplementation combined with propranolol 40 mg three times daily resulted in prompt recovery without rebound hyperkalemia.

Early recognition of TPP, targeted β-blockade, cautious potassium replacement, and definitive treatment of the underlying thyrotoxicosis are essential to prevent recurrent attacks and iatrogenic complications.

Conclusion

This case illustrates thyrotoxic periodic paralysis as an important and reversible cause of recurrent hypokalaemia and acute flaccid weakness, particularly among Southeast Asian males. It highlights the importance of early recognition and cautious potassium replacement to prevent rebound hyperkalemia. Furthermore, it identifies post-fasting refeeding during Ramadan as a potential precipitating factor, emphasizing the clinical relevance of cultural and dietary practices in TPP presentation.

Declarations

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Nil.

Conflicts of Interest

The authors declare no conflicts of interest.

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Ethical Considerations

This is a single-patient case report. We obtained written informed consent for publication from the patient.

Authors' Contributions

All authors contributed to the conception, clinical management, drafting, and critical revision of the manuscript, and approved the final version for publication.

AI Statement

The authors confirm that no Artificial Intelligence (AI) tools or technologies were used in the preparation of this manuscript.

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