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Atypical Presentation of Thyrotoxic Periodic Paralysis Without Overt Hyperthyroid Symptoms: A Case Report

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Abstract

Thyrotoxic Periodic Paralysis is a rare complication of hyperthyroidism characterised by acute muscle weakness due to hypokalemia. This condition is more commonly reported in Asian populations and may be overlooked when classic symptoms of hyperthyroidism are absent, potentially leading to recurrence and serious complications. A 40-year-old man presented with sudden weakness of all four extremities following consumption of a high-carbohydrate meal. The patient did not exhibit typical symptoms of hyperthyroidism. Laboratory evaluation revealed severe hypokalemia with elevated FT_4 levels and markedly suppressed TSH. The patient was treated with intravenous and oral potassium replacement, along with methimazole and propranolol. Muscle strength improved within 24 hours and recovered completely by the third day of hospitalisation. Thyrotoxic Periodic Paralysis occurs due to an intracellular shift of potassium mediated by increased Na^+/K^+-ATPase activity in the setting of thyrotoxicosis. Attacks are often triggered by high-carbohydrate intake or physical exertion. Diagnosis can be challenging in patients without overt hyperthyroid manifestations; therefore, thyroid function testing is essential in cases of acute hypokalaemic paralysis. Management includes careful potassium correction and control of thyrotoxicosis to prevent recurrence. Thyrotoxic Periodic Paralysis should be considered in any patient presenting with acute weakness accompanied by hypokalemia, even in the absence of classic hyperthyroid symptoms. Prompt treatment and adequate control of thyrotoxicosis are associated with favourable clinical outcomes.

INTRODUCTION

Thyrotoxic Periodic Paralysis (TPP) is a rare but potentially life-threatening complication of thyrotoxicosis, characterised by acute episodes of flaccid muscle weakness associated with hypokalemia (Alrashidi, 2025; Atrash et al., 2024). Although hyperthyroidism is generally more common in women, TPP predominantly affects males, particularly those of Asian descent, with most cases occurring between the second and fourth decades of life. The condition is frequently associated with Graves' disease, but it may also occur in other causes of thyrotoxicosis (Paul et al., 2024).

The pathophysiology of TPP is primarily related to a rapid intracellular shift of potassium rather than a true total body potassium deficit. Excess thyroid hormones increase the activity of Na^+/K^+-ATPase pumps in skeletal muscle, a process further stimulated by hyperinsulinaemia and heightened β-adrenergic activity (Obradovic et al., 2023; Romic et al., 2020). As a result, potassium shifts into the intracellular compartment, leading to hypokalemia and subsequent muscle paralysis (Lin & Huang, 2012). Common precipitating factors include high-carbohydrate meals, strenuous exercise followed by rest, alcohol intake, steroid use, and stress (Alrashidi, 2025; Chakrabarti, 2015).

Clinically, patients typically present with sudden onset of symmetrical proximal muscle weakness, which may progress to involve all four extremities (Alrashidi, 2025). Sensory function is usually preserved, and deep tendon reflexes are often diminished. Despite the underlying thyrotoxicosis, classic symptoms such as weight loss, tremor, heat intolerance, and palpitations may be mild or absent, making the diagnosis challenging (Karunarathna, n.d.; Ross et al., 2016). This atypical presentation can lead to misdiagnosis or delayed recognition, especially when TPP is not initially suspected in patients presenting with acute hypokalemic paralysis (Salih et al., 2017; Gadi & Schueler, 2024).

Early identification of TPP is crucial, as inappropriate management may increase the risk of rebound hyperkalemia or recurrent paralytic episodes (Matsui et al., 2025; Pisano et al., 2025). Therefore, evaluation of thyroid function should be considered in all patients presenting with unexplained hypokalemic paralysis. This case report aims to highlight the atypical presentation of thyrotoxic periodic paralysis in a patient without overt hyperthyroid symptoms, emphasising the diagnostic challenges and the importance of prompt management to achieve favourable clinical outcomes (Meseeha et al., 2017; Tadisina et al., 2023; Van den Broeck et al., 2024). The findings of this report provide practical benefits for clinicians, particularly in emergency and primary care settings, by increasing awareness that TPP should be considered in any patient presenting with acute hypokalemic paralysis—even in the absence of classic hyperthyroid manifestations—thereby preventing misdiagnosis, reducing the risk of recurrence, and guiding appropriate thyroid function testing and treatment.

METHOD

This study employed a clinical case report methodology, detailing the presentation, diagnostic evaluation, management, and clinical outcome of a single patient. Data collection included a comprehensive review of the patient's medical history, physical and neurological examinations, laboratory investigations, and diagnostic imaging where relevant. Therapeutic interventions, including intravenous and oral medications, were recorded along with the patient's response to treatment. Continuous monitoring of vital signs, serum electrolytes, and clinical progression was conducted throughout hospitalization. Follow-up assessments after discharge were planned to evaluate ongoing treatment efficacy and patient recovery. The case report methodology allows for an in-depth exploration of rare or clinically significant presentations, contributing to medical knowledge and guiding future clinical practice.

A 40-year-old Asian male with no prior history of chronic illness presented to the emergency department with sudden onset weakness of all four extremities, beginning approximately three hours prior to admission. The weakness initially involved the lower limbs and subsequently progressed to the upper limbs, accompanied by muscle fasciculations. The patient remained fully conscious and denied any sensory disturbances, dysphagia, or dyspnea. The episode occurred several hours after consuming a large carbohydrate-rich meal.

The patient reported two similar episodes within the past six months, previously diagnosed as hypokalemic paraparesis of unclear etiology. There was no history of diuretic, laxative, steroid, herbal medication use, or alcohol consumption. He did not report symptoms suggestive of thyrotoxicosis, such as weight loss, tremor, palpitations, or excessive sweating. There were also no gastrointestinal complaints, including nausea, vomiting, or diarrhea, and no history of polyuria. Family history was unremarkable for similar conditions.

On physical examination, the patient was alert (compos mentis) with stable vital signs: blood pressure 120/80 mmHg, heart rate 92 beats per minute (regular), temperature 36.8°C, and respiratory rate 18 breaths per minute. There was no evidence of goiter, fine tremor, or thyroid-associated ophthalmopathy. Neurological examination revealed decreased muscle strength with a Medical Research Council (MRC) grade of 2/5 in the lower extremities and 4/5 in the upper extremities. Muscle tone and deep tendon reflexes were reduced, while sensory examination was normal.

Laboratory investigations demonstrated significant hypokalemia with a serum potassium level of 2.3 mmol/L (reference range 3.5–5.0 mmol/L), elevated free thyroxine (FT4) at 3.44 ng/dL (reference range 0.8–1.8 ng/dL), and suppressed thyroid-stimulating hormone (TSH) <0.01 µIU/mL (reference range 0.4–4.0 µIU/mL). Serum magnesium and phosphate levels were not assessed. Random blood glucose at admission was 331 mg/dL, which decreased to 173 mg/dL after several hours of observation without antidiabetic therapy. Electrocardiography revealed normal sinus rhythm with prominent U waves, consistent with hypokalemia.

The patient was treated with intravenous potassium chloride (50 mEq diluted in 500 mL Ringer's lactate, administered at 20 drops per minute), repeated twice, followed by reassessment showing normalization of serum potassium to 3.7 mmol/L. Oral potassium supplementation was subsequently initiated. Antithyroid therapy was started with methimazole 10 mg three times daily and propranolol 20 mg twice daily.

Clinical improvement was observed within 24 hours, with progressive recovery of muscle strength. Full motor function was restored by the third day of hospitalization. The patient was discharged on oral methimazole and propranolol, with a scheduled outpatient follow-up for continued monitoring and management.

RESULT AND DISCUSSION

Thyrotoxic Periodic Paralysis is an uncommon but clinically significant endocrine emergency characterized by acute hypokalemic paralysis in the setting of thyrotoxicosis. This case highlights several classic yet often underrecognized features of TPP, including occurrence in an Asian male, recurrent episodes of hypokalemic weakness, and a clear precipitating factor in the form of a high-carbohydrate meal. Notably, the absence of overt symptoms of hyperthyroidism contributed to delayed recognition in previous episodes, underscoring the diagnostic challenge of atypical presentations (Alrashidi, 2025; Atrash et al., 2024).

The hallmark of TPP is a rapid intracellular shift of potassium rather than a true potassium deficit. Thyroid hormone excess enhances Na^+/K^+-ATPase activity in skeletal muscle, which is further potentiated by insulin and catecholamines (Lin & Huang, 2012). Following a carbohydrate-rich meal, insulin secretion increases, driving potassium into cells and precipitating hypokalemia (Alrashidi, 2025; Chakrabarti, 2015). This mechanism explains the temporal association between dietary triggers and the onset of paralysis observed in this patient. Additionally, β-adrenergic stimulation plays a synergistic role, which provides the rationale for the use of non-selective β-blockers such as propranolol in management (Wassner & Cheng, 2021).

Clinically, TPP typically presents with sudden-onset, symmetrical, flaccid muscle weakness predominantly affecting proximal muscles, often beginning in the lower extremities and progressing upward. Sensory function is preserved, and cranial nerve involvement is rare,

consistent with the findings in this case. Reflexes are commonly diminished or absent. Importantly, despite biochemical thyrotoxicosis, many patients, particularly males, may lack classical hyperthyroid symptoms, leading to underdiagnosis or misdiagnosis as primary hypokalemic periodic paralysis or other neuromuscular disorders (Alrashidi, 2025; Paul et al., 2024). Al Moteri and Aslam (2017) reported delayed diagnosis in a patient presenting with recurrent paralytic episodes before thyrotoxicosis was recognized. Therefore, thyroid function testing should be considered routinely in patients presenting with acute hypokalemic paralysis, particularly in Asian male patients.

Electrocardiographic findings in TPP reflect the degree of hypokalemia and may include U waves, ST-segment depression, and arrhythmias (Ramadurai et al., 2025; Wang et al., 2020). In this patient, the presence of prominent U waves was consistent with significant hypokalemia. Although the patient did not develop cardiac complications, severe hypokalemia in TPP carries a risk of life-threatening arrhythmias, emphasizing the need for prompt recognition and treatment.

Management of TPP involves careful potassium replacement and definitive control of thyrotoxicosis. Unlike other causes of hypokalemia, aggressive potassium repletion should be avoided due to the risk of rebound hyperkalemia once the intracellular shift reverses. Pisano et al. (2025) described a patient who developed severe rebound hyperkalemia requiring aggressive potassium-lowering therapy and vasopressor support after potassium replacement therapy. Similarly, Matsui et al. (2025) reported a rise in serum potassium from 1.7 mEq/L to 5.6 mEq/L within six hours after administration of 122 mEq potassium supplementation. These findings emphasize that hypokalemia in TPP primarily results from intracellular potassium redistribution rather than total body potassium depletion; therefore, excessive replacement may rapidly lead to hyperkalemia once the intracellular shift reverses (Matsui et al., 2025).

In this case, cautious intravenous and oral potassium supplementation led to rapid clinical improvement without complications. The use of antithyroid medication (methimazole) and β-blocker (propranolol) is essential not only for treating the underlying thyrotoxicosis but also for preventing recurrence. Propranolol, in particular, reduces β-adrenergic stimulation and decreases Na^+/K^+-ATPase activity, thereby mitigating further intracellular potassium shifts. Wassner and Cheng (2021) reported successful treatment of TPP using low-dose oral propranolol combined with potassium supplementation, without development of rebound hyperkalemia.

An additional noteworthy finding in this case was transient hyperglycemia at presentation, which resolved spontaneously without antidiabetic therapy. This phenomenon may be explained by stress-induced hyperglycemia and the metabolic effects of excess thyroid hormone, including increased gluconeogenesis and insulin resistance (Vedantam et al., 2022). However, the role of hyperglycemia as a potential trigger via hyperinsulinemia may also contribute to the pathogenesis of TPP episodes. A recent case reported by Wang and Li (2025) demonstrated severe TPP precipitated by insulin therapy in a patient with uncontrolled diabetes mellitus.

CONCLUSION

Thyrotoxic Periodic Paralysis should be considered in any patient presenting with acute flaccid paralysis and hypokalemia, even in the absence of overt hyperthyroid symptoms. This

case highlights that atypical presentations may delay diagnosis, particularly when previous episodes are attributed to unexplained hypokalemia. Early recognition and appropriate management, including cautious potassium replacement and control of thyrotoxicosis, are essential to ensure rapid recovery and prevent recurrence, while routine evaluation of thyroid function is strongly recommended in all cases of unexplained hypokalemic paralysis, especially in high-risk populations such as Asian males.

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