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# Thyrotoxic Hypokalemic Periodic Paralysis Presenting As Acute Tetraparesis: A Case Report

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## Abstract

Thyrotoxic hypokalemic periodic paralysis (TPP) is a rare but potentially life-threatening complication of hyperthyroidism, usually associated with Graves' disease. We report the case of a 29-year-old man with acute flaccid tetraparesis associated with severe hypokalemia. Prompt potassium replacement resulted in rapid clinical and biochemical improvement. The diagnosis of TPP was established based on the clinical presentation, the presence of thyrotoxicosis, severe hypokalemia, and rapid clinical improvement following potassium replacement. Awareness of this condition is essential for early diagnosis and appropriate management, as delayed recognition may result in potentially life-threatening complications, including cardiac arrhythmias and respiratory failure.

**Categories:** Emergency Medicine, Internal Medicine, Endocrinology/Diabetes/Metabolism
**Keywords:** endocrine emergency, graves disease, hypokalemia, thyrotoxicosis, thyrotoxic periodic paralysis

## Introduction

Periodic paralysis (PP) is a rare disorder characterized by recurrent, transient episodes of muscle weakness or paralysis associated with abnormalities in skeletal muscle ion channel function. Episodes are typically related to intracellular potassium shifts and may be precipitated by factors such as high-carbohydrate meals, rest after exercise, or stress.

PP can be classified into hereditary and acquired forms. Among hereditary forms, hypokalemic periodic paralysis (HypoPP) is the most common and is typically inherited in an autosomal dominant pattern. It is primarily caused by pathogenic variants in CACNA1S or SCN4A, which encode voltage-gated calcium and sodium channels expressed in skeletal muscle, respectively. HypoPP usually presents during late childhood or adolescence, with a mean age of onset of approximately 14 years, although the diagnosis may be established later in life [1,2].

Acquired forms include thyrotoxic hypokalemic periodic paralysis (TPP), a complication of thyrotoxicosis characterized by acute episodes of hypokalemic muscle weakness. TPP predominantly affects young adult men and is particularly prevalent in East and Southeast Asian populations. Among patients with hyperthyroidism, its prevalence has been estimated at approximately one in 50 in Asian populations, compared with approximately one in 500 to one in 1,000 in other populations [3,4]. Graves' disease is the most common underlying cause of TPP. Although thyrotoxicosis is more prevalent among women, TPP shows a striking male predominance [3,4].

TPP is characterized by the triad of acute muscle weakness, hypokalemia, and thyrotoxicosis. The hypokalemia results primarily from a rapid intracellular shift of potassium rather than true depletion of total-body potassium. Excess thyroid hormone increases $Na^+/K^+$-ATPase activity and enhances β-adrenergic sensitivity, while insulin release following carbohydrate-rich meals may further promote intracellular potassium uptake. Genetic susceptibility also contributes to TPP, with variants or polymorphisms involving genes such as KCNJ18, CACNA1S, and SCN4A having been implicated. These genetic factors differ from the established monogenic causes of hereditary HypoPP and are thought to modify susceptibility rather than independently cause TPP [1,5].

Clinically, TPP presents with acute, transient, and typically reversible muscle weakness, usually symmetrical and predominantly affecting proximal muscles. The lower limbs are generally more severely affected than the upper limbs, while respiratory and ocular muscles are usually spared. Severe attacks may, however, result in respiratory failure or life-threatening cardiac arrhythmias. Classical manifestations of thyrotoxicosis may be subtle or absent, which can delay recognition of the underlying endocrine disorder [3,5].

The differential diagnosis includes hereditary HypoPP and secondary causes of hypokalemia, particularly renal and gastrointestinal potassium losses. Renal tubulopathies such as renal tubular acidosis, Bartter syndrome, and Gitelman syndrome should also be considered, particularly when hypokalemia is persistent rather than associated with acute episodic weakness [6].

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Laboratory findings typically reveal marked hypokalemia (often <3.0 mEq/L), frequently associated with hypophosphatemia and hypomagnesemia, as well as low urinary potassium excretion in the absence of metabolic acidosis. These findings support an intracellular potassium shift rather than renal potassium wasting [1,2].

Confirmation of thyrotoxicosis is essential, and testing for thyroid-stimulating hormone (TSH) receptor antibodies should be considered, particularly in patients without a prior history of thyroid disease. Early recognition of TPP is critical because appropriate treatment can lead to rapid clinical improvement and prevent potentially life-threatening complications.

We report the case of a young patient with a recent diagnosis of hyperthyroidism who developed acute hypokalemic flaccid paralysis, highlighting the importance of early recognition and prompt treatment of TPP.

## Case Presentation

A 29-year-old Caucasian man presented with a one-year history of nocturnal diaphoresis, unintentional weight loss, and recurrent transient episodes of lower-limb weakness. Three months before admission, he was diagnosed with hyperthyroidism, with laboratory tests showing suppressed TSH (0.01 UI/mL), elevated free thyroxine (FT4) (2.37 ng/dL), elevated free triiodothyronine (FT3) (20 pg/mL), and positive thyroid antibodies, including anti-thyroglobulin antibodies (ATg) (414 UI/mL), anti-thyroid peroxidase antibodies (ATPO) (821 UI/mL), and TSH receptor antibodies (TRAb) (6.57 UI/mL). He was started on propranolol and methimazole two days before presentation.

On the night before admission, the patient developed paresthesias in the lower limbs. Unlike his previous episodes, the symptoms did not resolve spontaneously and progressively worsened, prompting hospital evaluation. No triggering factor was identified, including a high-carbohydrate meal, recent strenuous exercise, alcohol intake, or drug use.

On physical examination, mild proptosis was noted. Neurological evaluation revealed tetraparesis, with muscle strength graded as 4/5 in the upper limbs and complete paralysis of the lower limbs according to the Medical Research Council (MRC) scale. Cranial nerve examination was normal, and deep tendon reflexes were symmetrically diminished.

Laboratory analysis demonstrated severe hypokalemia (serum potassium, 2.2 mEq/L), contrasting with a previously normal value of 4.2 mEq/L measured two days earlier. Thyroid function tests showed persistently suppressed TSH (<0.008 UI/mL), FT4 of 0.80 ng/dL, and FT3 of 6.37 pg/mL. Serum phosphate and magnesium levels were within normal limits (Table *1*). Electrocardiography and brain computed tomography showed no abnormalities, and spinal computed tomography revealed no relevant findings.

| Laboratory parameter | Result | Reference range |
|---|---|---|
| Serum potassium | 2.2 mEq/L | 3.5-5.0 mEq/L |
| Serum phosphate | 2.9 mg/dL | 2.5-4.5 mg/dL |
| Serum magnesium | 2.04 mg/dL | 1.7-2.2 mg/dL |
| Thyroid-stimulating hormone (TSH) | <0.008 UI/mL | 0.4-4.0 UI/mL |
| Free thyroxine (FT4) | 0.8 ng/dL | 0.8-1.8 ng/dL |
| Free triiodothyronine (FT3) | 6.37 pg/mL | 1.71-3.71 pg/mL |
| Anti-thyroglobulin antibodies (ATg) | 414 UI/mL | <4.1 UI/mL |
| Anti-thyroid peroxidase antibodies (ATPO) | 821 UI/mL | <5.6 UI/mL |
| TSH receptor antibodies (TRAb) | 6.57 UI/mL | <1.5 UI/mL |

**TABLE 1: Laboratory results of the current case.**

The patient received 60 mEq of intravenous potassium chloride diluted in 1500 mL of normal saline over five hours, together with 20 mEq of oral potassium chloride administered with meals. Approximately one hour after treatment initiation, neurological deficits began to improve, with complete resolution of weakness within a few hours. Serum potassium increased from 2.2 mEq/L to 4.65 mEq/L after five hours of treatment. Continuous electrocardiographic monitoring was maintained during potassium replacement, and no

rebound hyperkalemia was observed. The patient remained clinically stable, asymptomatic, and was discharged approximately 12 hours later.

The clinical and biochemical evolution from the diagnosis of hyperthyroidism to the acute paralytic episode and subsequent recovery is summarized in Figure *1*.

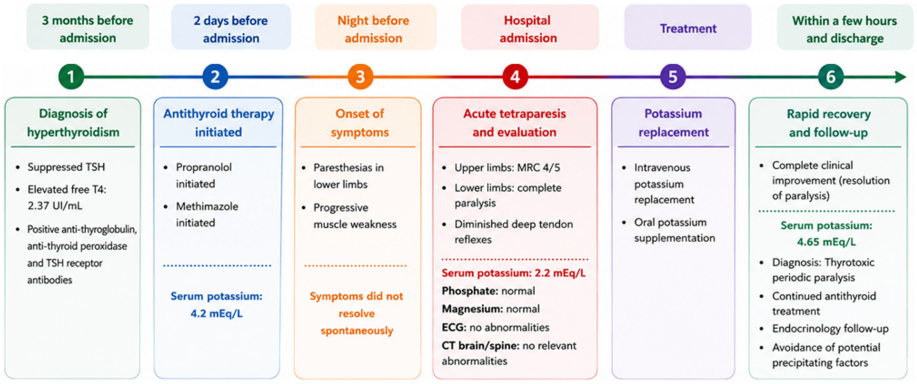



**FIGURE 1: Clinical and biochemical timeline of the patient's presentation and management.**

CT, computed tomography; ECG, electrocardiogram; TSH, thyroid-stimulating hormone

The patient was discharged with outpatient endocrinology follow-up, continuing antithyroid therapy, and was advised to avoid known precipitating factors, including high-carbohydrate meals, strenuous exercise, and alcohol intake until a euthyroid state was achieved.

## Discussion

TPP is a rare but potentially life-threatening neuromuscular emergency. This case highlights the importance of considering TPP in any patient presenting with acute flaccid paralysis and hypokalemia, even in the absence of classical clinical features of thyrotoxicosis [1,3,7,8,9]. TPP remains underdiagnosed, particularly in non-Asian populations and in patients without overt signs of thyrotoxicosis, which may contribute to diagnostic delays [9,10].

The underlying pathophysiology (Figure *2*) is primarily driven by increased $Na^+/K^+$-ATPase activity in skeletal muscle, together with alterations in skeletal muscle membrane excitability. This process reflects the combined effects of genetic susceptibility, excess thyroid hormones, and environmental triggers, ultimately leading to a rapid intracellular shift of potassium and marked hypokalemia (Table *2*) [1,5].

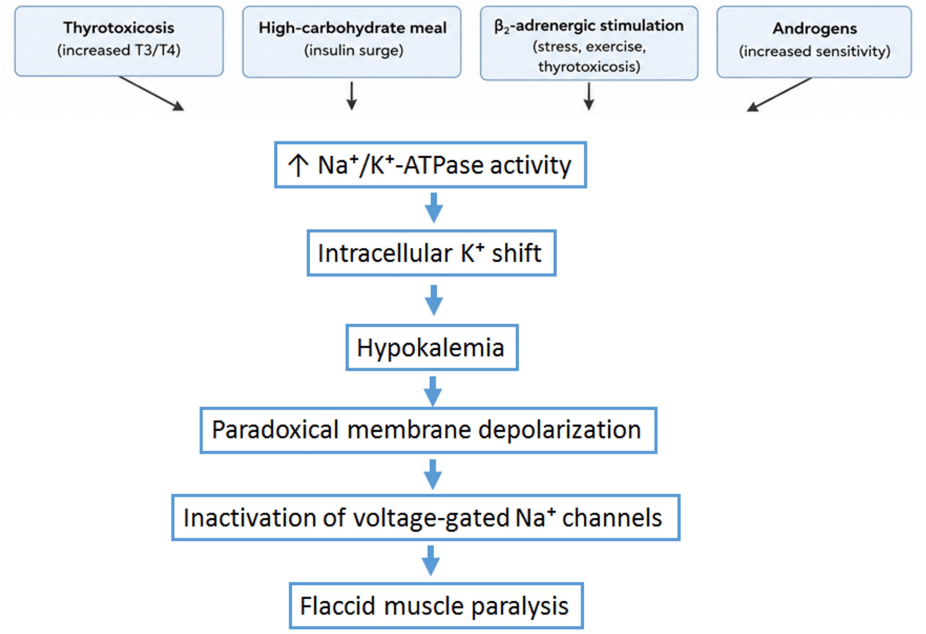



**FIGURE 2: Simplified pathophysiological mechanism of thyrotoxic periodic paralysis.**

Created by the authors based on previously described mechanisms [1].

| Laboratory parameter | Reference range | Typical findings in TPP | Clinical significance |
|---|---|---|---|
| Serum potassium | 3.5-5.0 mEq/L | Decreased, usually <3.0 mEq/L and often <2.5 mEq/L | Hypokalemia due to intracellular potassium shift is the main contributor to muscle weakness and paralysis |
| Serum phosphate | 2.5-4.5 mg/dL | Often decreased | Reflects intracellular shift and may support the diagnosis |
| Serum magnesium | 1.7-2.2 mg/dL | Normal or mildly decreased | May contribute to muscle weakness when reduced |
| Thyroid-stimulating hormone (TSH) | 0.4-4.0 mIU/L | Suppressed | Supports the presence of thyrotoxicosis |
| Free thyroxine (FT4) | 0.8-1.8 ng/dL | Elevated | Indicates a hyperthyroid state |
| Free triiodothyronine (FT3) | 2.3-4.2 pg/mL | Elevated | Indicates a hyperthyroid state |
| Urinary potassium excretion | <20 mEq/L in hypokalemia | Usually low (<20 mEq/L) | Supports intracellular potassium shift and argues against renal potassium wasting |
| Acid-base status | Arterial pH: 7.35-7.45 | Typically normal | Helps differentiate TPP from renal tubular acidosis and other causes of hypokalemia |

**TABLE 2: Laboratory findings typically observed in thyrotoxic periodic paralysis[1,5].**

Genetic variants in ion channel-related genes, including KCNJ18, CACNA1S, SCN4A, and KCNE3, have been associated with increased susceptibility; however, most cases occur sporadically [3,5].

The resulting reduction in extracellular potassium alters skeletal muscle membrane excitability and impairs muscle contraction, producing the characteristic episodes of reversible weakness and paralysis observed in TPP. If not promptly recognized, TPP may lead to severe complications, including cardiac arrhythmias and respiratory failure [1,2,5,11].

The differential diagnosis of TPP should include other causes of hypokalemic paralysis, such as familial

HypoPP, renal tubular disorders, and other secondary causes of hypokalemia (Table 3; Figure 3) [2].

| Characteristic | TPP | Familial HypoPP | Renal tubular acidosis | Bartter/Gitelman syndrome |
|---|---|---|---|---|
| Age of onset | Young adulthood (20-40 years) | Childhood/adolescence | Variable | Adolescence/early adulthood |
| Sex predominance | Male (>95%) | Both | Both | Both |
| Triggering factors | Carbohydrates, exercise, stress, thyrotoxicosis | Carbohydrates, rest after exercise | No clear pattern | No clear pattern |
| Serum potassium | Markedly decreased (<2.5 mEq/L) | Decreased | Decreased | Decreased |
| Urinary potassium | Low (<20 mEq/L) | Low | High | High |
| Urinary pH | Normal or alkaline | Normal | >5.5 (alkaline) | >5.5 (alkaline) |
| Serum phosphate | Decreased | Normal or decreased | Normal | Decreased (Gitelman) |
| Serum magnesium | Normal or mildly decreased | Normal | Normal | Decreased (Gitelman) |
| Acid-base status | Normal | Normal | Metabolic acidosis | Metabolic alkalosis |
| Acute treatment | Potassium replacement ± non-selective β-blocker (e.g., propranolol); treatment of thyrotoxicosis | Potassium replacement | Alkali therapy + potassium | Potassium ± magnesium replacement; potassium-sparing agents may be considered |
| Definitive treatment | Achieve and maintain euthyroidism (e.g., antithyroid therapy, radioactive iodine, or surgery depending on etiology) | Trigger avoidance and preventive therapy; no curative treatment | Treat underlying cause | Supportive therapy and treatment of the underlying cause |

**TABLE 3: Comparison between thyrotoxic periodic paralysis and other causes of hypokalemic periodic paralysis [2-4].**

HypoPP, hypokalemic periodic paralysis; TPP, thyrotoxic hypokalemic periodic paralysis

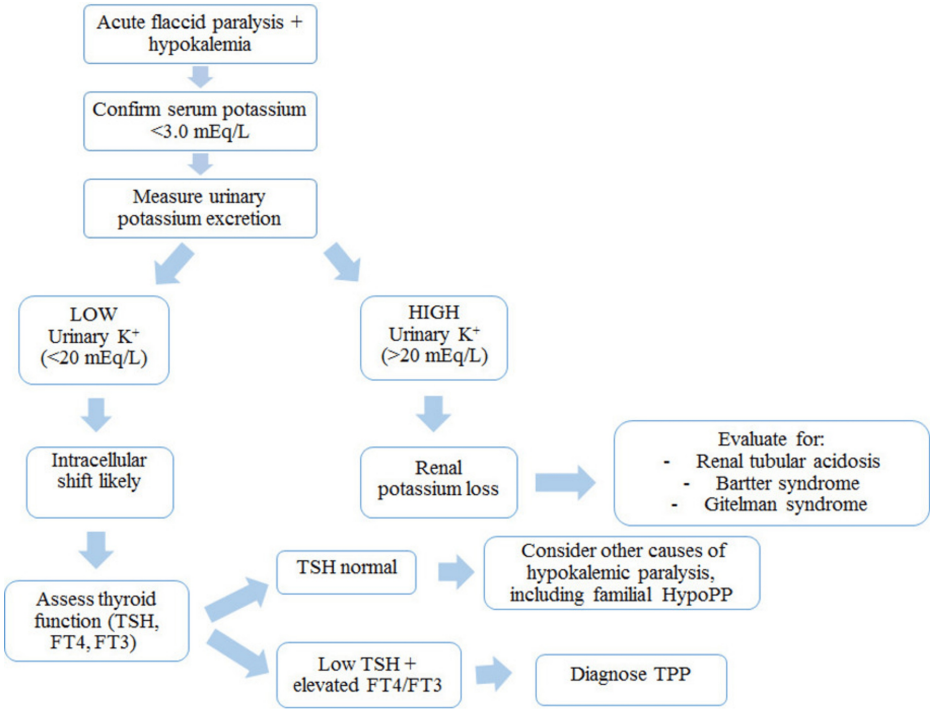



**FIGURE 3: Diagnostic approach to hypokalemic paralysis, highlighting the differentiation between thyrotoxic periodic paralysis and other causes based on urinary potassium excretion and thyroid function.**

Created by the authors based on previously described diagnostic principles [2,3,6].

FT3, free triiodothyronine; FT4, free thyroxine; HypoPP, hypokalemic periodic paralysis; TPP, thyrotoxic hypokalemic periodic paralysis; TSH, thyroid-stimulating hormone

Acute management consists of potassium replacement, preferably with oral potassium chloride in patients without cardiopulmonary compromise, together with close monitoring of serum potassium levels and cardiac rhythm. Unlike many other causes of severe hypokalemia, TPP results primarily from a rapid intracellular shift of potassium rather than true total-body potassium depletion. Consequently, excessive potassium replacement may lead to rebound hyperkalemia once potassium redistributes to the extracellular compartment. For this reason, potassium should be administered cautiously, with the total dose individualized according to the severity of hypokalemia, clinical manifestations, and serial potassium measurements. Although oral potassium supplementation is generally preferred, intravenous potassium may be required in patients with severe symptomatic hypokalemia, profound muscle weakness, cardiac arrhythmias, or inability to tolerate oral therapy. In the present case, the patient presented with acute tetraparesis and severe hypokalemia (2.2 mEq/L), warranting combined intravenous and oral potassium replacement under continuous electrocardiographic monitoring. This approach resulted in rapid neurological recovery without rebound hyperkalemia [5,8,11].

Patients should be advised to avoid known precipitating factors, including high-carbohydrate meals, strenuous exercise, and alcohol intake, until a euthyroid state is achieved. Routine prophylactic potassium supplementation is not recommended. Definitive treatment requires normalization of thyroid function through antithyroid drugs, radioactive iodine therapy, or thyroidectomy, depending on the underlying etiology. Achieving and maintaining euthyroidism effectively prevents recurrence of paralytic episodes [11].

## Conclusions

TPP should be considered in any patient presenting with acute flaccid paralysis and hypokalemia, regardless of ethnic background or the absence of typical symptoms of hyperthyroidism. Early diagnosis and appropriate management are essential to prevent severe complications, including cardiac arrhythmias and respiratory failure. Clinicians should maintain a high index of suspicion and pursue definitive control of hyperthyroidism to prevent recurrence and ensure complete neurological recovery.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Tânia Pereira da Silva, Joana Rego, Flávia Santos, Carlos Fernandes, Glória Alves

**Acquisition, analysis, or interpretation of data:** Tânia Pereira da Silva, Joana Rego, Flávia Santos, Carlos Fernandes, Glória Alves

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**Critical review of the manuscript for important intellectual content:** Tânia Pereira da Silva, Joana Rego, Flávia Santos, Carlos Fernandes, Glória Alves

**Supervision:** Tânia Pereira da Silva, Joana Rego, Flávia Santos, Carlos Fernandes, Glória Alves

## Disclosures

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# References

1. Lin SH, Huang CL: Mechanism of thyrotoxic periodic paralysis . J Am Soc Nephrol. 2012, 23:985-8. 10.1681/ASN.2012010046
2. Gumz ML, Rabinowitz L, Wingo CS: An integrated view of potassium homeostasis . N Engl J Med. 2015, 373:60-72. 10.1056/NEJMra1313341
3. Ahlawat SK, Sachdev A: Hypokalaemic paralysis. Postgrad Med J. 1999, 75:193-7. 10.1136/pgmj.75.882.193
4. Manoukian MA, Foote JA, Crapo LM: Clinical and metabolic features of thyrotoxic periodic paralysis . Arch Intern Med. 1999, 159:601-6. 10.1001/archinte.159.6.601
5. Patel M, Ladak K: Thyrotoxic periodic paralysis: a case report and literature review . Clin Med Res. 2021, 19:148-51. 10.3121/cmr.2021.1610
6. Kung AWC: Clinical review: Thyrotoxic periodic paralysis: a diagnostic challenge . J Clin Endocrinol Metab. 2006, 91:2490-5. 10.1210/jc.2006-0356
7. Okinaka S, Shizume K, Iino S, et al.: The association of periodic paralysis and hyperthyroidism in Japan . J Clin Endocrinol Metab. 1957, 17:1454-9.
8. Tadisina S, Asad R, Varakantam A, Weide L, Drees B: Thyrotoxic periodic paralysis as an ongoing diagnostic challenge: a case report and literature review. Cureus. 2023, 15:e46272. 10.7759/cureus.46272
9. Paul J, Joseph A, Jebasingh F, et al.: Thyrotoxic periodic paralysis - a retrospective study from Southern India. Eur Thyroid J. 2024, 13: 10.1530/ETJ-24-0164
10. Matsui D, Mugikura S, Goshima T, Ishizuka N, Jingushi N, Uenishi N, Iwata M: Rebound hyperkalemia after potassium repletion in thyrotoxic periodic paralysis: a case report and review of management implications. Cureus. 2025, 17:e85551. 10.7759/cureus.85551
11. Yoon SH, Raza H: Thyrotoxic periodic paralysis: the peril of potassium replacement . Cureus. 2022, 14:e32926. 10.7759/cureus.32926