“A Paralyzing Snack”: An Endocrine Cause of Paralysis

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ABSTRACT

A case of a young Caucasian man presenting to the Emergency Department (ED) with lower limb weakness, anxiety, and sweating is described. Clinical and laboratory evaluations revealed severe hypokalemia (1.4 mmol/L) associated with thyrotoxicosis, leading to a diagnosis of Thyrotoxic Periodic Paralysis (TPP). After initial improvement following potassium infusion, the patient experienced symptom exacerbation. Further investigation linked the recurrence to excessive carbohydrate intake from vending machine snacks. TPP, a complication of hyperthyroidism, is extremely rare in Western countries but must be promptly recognized due to its potential life-threatening complications.

KEYWORDS

thyrotoxic periodic paralysis; thyrotoxicosis; hypokalemia; paralysis; hyperthyroidism

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INTRODUCTION

Thyrotoxic Periodic Paralysis (TPP) is a rare neurological disorder characterized by muscle paralysis, hypokalemia, and hyperthyroidism. It primarily affects Asian males, but is far less common in non-Asian populations. This makes the disease little known in Western countries, where it is uncommon, thus making it potentially dangerous if not diagnosed early. A peculiarity of this clinical case is that TPP represents the initial presentation of hyperthyroidism.

TIMELINE

2023-06-05
Onset of symptoms.
2023-06-07
Hospital admission.
2023-06-07
Neurological evaluation, cranial CT scan, ECG, EMG, laboratory tests.
2023-06-07
Initial diagnosis of hypokalemic paralysis and initiation of potassium infusion therapy.
2023-06-08
Diagnosis of thyrotoxic periodic paralysis. Therapy with methimazole.
2023-06-10
Recurrence of neurological symptoms, with marked asthenia and low potassium levels, after consumption of a carbohydrate-rich snack.
2023-06-14
Discharge in good health.
2023-07-10
After about a month from discharge, the patient is in good health and continues therapy with methimazole.

NARRATIVE

A 31-year-old Caucasian man presented to the ED with a sudden onset of lower limb weakness upon awakening. His medical history included arterial hypertension managed with bisoprolol 1.25 mg daily. He denied any trauma, alcohol, tobacco, or drug use, or other medications. On physical examination, the patient appeared anxious with mildly warm, sweating skin. Vital signs showed a heart rate of 106 bpm, blood pressure of 120/70 mmHg, and tympanic temperature of 37.2 °C. Neurological evaluation revealed flaccid weakness and absent tendon reflexes in the lower limbs, while the rest of the neurological exam was unremarkable. Electrocardiogram (ECG) showed sinus tachycardia, incomplete right bundle branch block, and nonspecific ventricular repolarization abnormalities. The patient was admitted for further evaluation, including laboratory tests and a cranial CT scan, which was unremarkable. An Electromyography was scheduled. Laboratory findings revealed significant hypokalemia (1.4 mmol/L) and hypophosphatemia (0.5 mg/dL), aldosterone 19.0 ng/dL, renin (ortho) 21.5 IU/mL, blood gas analysis: pH 7.47, PaO2 91 mmHg, PaCO2 47 mmHg, HCO3 28.2 mEq/L, K+ 1.2 mmol/L. Intravenous potassium chloride was administered at 20 mEq/hour. A thorough review of personal and family history revealed no prior neurological conditions. Weakness improved on the first day as serum potassium levels gradually increased. A careful family and personal history helped exclude, in the differential diagnosis, Familial Hypokalemic Periodic Paralysis (HypoPP), secondary hypokalemia-related paralysis (absence of vomiting and diarrhea, excluding diuretic abuse, and normal aldosterone values), and Guillain-Barré syndrome due to the normal EMG picture and the lack of the characteristic evolutionary progression. However, anxiety, warm sweating, and tachycardia persisted, prompting evaluation of thyroid function. Thyroid function tests showed FT3 7.77 pg/mL, FT4 2.45 ng/dL, and TSH 0.004 µIU/mL, TRAb 18.4 IU/L, AbTG 380 IU/mL; TPOAb 450 IU/mL (Tab. 1). Thyroid ultrasound revealed diffuse thyroid enlargement with hypoechogenicity and no nodules. Antithyroid therapy with methimazole 30 mg/day and propranolol 120 mg/day was initiated. By the third day, the patient experienced an unexpected recurrence of neurological symptoms, with marked asthenia and a drop in serum potassium levels to 2.8 mmol/L. Investigation revealed excessive consumption of carbohydrate-rich snacks and chocolate from a vending machine the previous evening. Potassium chloride infusion resolved the symptoms. Subsequent oral glucose tolerance testing and insulin levels were normal (insulin 14µU/mL). The patient was discharged on the seventh day in good health and remains asymptomatic on methimazole therapy.

Tab. 1 Thyroid function tests.

Test name	Normal range
Potassium	3.5–5.0 mmol/L
Phosphorum	2.5–4.5 mg/dL
FT3	2.0–4.4 pg/mL
FT4	0.7–1.9 ng/dL
TSH	0.4–4.0 µIU/mL
Aldosterone	3.0–30 ng/dL
Renin	4.4–46.1 IU/mL ortho
TRAb	<1.75 IU/L
AbTG	<20 IU/mL
TPOAb	<34 IU/mL
Insulin	2–25 µU/mL

DISCUSSION

Thyrotoxic Periodic Paralysis (TPP) is a rare neurological disorder characterized by muscle paralysis, hypokalemia, and hyperthyroidism. It primarily affects Asian males, with a prevalence of approximately 2%, but is far less common in non-Asian populations, with an incidence of 0.1–0.2% (1–3). Western physicians are often unfamiliar with TPP, which can lead to potential mismanagement and serious consequences (3). Although hyperthyroidism more commonly affects women, TPP predominantly occurs in men, with a male-to-female ratio of 20:1. The peak age of onset is between 20 and 40 years. All forms of hyperthyroidism can be associated with TPP, including

Graves' disease, toxic multinodular goiter, Plummer's disease, thyroiditis, TSH-secreting pituitary adenomas, and iatrogenic thyrotoxicosis. In TPP patients, there is a significant increase in Na+/K+ ATPase activity in skeletal muscle, driven by thyroid hormones. This occurs through both transcriptional activation of the Na+/K+ ATPase gene and direct receptor-mediated effects (4). Hyperadrenergic states in thyrotoxicosis further stimulate the Na+/K+ ATPase via cyclic AMP, causing potassium to shift intracellularly (5). Hypokalemia in TPP results from altered potassium distribution rather than total body potassium depletion (6, 7). Carbohydrate loads, such as in this case, precipitate TPP episodes by increasing insulin levels, which enhance Na+/K+ ATPase activity and intracellular potassium uptake (8). Other triggers include trauma, cold exposure, menstruation, emotional stress, and infections (2). Genetic predisposition also plays a role; mutations in the Kir2.6 potassium channel gene have been identified in some TPP cases (9, 10). A functional loss of Kir2.6, combined with Na+/K+ ATPase hyperactivity, leads to sarcolemmal depolarization, sodium channel inactivation, and muscle inexcitability (11). Neurological symptoms in TPP range from mild proximal weakness, usually affecting the lower limbs, to flaccid tetraplegia. Paralysis is typically symmetrical, sparing bulbar, respiratory, and ocular muscles, though severe cases with such involvement have been reported (12, 13). Thyrotoxicosis symptoms may be absent, and TPP can be the initial manifestation, as seen here (14, 15). Differentiating TPP from thyrotoxic myopathy, which causes persistent weakness, is crucial. Unlike thyrotoxic myopathy, TPP is reversible with potassium correction, and neuromuscular symptoms do not persist outside of attacks (16). Potentially fatal complications include cardiac arrhythmias related to hypokalemia and hyperthyroidism, such as sinus tachycardia, atrioventricular block, and ventricular fibrillation (17–19). Acute TPP management involves intravenous potassium chloride to normalize plasma potassium levels. However, since hypokalemia in TPP results from redistribution rather than depletion, potassium supplementation must be cautious to avoid rebound hyperkalemia, which occurs in approximately 40% of patients, particularly with doses greater than 90 mEq/24 hours (20). Rebound hyperkalemia can cause life-threatening arrhythmias and must be promptly recognized and treated (21, 22). Long-term treatment involves controlling hyperthyroidism with antithyroid medications (methimazole), nonselective β-blockers (propranolol), or definitive therapy with radioiodine or thyroidectomy.

CONCLUSION

Therefore, though rare in Western countries, TPP requires early recognition and prompt treatment to prevent severe complications. Education on this condition is essential for timely diagnosis and appropriate management.

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