Case Report

A Case of Hashitoxicosis in a 39-year-old Male presenting as Acute Hypokalaemic Quadriparesis

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Abstract:

Background: *Thyrotoxic periodic paralysis (TPP) is an uncommon endocrine emergency characterized by acute reversible muscle weakness secondary to intracellular potassium shift in the setting of thyrotoxicosis. Although Graves' disease accounts for most reported cases, TPP associated with Hashitoxicosis remains exceptionally rare. Early diagnosis is crucial because delayed treatment may result in life-threatening cardiac arrhythmias and respiratory failure.*

Case Presentation: *A 39-year-old Asian man presented to the emergency department with sudden-onset flaccid paralysis involving all four limbs for eight hours. Weakness was symmetrical, predominantly affecting proximal muscles without sensory deficits, cranial nerve involvement, respiratory compromise or bowel and bladder dysfunction. Laboratory investigations demonstrated profound hypokalemia (1.7 mmol/L), hypomagnesemia (1.4 mg/dL), hyperphosphatemia (5.3 mg/dL) and biochemical thyrotoxicosis with suppressed thyroid-stimulating hormone (<0.01 μIU/mL), elevated free thyroxine (3.53 ng/dL) and free triiodothyronine (11.2 pg/mL). Autoimmune evaluation revealed markedly elevated anti-thyroid peroxidase and anti-thyroglobulin antibodies with negative TSH receptor antibodies, suggesting Hashitoxicosis rather than Graves' disease. Electrocardiography showed sinus tachycardia with hypokalemic changes. Intravenous potassium replacement resulted in complete neurological recovery within 12 hours. The patient was subsequently placed on hormone replacement for thyrotoxicosis.*

Conclusion: *Hashitoxicosis is an uncommon but important cause of thyrotoxic periodic paralysis. Clinicians should consider autoimmune thyroiditis in patients presenting with acute hypokalemic paralysis, particularly when thyroid receptor antibodies are negative. Prompt correction of hypokalemia together with treatment of the underlying thyrotoxic state results in excellent neurological recovery and prevents recurrence.*

Keywords: *Hypokalemia; Hashitoxicosis; Hashimoto thyroiditis; Thyrotoxic periodic paralysis.*

Introduction

Thyrotoxic periodic paralysis (TPP) is an acquired form of hypokalemic periodic paralysis characterized by transient episodes of muscle weakness resulting from intracellular potassium redistribution in the presence of thyrotoxicosis.[1,2] Although thyrotoxicosis occurs predominantly in women, more than 90% of patients with TPP are men, particularly those of Asian ethnicity. The reported incidence among Asian patients with thyrotoxicosis ranges from 1.8% to 13%, compared with less than 0.2% in Western populations.[1-3]

The pathophysiology involves increased Na^+/K^+-ATPase activity stimulated by excess thyroid hormone, catecholamines and insulin, resulting in rapid intracellular potassium shift rather than total body potassium depletion.[1,3] Common precipitating factors include strenuous exercise, carbohydrate-rich meals, emotional stress, infection, alcohol intake and corticosteroid therapy.[1-3]

Graves' disease is responsible for nearly all reported cases of TPP. In contrast, Hashitoxicosis, a transient thyrotoxic phase of autoimmune Hashimoto thyroiditis caused by destruction of thyroid follicles and release of preformed thyroid hormone, rarely presenting with periodic paralysis.[4,5]

We report a rare case of Hashitoxicosis presenting as severe thyrotoxic periodic paralysis in a previously undiagnosed patient with complete neurological recovery following prompt electrolyte correction.

Case Presentation

A 39-year-old Asian male presented to the Emergency Department with acute onset weakness affecting all four limbs

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for approximately eight hours. The weakness developed suddenly after waking in the morning and rapidly progressed to inability to stand or walk. The weakness was symmetrical and predominantly involved the proximal muscles of the shoulders and hips.

The patient denied dysphagia, dysarthria, diplopia, respiratory difficulty, bowel or bladder dysfunction, sensory symptoms, fever, diarrhoea, trauma, chest pain, or recent viral illness. He reported performing strenuous physical activity during the preceding week. Further questioning revealed several previous episodes of transient mild limb weakness over recent years that resolved spontaneously after rest and never required medical attention.

One month before presentation, he developed bilateral knee pain and was treated with non-steroidal anti-inflammatory drugs by his primary physician. He had no previous diagnosis of thyroid disease, diabetes mellitus, hypertension, renal disease or neuromuscular disorders. There was no family history of periodic paralysis or thyroid disease.

On examination, he was conscious, alert and fully oriented. Blood pressure was 140/80 mmHg, pulse rate 86 beats/minute, respiratory rate 26 breaths/minute, temperature 36.7°C and oxygen saturation 98% on 2 L/min oxygen via nasal cannula.

Neurological examination demonstrated severe symmetrical flaccid quadriparesis with Medical Research Council grade - 1/5 muscle power involving both upper and lower limbs. Muscle tone was markedly reduced and deep tendon reflexes were diminished bilaterally. Cranial nerve examination was normal. Sensory examination, cerebellar testing and meningeal signs were unremarkable. Neck flexor strength was preserved and there was no clinical evidence of ophthalmopathy or palpable thyroid enlargement.

Initial laboratory investigations demonstrated profound hypokalemia of 1.7 mmol/L. Serum magnesium was reduced to 1.4 mg/dL, while serum phosphate was elevated at 5.3 mg/dL. Serum calcium, albumin, renal function, and liver function tests were within normal limits. C-reactive protein was mildly elevated (12 mg/L). HbA1c was 6.9%, consistent with prediabetes, while vitamin D concentration was low at 22 ng/mL.

Electrocardiography demonstrated sinus tachycardia with T-wave inversion and ST-segment depression consistent with severe hypokalemia. No significant arrhythmias were detected.

Considering the severe hypokalemia associated with acute flaccid paralysis, thyroid function testing was performed urgently. Biochemical analysis demonstrated suppressed thyroid-stimulating hormone <0.01 μIU/mL (0.3-4.5), elevated free thyroxine 3.53 ng/dL (0.89-1.76) and elevated free triiodothyronine, 11.2 pg/mL (2.3-4.2), confirming thyrotoxicosis.

Autoimmune thyroid antibody testing revealed markedly elevated anti-thyroid peroxidase antibodies 120 IU/mL (<10) and positive anti-thyroglobulin antibodies 7.5 IU/mL (<4.5). TSH receptor antibodies were negative 1.45 IU/L (<1.5), arguing against Graves' disease.

High-resolution thyroid ultrasonography demonstrated diffuse thyromegaly with heterogeneous parenchymal echotexture. Thyroid radionuclide imaging showed increased tracer uptake compatible with an active thyrotoxic state. Rheumatological investigations, including rheumatoid factor and serum uric acid, were normal. Adrenocorticotropic hormone concentration was also normal.

The patient was admitted to the intensive care unit where intravenous potassium chloride was administered through a central venous catheter at a controlled rate of 10 mmol/hour with continuous cardiac monitoring. Serial potassium measurements showed progressive correction to 3.4 mmol/L after eight hours and complete normalization to 4.4 mmol/L after twelve hours.

Clinical improvement paralleled with correction of hypokalemia. Distal muscle movement returned after eight hours of potassium replacement, and complete neurological recovery with restoration of full muscle power (Medical Research Council grade 5/5) was achieved within twelve hours. No rebound hyperkalemia or cardiac complications occurred during treatment.

Following stabilization, the patient was transferred to the Internal Medicine Department where definitive therapy for thyrotoxicosis was initiated. He was discharged in stable condition with outpatient endocrinology follow-up for monitoring of thyroid function, serum electrolytes and glycaemic status.

Discussion

Thyrotoxic periodic paralysis is an acquired channelopathy caused by intracellular potassium redistribution induced by excess thyroid hormone.[1,3] Thyroid hormone increases expression and activity of Na^+/K^+-ATPase pumps through direct genomic effects and enhanced β-adrenergic responsiveness, promoting rapid intracellular potassium uptake.[3] Hyperinsulinemia following carbohydrate ingestion and catecholamine release following strenuous exercise further amplify this process.[1-3]

Our patient possessed several characteristic risk factors, including male sex, Asian ethnicity and recent strenuous physical activity,[1,2] which probably precipitated the paralytic attack. Interestingly, despite marked biochemical thyrotoxicosis, he lacked classical symptoms such as weight loss, tremor, heat intolerance or palpitations, a common feature reported in patients with TPP.[2,6]

Most cases of TPP are associated with Graves' disease, whereas Hashitoxicosis is rarely implicated.[2,7] Hashitoxicosis represents the transient hyperthyroid phase of autoimmune Hashimoto thyroiditis resulting from inflammatory destruction of thyroid follicles with passive release of stored thyroid hormones.[4,5] Unlike Graves' disease, thyroid hormone excess is not mediated by TSH receptor stimulation.

The diagnosis in our patient was supported by strongly positive anti-thyroid peroxidase and anti-thyroglobulin

antibodies together with negative TSH receptor antibodies, findings highly suggestive of autoimmune thyroiditis rather than Graves' disease.[4,5,7] Although radionuclide uptake demonstrated increased tracer accumulation, overlap between autoimmune thyroid disorders has been described, emphasizing that serological findings remain essential for accurate etiological diagnosis.[4,5,7]

The differential diagnosis included familial hypokalemic periodic paralysis, Guillain–Barré syndrome, acute transverse myelitis, cervical spinal cord disease, renal tubular disorders, primary hyperaldosteronism, Bartter syndrome and Gitelman syndrome.[8,9] Preservation of sensation, rapid recovery following potassium replacement, biochemical thyrotoxicosis and absence of a positive family history strongly supported TPP.[1,8]

Treatment consists of cautious potassium replacement with continuous cardiac monitoring because total body potassium stores remain normal.[1,3] Excessive potassium administration may produce rebound hyperkalemia after reversal of intracellular potassium redistribution.[1] Several studies have demonstrated that non-selective β-blockers, particularly propranolol, rapidly reverse paralysis by inhibiting β-adrenergic stimulation of Na^+/K^+-ATPase and should be considered in appropriate patients.[2,7] Long-term prevention depends on achieving a euthyroid state, after which recurrent paralytic episodes rarely occur.[2,7]

This case is noteworthy because of the uncommon underlying etiology, severe degree of hypokalemia, rapid neurological recovery, absence of overt hyperthyroid manifestations and recurrent undiagnosed episodes preceding the definitive diagnosis. Recognition of Hashitoxicosis as a potential cause of TPP expands the differential diagnosis of acute hypokalemic paralysis and highlights the importance of comprehensive thyroid antibody testing in patients presenting with unexplained thyrotoxicosis.

Conclusion

Hashitoxicosis is a rare but clinically significant cause of thyrotoxic periodic paralysis. Patients may present with profound hypokalemia and acute flaccid quadriparesis despite lacking classical symptoms of hyperthyroidism. Early recognition, prompt potassium replacement, continuous cardiac monitoring and definitive management of the underlying thyroid disorder are essential to ensure complete recovery and prevent recurrence. Comprehensive thyroid antibody evaluation should be performed in all patients with TPP to distinguish Hashitoxicosis from Graves' disease and guide appropriate long-term management.

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