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Case Report

Thyrotoxic periodic paralysis presenting as acute quadriparesis in a middle-aged male: a case report

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare, potentially life-threatening complication of hyperthyroidism characterised by acute flaccid paralysis due to hypokalaemia. We report the case of a 46-year-old man who presented with sudden-onset generalised weakness that progressed to quadriparesis over a few hours. Laboratory investigations revealed severe hypokalaemia (2.6 mmol/l) and thyrotoxicosis (FT3 8.76 pmol/l, FT4 32.71 pmol/l, TSH 0.01 µIU/ml), with elevated thyrotropin receptor antibody (TRAb 12.2 IU/l). Ultrasound revealed diffuse thyroid enlargement with increased vascularity. The patient received intravenous potassium supplementation, propranolol, and carbimazole and completely recovered within 24 hours. He was advised to avoid precipitating factors such as high-carbohydrate meals, alcohol, and strenuous activity, and was referred for definitive management of hyperthyroidism. This case highlights the importance of considering TPP in Asian males presenting with acute flaccid paralysis and hypokalaemia. Early recognition, cautious potassium replacement, and timely initiation of antithyroid therapy are essential to prevent serious complications and ensure complete recovery.

Keywords: Acute paralysis, Graves' disease, Hypokalaemia, Thyrotoxic periodic paralysis

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is an uncommon manifestation of thyrotoxicosis, characterised by acute episodes of muscle weakness associated with hypokalaemia. The disorder occurs predominantly in men, despite the higher prevalence of thyrotoxicosis in women, and is seen more frequently among Asian populations.[1]

The pathophysiology involves a sudden intracellular shift of potassium mediated by thyroid hormone-induced upregulation of Na^+/K^+-ATPase activity, further enhanced by catecholamines and insulin. This leads to rapid development of flaccid paralysis that can progress within hours.[2,3]

Clinically, patients present with acute, symmetrical limb weakness, often involving the lower extremities more severely, while the sensory function remains intact. Life-threatening complications, including arrhythmias and respiratory compromise, may occur if diagnosis and management are delayed.[4] Differential diagnoses include familial periodic paralysis, Guillain-Barré syndrome, and hypokalaemia due to renal or gastrointestinal loss of potassium. However, the presence of hyperthyroidism with hypokalaemia is considered diagnostic for TPP.[5,6]

This report describes a middle-aged man with TPP who presented with acute quadriparesis. The aim of this study was to highlight the importance of early recognition, appropriate correction of hypokalaemia, and initiation of antithyroid therapy to prevent recurrence and complications.

CASE REPORT

History of presenting illness

A 46-year-old male presented with a sudden onset of generalised weakness that progressed within hours to quadriparesis. He reported difficulty in standing and walking but denied trauma, recent illness, vomiting, or similar past episodes. There was no significant family history of periodic paralysis. On examination, he was alert with stable vitals (BP-130/80 mmHg, HR-96 bpm, SpO_2-98% on room air, afebrile). Neurological evaluation revealed symmetric weakness in all four limbs, more pronounced in the lower extremities, and decreased deep tendon reflexes. The sensory function was preserved.

Pathological investigations

Serum potassium levels were markedly reduced (2.6 mmol/l). Thyroid function tests revealed elevated FT3 (8.76 pmol/l) and FT4 (32.71 pmol/l) levels with suppressed TSH (0.01 µIU/ml). The thyrotropin receptor antibody levels were also strongly positive (12.2 IU/l). Electrocardiography showed a normal sinus rhythm without U-waves or arrhythmias. Echocardiography revealed concentric left ventricular hypertrophy with a preserved ejection fraction (55%) and mild mitral regurgitation. Neck ultrasound revealed diffuse thyroid enlargement with increased vascularity. Nerve conduction studies were normal, and the random blood glucose level was 108 mg/dl.

Management and outcomes

The patient received intravenous potassium chloride (40 mEq over 6 hours under ECG monitoring), propranolol 40 mg three times daily, and carbimazole 20 mg daily. Muscle strength improved within 12 hours, with complete neurological recovery by 24 hours. He was counselled to avoid precipitating factors such as high-carbohydrate meals, alcohol, and strenuous exercise. An endocrinology consultation was arranged for the definitive management of thyrotoxicosis.

DISCUSSION

TPP is a rare complication of hyperthyroidism, characterised by acute, reversible, flaccid paralysis associated with hypokalaemia. Although hyperthyroidism is more common in women, TPP predominantly affects men, particularly those of Asian ethnicity. The pathophysiology involves increased Na^+/K^+-ATPase activity stimulated by thyroid hormones, β-adrenergic activation, and insulin, leading to a rapid intracellular shift of potassium and subsequent muscle weakness.[7,8] In our patient, the sudden onset of quadriparesis with a serum potassium level of 2.6 mmol/l was consistent with this mechanism.

The diagnosis of TPP relies on the recognition of the association between acute hypokalaemic paralysis and thyrotoxicosis. Differentiating TPP from familial periodic paralysis, Guillain-Barré syndrome, and secondary hypokalaemia due to renal or gastrointestinal losses is essential.[9] Our patient had no family history or preceding illness, and the nerve conduction studies were normal, supporting the diagnosis of TPP. The presence of elevated TRAb and ultrasound findings of a diffusely enlarged thyroid further confirmed underlying Graves' disease, which aligns with reports that autoimmune hyperthyroidism is the most common cause of TPP.[10]

Complications of TPP include the development of cardiac arrhythmias and respiratory muscle weakness, both resulting from severe hypokalaemia.[9] Careful monitoring during potassium replacement is essential, as excessive supplementation may lead to rebound hyperkalaemia once potassium shifts back into the extracellular space. In this case, the patient received cautious intravenous potassium, along with propranolol and carbimazole, which resulted in rapid recovery within 24 hours.[11]

Long-term management requires definitive control of hyperthyroidism to prevent further paralytic episodes. Available options include antithyroid drugs, radioiodine ablation, and thyroidectomy.[12] The patient was referred for endocrinology follow-up to plan definitive therapy. He was also counselled to avoid recognised precipitating factors, such as high-carbohydrate meals, alcohol intake, and strenuous physical exertion, in line with preventive strategies reported in clinical studies.[10,13]

CONCLUSION

TPP should be considered in Asian men presenting with acute flaccid paralysis and hypokalaemia. Optimal management involves cautious potassium supplementation with concomitant initiation of beta-blockers and antithyroid therapy, while definitive treatment of hyperthyroidism is essential to prevent recurrence and ensure sustained recovery.

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