

CASE REPORT

Thyrotoxic periodic paralysis: an unusual presentation of weakness

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Thyrotoxic periodic paralysis is a rare endocrine disorder seen predominantly in men of Asian origin. The case is reported of a patient who presented to the accident and emergency department with sudden onset of weakness of his lower limbs. Hypokalaemia was identified and treated with resolution of symptoms. Additional tests identified the patient as being thyrotoxic. He was treated with oral antithyroid drugs. It is important to consider the diagnosis of thyrotoxic periodic paralysis in patients presenting with acute onset of weakness. The report discusses the epidemiology, presentation, treatment, and complications of this condition.

Thyrotoxic periodic paralysis (TPP) is a rare endocrine disorder. The prevalence as determined in a study of hyperthyroid patients in North America was 0.1%–0.2%.¹ TPP is more common in Asians and more cases are being seen in Europe and America because of migration. The condition may present as a life threatening emergency and unfamiliarity with the syndrome could result in a fatal outcome.² Compliance with therapeutic management plays an important part in the treatment of this condition.

CASE REPORT

A 35 year old Chinese man presented to the accident and emergency (A&E) department complaining of inability to walk. He described a sudden weakness of his legs two days before admission and was unable to walk or stand unassisted. His symptoms were progressive. There was no history of trauma, fever, diarrhoea, weight loss, heat intolerance, or palpitations. There was no significant past illness or family history.

On examination there was no tremor or goitre. His pulse was 72/min, blood pressure 116/61 mm Hg, and Glasgow Coma Score was 15/15. Neurological examination revealed increased tone in the lower limbs, 2/5 power at the hip, and 4/5 power distally. Reflexes were brisk and plantars were flexor bilaterally. Power in the upper limb was 4/5 proximally and 5/5 distally. Tone and reflexes were normal bilaterally. There were no sensory abnormalities and the cranial nerves were intact. Systemic examination was normal. Laboratory studies revealed potassium of 2.6 mmol/l (normal range 3.5–5 mmol/l), glucose 5.1 mmol/l, thyroid stimulating protein <0.001 mU/l, free T₄ 45.90 pmol/l (normal range 9.1–24 pmol/l), antithyroid peroxidase antibody titre 2134 U/ml (normal range 0–50), and IgM 2.72 (normal range 0.5–1.5). His ECG showed prominent U waves typical of hypokalaemia.

The patient was admitted and given intravenous and oral potassium supplements. After replacement with 100 mmol of potassium chloride intravenously and 40 mmol orally his symptoms resolved. He was given carbimazole 20 mg once daily when his hyperthyroid state had been confirmed. This was increased to 40 mg once daily on the third day. He was discharged on the sixth day with complete resolution of symptoms and advice regarding follow up and compliance to treatment.

DISCUSSION

The first case of non-specific periodic paralysis was described in 1882 and a relation with hyperthyroidism was identified in 1902.³ Periodic paralysis is a rare complication of hyperthyroidism, more common in Asian men between the second and fourth decades of life. A high carbohydrate meal, warm weather, increased physical exertion, insulin, adrenaline, and potassium sparing diuretics are usual precipitants.⁴ Proximal muscles of the limbs (lower > upper) are affected with sparing of the sensory system, higher mental functions, and cranial nerves. Patients can present with respiratory failure,⁵ cardiac arrhythmias, and thyrotoxic crisis.⁶ A differential diagnosis of familial periodic paralysis, barium poisoning, and TPP should be considered. Familial periodic paralysis is differentiated by the lack of hyperthyroidism, positive family history, and earlier onset. It is transmitted as an autosomal dominant disorder and is more common in the white population.⁷

The primary defect in TPP is an intracellular sequestration of potassium with normal potassium stores in the body.⁸ Thyroid hormones change the plasma membrane permeability to potassium by increasing the Na/K ATPase activity.⁹ There is an increase in the β adrenergic receptors in skeletal muscles, which increase the Na/K ATPase activity.⁷ These factors along with insulin and testosterone increase the intracellular shift of potassium. Electron microscopic sections reveal a dilatation of the sarcoplasmic reticulum.¹⁰ The condition is associated with HLA-DRw8 5 and A2BW22/AW19B17.¹¹ In contrast, in familial periodic paralysis the intracellular shift of potassium is Na/K ATPase independent and an increase in non-aldosterone mineralocorticoids is present. It responds well to treatment with spironolactone.¹²

In an A&E department a diagnosis of TPP should be considered in men of Asian descent presenting with acute paralysis. A higher index of suspicion should be exercised when there is symmetrical muscle weakness affecting the proximal muscles more than the distal. A history of exacerbation after a large carbohydrate meal, warm weather, or heavy exertion should prompt investigations towards TPP. Urgent blood investigations to support a clinical diagnosis of weakness should include a full blood count, electrolytes, glucose, calcium and phosphorous, C reactive protein, erythrocyte sedimentation rate, and liver function tests.¹³ Thyroid function tests must be requested on a routine basis. Patients suspected to have TPP should be monitored while in the department. Continuous ECG monitoring to check for arrhythmias, arterial blood gas pressures, and peak flows to monitor respiratory function¹⁴ and potassium levels should be monitored.

Treatment of TPP requires urgent correction of potassium levels. Patients with potassium concentrations above 2.5 mmol/l and mild weakness should be treated with 80 mmol/24 h of oral potassium. Patients presenting with potassium concentrations below 2.5 mmol/l or with symptoms of paralysis should be treated with intravenous potassium. This should be given cautiously at a rate of 20 mmol/h¹⁵ as aggressive treatment with potassium supplements is known to cause rebound hyperkalaemia. As serum potassium concentrations

rise oral potassium supplements should be introduced. Treatment of hyperthyroidism with antithyroid drugs is central to the management of TPP. Propranolol added to the initial treatment counteracts the peripheral effects of thyrotoxicosis and improves muscle strength. Glucocorticoids decrease the release of T3 and T4 from the thyroid and inhibit the peripheral conversion to T3.

Long term treatment of TPP entails control of hyperthyroidism. Propylthiouracil has been shown to effectively control hyperthyroidism and the symptoms. Euthyroidism must be maintained for at least six months before a cure of TPP may be considered.¹⁶ Symptoms recur with poor control. Iodine ablation and surgical management with subtotal thyroidectomy are curative.

To the best of our knowledge this is the third reported case of TPP in the UK. The diagnosis must be considered in patients of Asian origin presenting with acute paralysis. Considering migration trends into Europe and America, an increase in the number of cases in the UK can be expected.

Contributors

B Paul, the principal investigator initiated, planned, and researched into the writing of the paper. P Hirudayaraj participated in the study design, core issue discussion, data collection, research, and editing of the paper. M W Baig coordinated the research, discussed core aspects, and edited the paper.

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