

EDITOR'S
CHOICE

CASE REPORT

Sudden flaccid paralysis

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Accepted 25 December 2014

SUMMARY

Periodic thyrotoxic paralysis is a genetic condition, rare in the West and in Caucasians. Thyrotoxicosis, especially in western hospitals, is an easily overlooked cause of sudden-onset paralysis. We present a case of a 40-year-old man who awoke one morning unable to stand. He had bilateral lower limb flaccid weakness of 0/5 with reduced reflexes and equivocal plantars; upper limbs were 3/5 with reduced tone and reflexes. ECG sinus rhythm was at a rate of 88/min. PR interval was decreased and QT interval increased. Bloods showed potassium of 1.8 mEq/L (normal range 3.5–5), free T4 of 29.2 pmol/L (normal range 6.5–17) and thyroid-stimulating hormone (TSH) of <0.01 mIU/L (normal range 0.35–4.94). Random urinary potassium was 8.8 mEq/L (normal range 12.5–62.5). The patient was admitted initially to intensive therapy unit and given intravenous potassium. His symptoms resolved within 24 h. He was diagnosed with thyrotoxic periodic paralysis. He was discharged on carbimazole and propranolol, and follow-up was arranged in the endocrinology clinic.

BACKGROUND

We present an unusual cause of sudden-onset quadriplegia in a young, previously fit patient.

Periodic thyrotoxic paralysis is a genetic condition, rare in the West and in Caucasians. It is more common in Asia, especially in Asian men: up to 2% of thyrotoxic patients of Asian descent may present with paralysis.¹

CASE PRESENTATION

A 40-year-old man awoke one morning unable to stand.

He had a flu-like illness 2 weeks previously, and had experienced occasional muscle aches. There was no family history of note. His family were originally from the Philippines.

He presented to the accident and emergency department with bilateral lower limb flaccid weakness of 0/5 with reduced reflexes and equivocal plantars; upper limbs were 3/5 with reduced tone and reflexes. Sensation was normal. Cranial nerves were intact and Glasgow Coma Scale (GCS) was 15. He had urinary retention, but there was no saddle anaesthesia. The patient was haemodynamically stable; respiratory and cardiovascular examinations were unremarkable.

INVESTIGATIONS

ECG sinus rhythm was at a rate of 88/min. PR interval was decreased and QT interval increased. Bloods showed potassium of 1.8 mEq/L (normal range 3.5–5), free T4 of 29.2 pmol/L (normal

range 6.5–17) and TSH of <0.01 mIU/L (normal range 0.35–4.94). Other electrolytes, including magnesium, were normal. Random urinary potassium was 8.8 mEq/L (12.5–62.5).

DIFFERENTIAL DIAGNOSIS

- ▶ Periodic paralysis—multiple types exist
- ▶ Guillain-Barré syndrome
- ▶ Other cord lesion

TREATMENT

The patient was admitted initially to intensive therapy unit and given intravenous potassium. His symptoms resolved within 24 h and he was stepped down to a normal acute medical bed.

On specialist neurological advice we arranged for MRI of the head and spine. It showed a 'developmentally compromised' image with sagittal diameter of cervical canal measuring less than 10 mm, with localised spinal stenosis at C5/C6, but no spinal cord impingement (figure 1). MRI of the head was normal (figure 2). Ultrasound of the thyroid was unremarkable.

We started the patient on carbimazole 20 mg twice daily and propranolol on the advice of the endocrinology consultant.

OUTCOME AND FOLLOW-UP

After discussion with the neurological specialist centre, the patient was diagnosed with thyrotoxic periodic paralysis. He was discharged on carbimazole and propranolol, and follow-up was arranged in the endocrinology clinic, as well as with the



Figure 1 T1-weighted MRI of the cervical spine.



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To cite: Tariq M, Peshin R, Ellis O, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-206872

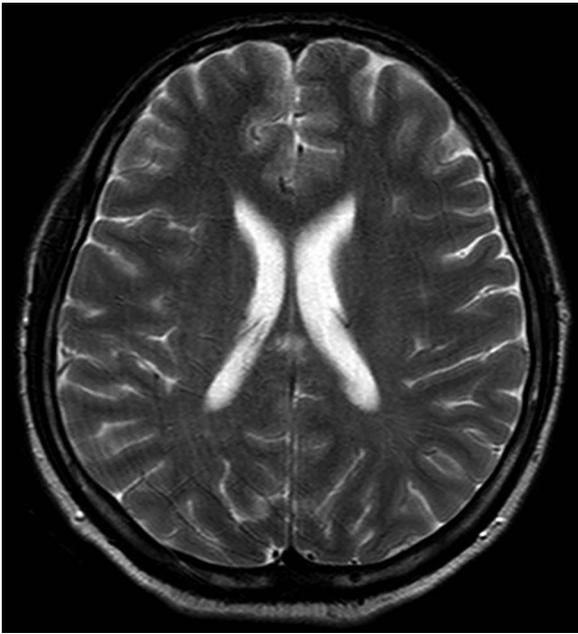


Figure 2 T1-weighted MRI of the cervical spine.

patient's general practitioner to monitor for carbimazole side effects.

Unfortunately, the patient was readmitted 3 days later with the same symptoms. His free T4 was 23.7 pmol/L and TSH was <0.01 mIU/L. Potassium was again replaced, and the patient was again discharged with full resolution of his symptoms. His carbimazole was increased to 30 mg twice daily, and he was additionally advised to reduce intake of carbohydrate-rich foods such as rice and bread, which could contribute to a recurrence.

DISCUSSION

Thyrotoxic periodic paralysis is a rare condition but can be a life-threatening complication of thyrotoxicosis. It is commonly seen in the Asian population and predominantly in males, with an incidence of approximately 2% in patients with thyrotoxicosis of any cause.¹

The attack is characterised by recurrent, transient episodes of muscle weakness, which can range from mild weakness to complete flaccid paralysis. Proximal muscles are affected more severely than distal muscles. The attack usually first involves the lower limbs and subsequently the upper limbs. Sensory function, bowel and bladder function are never affected.^{2–4} Patients may experience recurrent episodes of weakness that last from a few hours up to 72 h, with complete recovery in-between the attacks. In the majority of patients, deep tendon reflexes are markedly diminished or absent, although some patients may have brisk or normal jerks, even during paralysis.⁵

Patients experience the attack after strenuous physical activity or consumption of a high-carbohydrate diet.

The mechanism leading to periodic paralysis is uncertain. It has been shown that these patients have a higher number and more activity of the Na-K-ATPase pump.⁶ Pump activity is increased by thyroid hormone, catecholamine and insulin, which leads to shift potassium into the cells.⁷

Diagnosis is based on history and findings of hypokalaemia, which is the hallmark of periodic thyrotoxic paralysis and hyperthyroidism. Standard treatment for paralytic attack is administration of potassium to prevent major cardiopulmonary complications. Definitive therapy is the management of the thyrotoxic state. To prevent further attacks, β -adrenergic blockers such as propranolol are administered until a euthyroid state is achieved. Low carbohydrate diet and potassium sparing diuretics are effective in preventing further attacks.⁸ Potassium supplements are not indicated for prophylaxis.⁹

Learning points

- ▶ Periodic thyrotoxic paralysis should be considered in the differential diagnosis in patients with sudden-onset paralysis.
- ▶ Failure to diagnose and treat this condition can be fatal.
- ▶ There is a risk of rebound hyperkalaemia if potassium is replaced too aggressively.
- ▶ Oral potassium has no role in preventing recurrence.

Contributors All authors contributed to reviewing the evidence and writing and correcting the article.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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