CASE REPORT

Paralysis that easily reverses: a case of thyrotoxic periodic paralysis

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Accepted 11 January 2017

SUMMARY

Thyrotoxic periodic paralysis is a potentially life-threatening condition associated with recurrent episodes of muscle weakness and hypokalaemia due to hyperthyroidism. Diagnosis is often delayed or misdiagnosed due to its rarity in the western world and subtle features of hyperthyroidism on initial presentation. Here we present the case of a 25-year-old man who presented to the emergency department (ED) with sudden onset weakness of bilateral upper and lower extremities. His labs revealed hypokalaemia with elevated T4 and suppressed thyroid-stimulating hormone and he was diagnosed with thyrotoxic periodic paralysis. He was treated with potassium repletion, atenolol and methimazole with complete reversal of his paralysis within the next day. Unfortunately, he failed to keep the follow-up appointment after discharge, ran out of his methimazole and landed up in the ED again.

BACKGROUND

Thyrotoxic periodic paralysis (TPP) is a type of hypokalaemic periodic paralysis. A case with hypokalaemic periodic paralysis can be either due to a familial (primary) cause or secondary cause. Secondary causes include thyrotoxicosis, hyperaldosteronism, diabetic ketoacidosis, nephrotic syndrome, drugs, acute tubular necrosis, laxative or diuretic abuse, diarrhoea and vomiting.¹ TPP is characterised by recurrent episodes of transient muscle weakness and hypokalaemia due to uncontrolled hyperthyroidism.² It is often missed during initial attacks due to its rarity in the Western world and often mild hyperthyroid symptoms.^{3–4} TPP is rare but easily reversible. However, if missed, it can be debilitating and fatal.⁴ It is crucial to differentiate TPP from familial hypokalaemic periodic paralysis (FHPP) as management differs significantly despite similar clinical manifestations.

CASE PRESENTATION

A 25-year-old man with no significant medical history presented to the emergency department (ED) after he woke up with sudden onset weakness in his bilateral thighs and arms. His weakness was so profound that he required assistance even to stand up. On further questioning, he revealed a history of heat intolerance, palpitations and weight loss of 40 pounds over the past 3 months but denied any changes in skin, hair or bowel movements. He denied any diarrhoea, vomiting, laxative or diuretic abuse. He denied any intake of supplements, alcohol or drug abuse. His history was negative for any recent contrast scans or thyroid disease

in the family. Vital signs included blood pressure of 105/54 mm Hg, pulse 106/minute and regular, temperature 36.4°C and respiratory rate 20/minute. Body mass index was 26.93 kg/m². He was alert, oriented and in no acute distress. Physical examination revealed a diffusely enlarged thyroid gland with bruit but no orbitopathy. Power was 3/5 and reflexes were diminished but sensation was intact.

INVESTIGATIONS

Pertinent labs included serum potassium of 1.9 mEq/L (3.5–5.1 mEq/L), TSH <0.006 µIU/mL (0.3–5 µIU/mL), free T₄ 3.71 ng/dL (0.58–1.64 ng/dL), free T₃ 20.1 pg/mL (2.2–4.1 pg/mL), Thyroid-stimulating immunoglobulin 391% (<122%) and antithyroid peroxidase antibody 415 IU/mL (<8 IU/mL). Serum magnesium was 1.5 mg/dL (1.9–2.7 mg/dL), calcium 10.5 mg/dL (8.6–10.3 mg/dL) and parathyroid hormone 6 pg/mL (12–88 pg/mL). Complete blood count, serum sodium, creatinine and liver function test were unremarkable. ECG revealed sinus tachycardia. Ultrasonography of the neck revealed a diffusely enlarged vascular thyroid gland.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of periodic paralysis includes primary (familial) and secondary causes. Primary causes include hypokalaemic or hyperkalaemic periodic paralysis and Andersen syndrome while TPP is a secondary cause. All of these four disorders are identical in presentation but TPP is rarely associated with a positive family history and has late onset of paralytic attacks. Unlike TPP, the former three conditions are autosomal dominant and manifest within the first two decades of life. Obviously, other secondary causes of hypokalaemia such as diarrhoea, vomiting, diuretic or laxative abuse, hyperaldosteronism, nephrotic syndrome, drugs (theophylline, corticosteroids, tocolytics, amphotericin B), etc should be ruled out before diagnosing TPP.

TREATMENT

His clinical presentation and laboratory abnormalities were consistent with thyrotoxic periodic paralysis. He was started on potassium supplementation, atenolol and methimazole after which he started feeling better within the next few hours. His power returned to 5/5 and deep tendon reflexes 2+ on all extremities.

OUTCOME AND FOLLOW-UP

His paralysis resolved completely and he was discharged the next day. He was discharged home on

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To cite: Tachamo N, Lohani S, Nazir S, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-218951

atenolol 25 mg daily and methimazole 30 mg daily with instructions to follow-up with outpatient endocrinology for further management. However, he did not follow-up with his family doctor or endocrinology. He ran out of his methimazole and did not take them for a couple of weeks. Subsequently, he landed up in the ED again with symptoms of hyperdefecation, vomiting, intermittent tremors and palpitations. He was found to be hyperthyroid with TSH of 0.041 μ IU/mL, total T4 of 25.1 μ g/dL and free thyroxine index of 32 μ g/dL (5.9–13 μ g/dL). His methimazole and atenolol were resumed, his symptoms improved and he was discharged home to follow-up with outpatient endocrinology for further management.

DISCUSSION

TPP is a potentially life-threatening complication of thyrotoxicosis characterised by recurrent episodes of muscle weakness and hypokalaemia.^{3–5} It is usually associated with Grave's disease but may occur with any other form of thyrotoxicosis.^{6 7} It is rare and occurs in just 0.1–0.2% of the hyperthyroid population in North America.^{2 8} TPP usually affects middle-aged patients in their 20s–40s.⁸ The prototypical patient is a man of age >20 years and of Asian descent; however, it is being increasingly seen in the western world due to population mobility, admixture and increasing obesity leading to insulin resistance.³ Interestingly, unlike other thyroid disorders which are more common in females, TPP occurs more in men (male:female ratio of 20:1)⁷ due to the increased Na-K-ATPase activity from testosterone.⁶

The exact pathophysiology of TPP is still unclear. Increase in the number and activity of Na-K-ATPase from hyperthyroidism, the hyperadrenergic state and hyperinsulinaemia results in massive rapid intracellular potassium influx precipitating the attacks of TPP.^{6 7} Recently, mutations in Kir2.6, an inwardly rectifying potassium channel, has been identified, whose paradoxical activity during periods of thyrotoxicosis can trigger attacks of TPP.³

TPP is diagnosed based on clinical and biochemical evidence of hyperthyroidism with hypokalaemia in a patient with a history of paralytic attacks.³ Attacks tend to occur more at night or early in the morning on awakening,⁵ as in our patient. Attacks are usually precipitated by strenuous exercise, high carbohydrate meals or alcohol intake.^{5 6 8} Proximal muscles are affected more severely than distal ones and lower extremities are affected more than the upper ones. The severity of muscle weakness correlates with the degree of hypokalaemia. Deep tendon reflexes are usually diminished or absent.⁶ Bulbar, ocular and respiratory muscles are rarely involved. Sensory functions and cognition remain intact.⁴ Rare complications include life-threatening ventricular arrhythmias, acute hypercapnic respiratory failure and colonic pseudo-obstruction secondary to hypokalaemia.⁶ Many patients do not have obvious signs and symptoms of hypothyroidism and diagnosis may go undetected if not carefully sought for.³

Definitive management of TPP involves management of thyrotoxicosis with antithyroid drugs, surgery or radioactive iodine.⁴ An acute attack is usually transient and lasts hours to days, even without treatment.³ An acute episode is treated with potassium replacement but rebound hyperkalaemia can occur if excess potassium is supplemented as total body potassium is

normal. Either oral or parenteral potassium may be used.⁶ It should be noted that vigorous potassium replacement can be potentially fatal in these cases as this electrolyte disorder occurs due to relative deficiency from increase in β -adrenergic sensitivity and potassium influx into the cells rather than potassium depletion.¹ Unlike FHPP, there is no role of regular potassium supplementation or potassium sparing diuretics in between attacks.⁶ Future attacks can be prevented only by maintaining a euthyroid state, not by potassium supplementation; however, non-selective β -blockers can be used to prevent attacks until a euthyroid state is achieved.⁴ Precipitating factors such as a high carbohydrate diet or strenuous exercise should be avoided until a euthyroid state is achieved. As long as a euthyroid state is maintained, recurring attacks are virtually non-existent.³

Learning points

- ▶ Thyrotoxic periodic paralysis (TPP) should be considered in all cases of acute paralysis with hypokalaemia even without preceding hyperthyroid symptoms.
- ▶ It is critical to differentiate TPP from familial hypokalaemic periodic paralysis as management differs significantly despite similar clinical presentations.
- ▶ Diagnosis of TPP is made by finding hypokalaemia in the presence of hyperthyroidism during attacks of paralysis.
- ▶ Potassium supplementation is not helpful to prevent further attacks of TPP but can only be prevented by maintaining a euthyroid state.

Contributors NT contributed by writing bulk of the case report. He also participated in concept, presentation style and literature search. SL wrote part of the case presentation. He also gave valuable feedback on the initial draft and carried out some major corrections. SN participated in discussing and planning of the case report format and presentation style. He also helped to carry out literature search and also edited the manuscript before submission. NJ proofread the original draft and gave feedback and carried out corrections including typographical errors. NJ was also part of the patient care team.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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