Brief Communication

Three cases of thyrotoxic periodic paralysis due to painless thyroiditis

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ABSTRACT

We present three cases of thyrotoxic periodic paralysis (TPP) due to painless thyroiditis presenting as acute quadriparesis. All responded to potassium supplementation and propranolol. TPP may be due to thyrotoxicosis of any etiology, commonly Grave's disease. The absence of clinical signs of thyrotoxicosis can delay diagnosis and treatment. Thyroid function tests should be a routine evaluation in all cases of hypokalemic periodic paralysis.

Key words: Hypokalaemia, periodic paralysis, thyrotoxicosis, thyroiditis

Introduction

TPP is a rare complication of thyrotoxicosis, mostly due to Graves' disease but rarely due to thyroxine overdose, toxic multinodular goiter, toxic adenoma, and thyroiditis. TPP is more common in Asians compared to Caucasians.[1-3] We present three cases of TPP due to painless thyroiditis who presented with acute onset quadriparesis. All responded very well to potassium supplementation and propranolol.

Case Reports

Case 1

A 21-year-old male presented with sudden onset flaccid quadriparesis with no preceding history of fever, dysphagia, dysphonia, diplopia, bladder, or bowel involvement. There was no relation with heavy carbohydrate meal, exercise or diarrhea, and no past history of similar episode.

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Examination revealed lower limb proximal muscle power of 3/5, diminished deep tendon reflexes (DTR) in all four limbs, bilateral flexor plantar responses, no cranial nerve palsy, and no respiratory muscle involvement. Goiter and clinical feature of thyrotoxicosis were absent. Serum potassium was 2.2 mmol/l (normal [nl.] –3.5-5), and ESR 38 mm/h (nl.–1-25). Thyroid function tests (TFT) showed TSH 0.06 μU/l (nl. –0.5–5), free T4 2.4 ng/dl (nl. 0.7–2.0), T3 210 ng/dl (normal –70–190), and TPO antibody 54 IU/ml (nl. <35). $^{99\ m}$Tc pertechnetate thyroid scan uptake was 0.15% (nl.–0.4–1%).

Case 2

A 35-year-old male presented with sudden onset quadriparesis with a history identical to that of case 1. He had upper limb proximal muscle powers MRC 3/5, distal muscles 4/5, lower limbs proximal, and distal muscles 2/5. Mild respiratory muscle involvement was evidenced by reduced single breath count of 11 seconds. DTR on all limbs were diminished with bilaterally flexor plantar responses and no cranial nerve palsy. He had a grade 1 goiter and no signs of toxicity. His serum potassium was 1.5 mmol/l, ESR 42 mm/h, TSH 0.005 μU/l, free T4 2.5 ng/dl, TPO antibody 68 IU/ml, and decreased uptake (0.1%) on ^{99m}Tc scan.

Case 3

A 32-year-old male presented with acute onset flaccid paraparesis, tremors of both hands and palpitations. He

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had a history of transient proximal weakness 6 weeks back which recovered spontaneously. He had no dysphagia dysphonia, bladder, or bowel involvement.

He had power 2/5 in proximal muscles, depressed jerks on all four limbs, bilateral flexor plantar responses, and no signs of cranial nerve palsy. He had tachycardia, fine tremors of both hands, and grade1 diffuse goiter. His serum potassium was 1.8 mmol/l, ESR 30 mm/h, TSH 0.03 μU/l, free T4:3.2 ng/dl, TPO antibody 117 IU/ml (nl < 35), and there was decreased uptake (0.21%) on ^{99m}Tc scan.

All three cases had normal TSH receptor antibodies, serum creatine kinase (CK) and collagen vascular profile. Blood gas analysis in all three patients excluded acidosis. NCV was normal but EMG showed a myopathic pattern with no decremental muscle action potential. In all three cases fine-needle aspiration cytology (FNAC) was consistent with lymphocytic thyroiditis. Based on clinical features and investigations our diagnosis for each patient was thyrotoxic periodic paralysis secondary to painless or silent thyroiditis.

Case 1 was managed with oral potassium chloride (KCl) at the rate of 0.2-0.4 mmol/kg and propanolol 120 mg/day. Cases 2 and 3 initially required intravenous KCl at 10 meq/h. All showed marked improvement of paralysis within a week of treatment. All patients were discharged after a week with oral propranolol (80-120 mg/day). After 4 weeks of mean follow-up none had any residual paralysis. At the end of 2 months case 1 presented with normal thyroid profile and propranolol was completely withdrawn. At 3 months the thyroid profile became completely normal in cases 2 and 3 and propanolol was stopped. There was no relapse in any of the three cases even at 6-month follow-up.

Discussion

TPP is a rare disorder characterized by episodic muscle weakness due to hypokalemia. Diagnosis at initial presentation may be delayed because of the subtleness of the clinical features of thyrotoxicosis. Cases of painless thyroiditis presenting with TPP as seen in our series have rarely been reported, although thyrotoxicosis is more common in females TPP is more common in 20- to 40-year-old males, as observed in our series. Many patients of TPP may not have obvious signs and symptoms of thyrotoxicosis, as seen here.

In TPP, involvement is mostly proximal and motor resulting in quadriparesis without bowel and bladder affection as in our cases. Impairment of bulbar or respiratory muscles is rare except in a severe attack of TPP, like our second case.[4] The hallmark of TPP is hypokalemia, usually less than 3.0 mmol/l, present in all our cases. The degree of initial hypokalemia has a direct correlation with the severity of paralysis but not with the thyroid hormone level. Hypokalemia is most likely due to high levels of thyroid hormone causing overactivity of the Na + K^+ ATPase pump triggering intracellular shift of the potassium causing muscle hyperpolarization.[1,5]

In cases of acute attacks of TPP, immediate restoration of serum potassium via intravenous route and oral propranolol is necessary.[1,6,7] Because TPP does not recur once the patient is euthyroid, adequate control of hyperthyroidism by antithyroid drugs, radioiodine therapy, or thyroidectomy is imperative.[1] β-adrenergic blockers like propranolol prevent attacks until euthyroidism is achieved.

Conclusion

Quadriparesis due to TPP may be the first presentation of thyrotoxicosis of any etiology including painless thyroiditis. The painless nature of thyroiditis and absence of clinical signs of thyrotoxicosis can delay diagnosis and treatment. A thyroid function assay should be done routinely while evaluating patients with hypokalemic paralysis to distinguish TPP from other forms of hypokalemic paralysis.

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