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Case Report

Painless but not Harmless: Paradoxical Hypokalemia in Thyrotoxic Periodic Paralysis Due to Thyroiditis

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Abstract

Background: Thyrotoxic Periodic Paralysis (TPP) is a rare but life-threatening complication of thyrotoxicosis, characterized by recurrent episodes of muscle weakness associated with hypokalemia. Although Graves' disease is the most common cause, thyroiditis is an unusual etiology.

Case: We report a 20-year-old male who presented with acute flaccid quadriparesis. Investigations revealed hypokalemia, suppressed TSH with elevated free T4, positive thyroid autoantibodies and reduced radionuclide uptake suggestive of thyroiditis. He improved with potassium replacement and β-blockers.

Conclusion: This case highlights the importance of thyroid function testing in all patients presenting with hypokalemic paralysis, as silent thyroiditis may rarely present as TPP.

Keywords: Catecholamines; Iymphocytic infiltration; Thyroid radionuclide; Normokalemic; Diplopia

Introduction

Thyrotoxic Periodic Paralysis (TPP) is an acquired channelopathy characterized by episodes of acute muscle weakness associated with hypokalemia in the setting of thyrotoxicosis. It is most frequently reported in Asian populations, with an estimated incidence of 2% in thyrotoxic men of Chinese and Japanese origin, but is much rarer in Western countries [1]. The male predominance (male: female ratio ~20:1) is paradoxical given the higher prevalence of thyrotoxicosis in women [2]. Pathophysiology involves increased Na^+/K^+-ATPase activity in skeletal muscle, stimulated by thyroid hormone, catecholamines and insulin, leading to an abrupt intracellular shift of potassium and subsequent muscle membrane hyperpolarization [3]. Although Graves' disease accounts for the majority of cases, TPP has also been described in toxic multinodular goiter, solitary toxic adenoma and very rarely, thyroiditis [4]. The latter is easily missed due to the absence of classic clinical signs of thyrotoxicosis. Early recognition and treatment are critical because untreated TPP can lead to respiratory compromise, arrhythmias and sudden death [5]. We report a case of TPP precipitated by painless thyroiditis in a young male, underscoring the diagnostic challenges and importance of thyroid function assessment in hypokalemic paralysis. Recent case reports have emphasized that thyrotoxic periodic paralysis can present across a wide spectrum of thyroid disorders and biochemical patterns, including normokalemic and atypical cases of Graves' disease.

Case Description

A 20-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. Examination revealed lower limb proximal muscle power of 2/5, 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 features of thyrotoxicosis were absent. Serum potassium was 2.2mmol/l and ESR 38mm/h. TFT: fT4 2.4ng/dl, TSH 0.06, T3 210ng/dl and TPO antibody 54IU/ml. 99mTc pertechnetate thyroid scan revealed decreased uptake: 0.1% consistent with thyroiditis. Thyroid FNAC found features suggestive of Thyroiditis (lymphocytic infiltration, no giant cells). EMG showed myopathic pattern with no decremental muscle action potential.

Discussion

Our patient presented with acute hypokalemic quadriparesis without overt features of thyrotoxicosis. The differential diagnoses of acute flaccid weakness with hypokalemia include Familial Hypokalemic Periodic Paralysis (FHPP), renal tubular disorders, gastrointestinal potassium loss, thyrotoxic myopathy and drug-induced hypokalemia [6]. The absence of family history, normal urinary potassium excretion, suppressed TSH, elevated free T4 and low thyroid radionuclide uptake established the diagnosis of TPP secondary to thyroiditis. TPP differs from FHPP in several aspects: FHPP usually presents before 20 years of age, has a positive family history, and is not associated with thyroid dysfunction [7]. In contrast, TPP typically occurs in young adult males with subtle or absent clinical signs of thyrotoxicosis, as seen in our case. The rarity of thyroiditis as a cause of TPP is well recognized. Most reported cases are associated with Graves' disease [8], while painless thyroiditis is an unusual trigger. Previous reports have emphasized that lack of thyrotoxic stigmata often delays diagnosis [9]. In our patient, thyroiditis was confirmed by radionuclide imaging and FNAC showing lymphocytic infiltration. The acute management of TPP includes cautious potassium supplementation and non-selective β-blockade to counteract adrenergic stimulation of Na^+/K^+-ATPase [10]. Definitive prevention requires restoration of euthyroidism. In cases due to thyroiditis, TPP episodes usually resolve spontaneously with resolution of thyrotoxicosis, unlike Graves' disease where long-term antithyroid therapy may be necessary. Our case reinforces the importance of thyroid function testing in all patients with hypokalemic paralysis, regardless of clinical suspicion. Prompt recognition avoids unnecessary neurological investigations and prevents recurrence through appropriate management of the underlying thyroid disorder [3]. Similar atypical presentations have been reported in the literature, where TPP was the initial manifestation of Graves' disease, including cases with normal potassium levels or delayed recognition of thyrotoxicosis [11-13].

Conclusion

Quadriparesis due to TPP may be the first manifestation of thyrotoxicosis from any cause, including painless thyroiditis. The absence of goiter and overt clinical signs of hyperthyroidism should not preclude thyroid evaluation in patients with hypokalemic paralysis. Early diagnosis and management are essential to prevent potentially fatal complications.

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