Case Report

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Thyrotoxic Periodic Paralysis

Dr. Anju Paul DNB. (Med), Dr. Saranya MD (Med), Dr. V Mathew MD

G. Kuppuswamy Naidu Memorial Hospital
Coimbatore.

Hypokalemic periodic paralysis is a muscle disease in the family of diseases called channelopathies, manifested by episodes of painless muscle weakness associated with hypokalemia. Although most cases of periodic paralysis are hereditary, acquired cases of hypokalemic PP have also been described in association with hyperthyroidism. Thyrotoxic periodic paralysis is an endocrinological emergency characterized by a triad of muscle paralysis, acute hypokalemia, and hyperthyroidism.

29 yrs old gentleman from Salem, working as a production manager at a garment factory, non-alcoholic, with no known comorbidities presented with recurrent episodes of sudden onset, bilateral, symmetrical lower limb weakness since February 2019 treated locally. The last episode was two days back. Laboratory examination during these attacks revealed severe hypokalemia associated with ECG changes. Other blood parameters include sugar, renal profile, LFT, sodium, and magnesium, were within normal limits. He improved dramatically with oral and IV potassium supplements, was diagnosed with periodic hypokalemic paralysis, and was advised of lifelong oral potassium supplements. He now complained of the increasing frequency of these attacks for the past six months and had a recent attack two days back. He also complained of palpitation and tremors of the hand and came to our hospital in November 2020.

On examination, he was anxious, BP 120/80mm Hg and PR 92/min in a regular rhythm. Examination of the neck revealed a diffuse thyromegaly. He had no features of ophthalmopathy. Examination of the central nervous system revealed fine tremors of the bilateral upper limb and no hypotonia. Power was normal in all four limbs. Deep tendon reflexes were diminished, and bilateral plantar showed a withdrawal response. Other system examinations were within normal limits.

Because of recurrent hypokalemic periodic paralysis and thyromegaly, thyroid function tests were done, which revealed severe hyperthyroidism (TSH < 0.003, FT3- 497, FT4 29); serum potassium repeated in our hospital was normal. (4.6mmol/L) He was started on Neomercazole, beta-blockers, and oral potassium supplements.

The patient was followed up after a month of antithyroid treatment. He improved symptomatically, features of hyperthyroidism improved, had no further episodes of paraparesis or hypokaleamia. Serial potassium was normal, and thyroid function showed an improving trend and hence continued on antithyroid treatment. We discussed treatment options with the patient as he was young, married, and keen on having a child. After explaining the pros and cons and the need to delay having a child if he underwent RAI, to which he was agreeable. He underwent TC 99, Thyroid scan on 1.7.21, which revealed a mildly enlarged thyroid gland with increased trapping function (9.8% uptake) with no definite hot or cold areas suggestive of Graves' disease.

He was given RAI treatment 13.07.2021 – 5mCI of I 131 and followed up on antithyroid drugs and low dose potassium. Serial monitoring of thyroid function was done. 11.11.2021 – TSH 53.7. Thyroid replacement started. 6.12.2021- TSH 45 8.3.2022 – TSH 6. Patient on 100mcg Thyroxine remained well, with no further episodes of limb weakness or hypokalemia. He continued on Thyroxine 100mcg and was advised to avoid a high carbohydrate diet and severe exertion. Monitoring of thyroid profile six-monthly and serum K in case of recurrence of weakness.

Discussion

Thyrotoxic period paralysis (TPP) is a rare and life-threatening manifestation of thyrotoxicosis, documented in 1.9% of the population. Unlike thyrotoxicosis, which is common in females, TPP is commonly seen in Asian men in their third decade of life. It is characterized by recurrent, transient episodes of acute painless bilateral symmetrical flaccid paralysis, hypokalemia, and hyperthyroidism. Acute episodes are usually preceded by muscle aches, cramps, and muscle stiffness and precipitated by strenuous exertion, high-carbohydrate meal, trauma, exposure to cold, emotional stress, infection, alcohol ingestion, menses, and drugs like diuretics, insulin, or steroids. Patients usually present with acute onset proximal symmetrical ascending lower extremity muscle weakness during the early morning hours or while resting after strenuous exertion with absent or decreased deep tendon reflexes and sensory sparing and findings of thyrotoxicosis such as warm and moist skin, fever, tachycardia, exophthalmos or goitre. Hypokalemia in TPP results from an intracellular shift of potassium rather than total body potassium depletion. The pathogenesis of hypokalemic periodic paralysis in certain populations with thyrotoxicosis still remains unclear, and it is largely attributed to the intracellular shift of potassium induced by the direct and indirect thyroid hormone sensitization of N1+/K+ - ATPase leading to an increase in the number and sensitivity of beta receptors. Patients with TPP have been found to have hyperinsulinemia during episodes of paralysis, thus explaining the attacks after high-carbohydrate meals.

In our case, the absence of a family history of paralysis, male sex, presentation in the second to fourth decades of line, and signs of thyrotoxicosis like thyromegaly and fine tremors helped diagnose this disorder. Management of TPP includes correction of hypokalemia and treatment of the underlying hyperthyroid state. Traditionally, patients are given intravenous or oral potassium to hasten muscle recovery and prevent cardiopulmonary complications; however, there is a danger of rebound hyperkalemia due to the release of potassium and phosphate from the cells on recovery. Rebound hyperkalemia had been documented in approximately 40% of patients with TPP, especially if they received >90mEq of potassium chloride within the first 24 hours. Propranolol, a nonselective beta-blocker, prevents the intracellular shift of potassium and phosphate by blunting the hyperadrenergic stimulation of Na+/K+-ATPase. It has been observed that propranolol was given alone either orally (3mg/kg) or intravenously can normalize the serum potassium levels within an average of 120minutes. Thus, in a setting of TTP, a combination of nonselective beta-blockers and low-dose potassium appears to be the treatment of choice for facilitating recovery and reducing rebound hyperkalemia. The definitive therapy for TPP includes treating hyperthyroidism with antithyroid medications, surgical thyroidectomy, or radioiodine therapy. TPP is a curable disorder once a euthyroid state is achieved. Hence, the early diagnosis aids in definitive management and cure and prevents the risk of rebound hyperkalemia due to excessive potassium replacement.

Conclusion

Thyrotoxic periodic paralysis (TPP) is an alarming and potentially lethal but curable complication of hyperthyroidism. Diagnosis of TPP at the initial encounter is often delayed and confused with more familiar causes of hypokalemia and lower extremity paralysis, partially because of the subtleness of the Thyrotoxicosis and partially because of unawareness about this disorder to delay in initiation of definitive treatment. Thus an increased awareness among physicians about this disorder will result in early diagnosis, appropriate treatment, and the prevention of rebound hyperkalemia and also prevention of frequent hospitalization for hypokalemic episodes.

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