



Thyrotoxic Periodic Paralysis - Are We Underestimating the Incidence

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a condition featuring attacks of muscle weakness in the presence of hyperthyroidism. This study was conducted to know the incidence and method of prevention of thyrotoxic periodic paralysis in patients with thyrotoxicosis treated with radioactive iodine. 200 Patients with thyrotoxicosis after withholding anti thyroid drugs were treated with radioactive iodine from 2006 September to 2008 March were analysed for symptoms of periodic paralysis post radio iodine treatment setting. Out of 200 patients 100 patients included in control group were educated about thyrotoxic periodic paralysis and were asked to take natural potassium supplements like coconut water among which 83 patients were given natural supplementation with high potassium diet and the remaining 17 patients received syrup potchlor 2 mL twice daily based on the severity of the thyrotoxicosis. Other 100 patients who were in the test group were not supplemented with potassium and not educated about thyrotoxic periodic paralysis.

Out of the 100 patients who did not receive any potassium supplementation 44 patients presented with generalised muscle weakness in the first 4 weeks post radio iodine therapy, 5 patients presented with periodic paralysis and 1 patient expired due to arrhythmia with ECG evidence of hypokalemic changes, Serum potassium levels less than 3 mEq/L. Among the rest of the 100 patients who received oral supplementation only 1 presented with symptoms suggestive of thyrotoxic periodic paralysis. The incidence of thyrotoxic periodic paralysis is high in the first four to six weeks post radio iodine therapy because of withholding antithyroid drugs as well as initial radio iodine induced thyroiditis. Oral natural potassium supplementation post radio iodine therapy for initial 6 weeks is an adequate prophylactic measure to prevent the morbidity and remote mortality from thyrotoxic hypokalemic periodic paralysis in patients being treated with radioactive iodine.

Key Words: *Thyrotoxic Periodic Paralysis, Hypokalemia, Thyrotoxicosis.*

Introduction

Thyrotoxic periodic paralysis (TPP) is a condition featuring attacks of muscle weakness in the presence of hyperthyroidism. Hypokalaemia is usually present during attacks. The condition may be life-

threatening if weakness of the intercostal muscles leads to respiratory failure, or if the low potassium levels lead to cardiac arrhythmias. Treatment of the hypokalaemia, followed by correction of the hyperthyroidism, leads to complete resolution of the at-

tacks. This study was aimed at for assessing the incidence and method of prevention of thyrotoxic periodic paralysis in patients with thyrotoxicosis treated with radioactive iodine.

Material and Methods

200 Patients presenting to the Nuclear Medicine Department for Radioactive Iodine (RAI) therapy between 2006 September to 2008 March were studied. Two groups were made one group on potassium supplements (Control group) and one group with not on potassium supplements (Test group). Patients were not on antithyroid medication (ATD) and were administered RAI therapy.

Pathophysiology

Thyrotoxic periodic paralysis cause in certain populations with thyrotoxicosis is unclear. Transcellular distribution of potassium is maintained by the $\text{Na}^+/\text{K}^+-\text{ATPase}$ activity in the cell membrane, and it is mainly influenced by the action of insulin and beta-adrenergic catecholamines⁽¹⁾. Hypokalemia in TPP results from an intracellular shift of potassium and not total body depletion. It has been shown that the $\text{Na}^+/\text{K}^+-\text{ATPase}$ activity in platelets and muscles is significantly higher in patients with TPP⁽²⁾. Hyperthyroidism may result in a hyperadrenergic state, which may lead to the activation of the $\text{Na}^+/\text{K}^+-\text{ATPase}$ pump and result in cellular uptake of potassium^(1,3,4).

Thyroid hormones may also directly stimulate $\text{Na}^+/\text{K}^+-\text{ATPase}$ activity and increase the number and sensitivity of beta receptors^(1,5). Patients with TPP have been found to have hyperinsulinemia during episodes of paralysis. This may explain the attacks after high-carbohydrate meals⁽⁶⁾.

Results

200 patients of primary hyperthyroidism presented to the Nuclear Medicine Department for radioiodine therapy, TSH levels <0.02 mU/L (reference range, 0.30–5.00 mU/L). They were divided into two groups of 100 each. First group (Control group) were counselled regarding TPP and potassium supplementation and were started on natural supplements or syrup potchlor, second group (Test group)

consisted of patients who were not started/ counselled about potassium supplements. In first group out of 100 patients which included 63 males and 37 females, only 1 patient (1%) presented with symptoms suggestive of thyrotoxic periodic paralysis and in second group out of the 100 patients who did not receive any potassium supplementation 58 were male and 42 females, 44 patients presented with generalized muscle weakness in the first 4 weeks post radioiodine therapy (44%), 5 patients presented with periodic paralysis (5 %) and 1 patient died due to arrhythmia with ECG evidence of hypokalemic changes(1%). Serum potassium levels were less than 3 mEq/L (reference range, 3.6–5.0 mEq/L). About 44 patients out of 100 patients in test group presented with generalized muscle weakness (44%) whereas none of the patients in control group had muscle weakness.

Discussion

Patients with thyrotoxic periodic paralysis have recurrent muscle weakness of the four extremities affecting mainly the lower extremities. The onset of paralytic attacks usually coincides with onset of hyperthyroidism, though overt findings are rarely present with the initial paralytic attack⁽⁷⁾. In some cases the periodic paralysis is the sole manifestation of the hyperthyroidism⁽⁸⁾. Clinical manifestations of thyrotoxic periodic paralysis include Periodic flaccid paralysis of proximal muscles, mainly of lower extremities, hyperthyroidism, hypertension and negative family history of similar complaints. Laboratory findings include Hypokalemia, Hypophosphatemia, and mild hypomagnesemia and Normal acid base balance.

Thyrotoxic periodic paralysis is a well-established phenomenon. When the symptoms of thyrotoxicosis are separated from the clinical picture, many features of this disease are identical to those of familial hypokalemic periodic paralysis. Periodic paralysis can manifest with any condition associated with hypokalemia. Management of TPP includes correction of hypokalemia and treatment of the underlying hyperthyroid state In our study 5 (5%) patients in the test group presented with TPP where as only 1(1%) patient in the control group presented with

TPP. About 44 patients out of 100 patients in test group presented with generalised muscle weakness (44%) whereas none of the patients in control group had any complaints of muscle weakness, 1 patient in test group died due to arrhythmia with ECG evidence of hypokalemic changes(1%). Serum potassium levels were less than 3 mEq/L (reference range, 3.6–5.0 mEq/L) in patients presenting with TPP or muscle weakness. Because 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 release of potassium and phosphate from the cells on recovery^(9,10). Rebound hyperkalemia occurred in approximately 40% of patients with TPP, especially if they received >90 mEq of potassium chloride within the first 24 hours⁽⁹⁾. There is a positive correlation between the dose of potassium chloride administered and the degree of rebound hyperkalemia^(9,10,11). Patients receiving a total dose of ≤50 mEq of potassium chloride rarely develop rebound hyperkalemia. Hence in our study we could conclude that prophylactic potassium supplementation helped in preventing hospitalization and fatal complication like arrhythmias.

Conclusion

The incidence of thyrotoxic periodic paralysis is high in the first four to six weeks post radioiodine therapy because of withholding antithyroid drugs as well as initial radioiodine induced thyroiditis. Oral natural potassium supplementation post radioiodine therapy for initial 6 weeks is an adequate prophylactic measure to prevent the morbidity and remote mortality from thyrotoxic hypokalemic periodic paralysis in patients being treated with radioactive iodine.

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