



## A CASE OF THYROTOXIC PERIODIC PARALYSIS- AN UNDERDIAGNOSED & UNDERRECOGNISED CONDITION.

### Nephrology

**Dr. Deepthi Kanuganti**

Post Graduate, Dept. Of General Medicine, Government General Hospital, Guntur.

### ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare condition characterized by a triad of symptoms which include acute hypokalemia without total body potassium deficit, episodic muscle paralysis and uncontrolled hyperthyroidism. A 27yr old gentleman presented with complaints of loose stools, palpitations and weakness of all 4 limbs (predominantly lower limbs). Hypokalemia was documented in the patient. Prominent eyes and few signs of wide pulse pressure like water-hammer pulse, locomotor brachii and dancing carotids were also noticed. The above observations at first lead us to believe that the patient may have Hypokalemic Periodic Paralysis with Aortic Regurgitation. After the correction of hypokalemia, weakness regressed to a minimum but exophthalmos and signs of wide pulse pressure were still prevailed in the patient. After re-examining the clinical vignette and doing a thyroid profile & ultrasound of neck on the patient, we finally came to a conclusion and diagnosed the patient as having TPP associated with Hashitoxicosis. Subsequently the patient was treated with propranolol and carbimazole. TPP can often be the first manifestation of thyrotoxicosis as in our case but it is a very rare disorder to encounter in India when compared to western countries. So one should suspect this condition in patients with weakness & hypokalemia because it is a curable cause of hypokalemic periodic paralysis.

### KEYWORDS

#### BACKGROUND

Thyrotoxic periodic paralysis (TPP) is a thyroid related disorder that is manifested as recurrent episodes of hypokalemia and muscle weakness (hypokalemic periodic paralysis) lasting from hours to days. It is most commonly described among Asian men and is a well known complication of thyrotoxicosis. Proximal muscles are affected more severely than the distal muscles. Sensory function is preserved. Bladder symptoms are absent. In majority of patients, deep tendon reflexes are markedly diminished or absent although some patients may show evidence of normal or brisk jerks, even during paralysis[3]. TPP is identical to familial periodic paralysis (FPP), except for the fact that hyperthyroidism is an absolute requirement for the expression of the disease. Weakness rarely progresses to involve the muscles above the neck, thus sparing bulbar and respiratory musculature. Majority of patients have subtle or no symptoms of hyperthyroidism. Treatment consists of emergent correction of hypokalemia and management of thyrotoxic state.

#### CASE REPORT:

A 27 yr old gentleman presented to ER with weakness of all 4 limbs, palpitations since early morning. He denied usage of drugs like diuretics, laxatives. He gave the history of heavy carbohydrate meal in the last night. No history of similar episodes in the past. No family history of periodic paralysis.

#### ON EXAMINATION - He had,

- Exophthalmos,



- Goitre,



- Excessive sweating,
- Tachycardia,
- Wide pulse pressure,
- Quadriplegia with absent Deep tendon reflexes,
- No sensory and cranial nerve deficits were noted.

#### INVESTIGATIONS:

- Serum potassium: 1.7 meq/L (3.5-5 meq/L)
- 24 hr urinary potassium: 42.3 meq/L (25-125 meq/L)
- ABG: Normal, TTKG: <3

- Serum
- T3 : 2.17 ng/ml (0.87-1.78 ng/ml)
- T4 : 13.84 mcg/dl (6.09-12.23 mcg/dl)
- TSH : 0.001 uIU/ml (0.34-5.6 uIU/ml)
- Anti TPO antibodies : Negative.
- Usg neck : Increased size and vascularity of thyroid gland features suggestive of a Thyroiditis ((Hashimoto's).
- ECG : Sinus arrhythmia.
- Thyrotoxicosis was diagnosed and treated with propranolol 40 mg TID, methimazole 10 mg TID. The patient was discharged with the diagnosis of TPP secondary to HASHITOXICOSIS and advised follow up.

#### DISCUSSION:

- Hypokalemia in TPP results from an intracellular shift of potassium not due to total body depletion. (1)
- Hyperthyroidism may result in a hyperadrenergic state which leads to activation of  $\text{Na}^+ / \text{K}^+$  ATPase activity and result in cellular uptake of  $\text{K}^+$ . (2,3,4)
- Patients with TPP have been found to have hyperinsulinemia during episodes of paralysis. This explain the attack after high carbohydrate meals. (4)

#### DIFFERENTIAL DIAGNOSIS :

- **TRANSCELLULAR SHIFT** –  
Familial periodic paralysis  
Drugs : Insulin overdose, Theophylline toxicity.
- **RENAL LOSS OF  $\text{K}^+$**  –  
Drugs : Diuretics  
Renal tubular acidosis,  
Barter's syndrome and Gittleman's syndrome.
- **GASTROINTESTINAL LOSS OF  $\text{K}^+$**  –  
Infectious diarrhoea,  
Tropical sprue,  
Celiac disease.

#### REFERENCES :

1. McFadzean AJ, Yeung R. Periodic paralysis complicating thyrotoxicosis in Chinese. BMJ 1967;1(538):451-455.
2. Lin SH Thyrotoxic periodic paralysis. Mayo Clin Proc 2005;80(1):99-105.
3. Gennari FJ. Hypokalemia. N Engl J Med 1998;339(7):451-458.
4. Levey GS, Klein I. Catecholamine-thyroid hormone interactions and the cardiovascular manifestations of hyperthyroidism. Am J Med 1990;88(6): 642-646
5. Lee KO, Taylor EA, Oh VM, Cheah JS, Aw SE. Hyperinsulinemia in thyrotoxic hypokalemic periodic paralysis. Lancet 1991;337(8749): 1063-1064.