Case Report

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Hypokalaemic Periodic Paralysis in a Patient with Subclinical Hyperthyroidism: A Rare Case

Internal Medicine Section

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ABSTRACT

Thyrotoxic Periodic Paralysis (TPP) is an uncommon disorder. Though many cases of hypokalaemic periodic paralysis are reported in overt hyperthyroidism , hypokalaemic paralysis in subclinical hyperthyroidism is very rare. Subclinical hyperthyroidism is characterised by circulating TSH levels below reference range and normal thyroid hormone levels. We describe a case of 32-year-old Asian male who presented to the emergency department with acute onset weakness and hypokalaemia with no previous history of thyroid disorder or any signs and symptoms suggestive of hyperthyroidism. He was subsequently diagnosed with Graves' disease with subclinical hyperthyroidism.

Keywords: Graves' disease, Low TSH, TPP

CASE REPORT

A 32-year-old Asian man, farmer by occupation presented to the emergency department one evening with sudden onset weakness in all four limbs. His symptoms were sudden, progressive and he was unable to walk or get up from sitting position. He was even unable to lift his hands above shoulder level. Patient gave history of similar complaint involving only the lower limbs 3 months back which recovered spontaneously. He denied use of any medications or alcohol. He had no symptoms suggestive of thyrotoxicosis. He had history of thyroid problem in his mother, the details were not known. On physical examination he was conscious, oriented, afebrile with pulse rate of 90 beats per minute and blood pressure of 120/80 mmHg. He had motor weakness with power 2/5 in the lower limbs proximally and 3/5 distally. In upper limbs his power was 3/5. His cranial nerves and sensory system examinations were normal. Deep tendon reflexes were diminished. There was no bulbar weakness and no sphincter incontinence. There were no signs of hyperthyroidism like tachycardia, tremors, sweaty palms, goitre, exophthalmos, etc.

In the Emergency Department, his initial potassium level was 2.3mEq/l (reference range 3.5 – 5.5 mEq/l) with normal acid base status. His serum magnesium was 1.69mg/dl (reference range 1.5- 2.3 mg/dl), serum creatinine- 0.7mg/dl (reference range 0.5-1.4 mg/dl) and Blood urea nitrogen - 7.7mg/dl (reference range 7- 20mg/dl), spot urinary potassium was 28.12mmol/L (reference range 20-80mmolL) and urinary pH -7.09 (reference range 5.0-9.0). ECG was normal. Thus an initial diagnosis of hypokalaemic periodic paralysis was made and he was started on potassium infusion 20mEq in normal saline and oral potassium supplements.

On the second day his potassium increased to 4.9mEq/l. His power improved to 5/5 in all 4 limbs. His thyroid function test revealed serum TSH of <0.005mU/L (ref. range 0.5- 4.4 mU/L), T3 of 1.25nmol/L (ref. range 1.08-4.14nmol/L) and T4 of 89.76 (ref. range 59-135). As his T3 and T4 were normal fT4 was done which was 12.8pmol/L (12- 22pmol/L). A Technetium 99 thyroid scan was ordered which showed enhanced tracer trapping by a mildly enlarged hyperperfused thyroid gland, suggestive of diffuse hyperplastic goitre (Graves' disease). Nerve conduction study done was normal. His subsequent serum potassium levels were within normal range. Thus a diagnosis of Graves' disease with subclinical hyperthyroidism causing hypokalaemic periodic paralysis was established. He was discharged with Methimazole and Propranalol. On follow up after 3 months his TSH had increased and he had no further episodes of paralysis.

DISCUSSION

Thyrotoxic periodic paralysis is a rare complication of hyperthyroidism and is characterised by abrupt onset of hypokalaemia and weakness. Even though it is commonly seen in Graves' disease, it is not related to aetiology, severity, and duration of thyrotoxicosis [1]. Hypokalaemic paralysis can be the first manifestation of hyperthyroidism. Very few cases of hypokalaemic paralysis have been reported in subclinical hyperthyroidism. In a study by J Kalitha et al., there were two cases of hypokalaemic paralysis with subclinical hyperthyroidism [2]. To the best of our knowledge ours is the third such case reported from India. The other unusual presentations of TPP reported from India [3-8] have been tabulated [Table/Fig-1].

Sl. No	Author	Year	Salient features	Outcome
1	L Nilachandra et al., [3]	2004	2 cases of thyrotoxic periodic paralysis in *females*	Improved with medication
2	Satam N et al., [4]	2007	10-year-old with breathlessness and progressive weakness. Had features of thyrotoxicosis with *normokalaemia*.	Patient died.
3	Balakrishna RK et al., [5]	2011	Case of recurrent flaccid paralysis which was caused by a *silent thyrotoxicosis*.	Recovered
4	Sanyal D et al., [6]	2013	Case of *silent thyrotoxicosis* due to *thyroiditis* presenting as TPP	Recovered
5	Chakrabarti S et al., [7]	2015	Patient with previously untreated Grave's disease developed TPP *following administration of intravenous hydrocortisone* for control of severe anaphylaxis	Improved
6	Chakrabarti S et al., [8]	2015	27-year-old male with newly diagnosed but untreated Grave's disease and TPP who was *normokalaemic* during the acute phase of paralysis.	Improved

[Table/Fig-1]: List of case reports from India of patients of thyrotoxic periodic paralysis with unusual features
*unusual features are in italics

TPP is most commonly seen in Asian men. Though hyperthyroidism is more common in female, the tendency of hypokalaemic paralysis occurring more in men is unexplained. Age of presentation is usually between 20-40 years.

In a previous study by McFadzean and Yeung [1], they found that the manifestations of hyperthyroidism preceded the episodes of paralysis. However, in another case series by S Heong Goh [9] majority of the patients had no clinical signs or symptoms of hyperthyroidism during presentation.

The hypokalaemia that occurs in TPP is not due to potassium depletion but due to transcellular shift of potassium. Thyroid hormone promotes Na+/K+ATPase mediated cellular uptake of K+, leading to hypokalaemia. Insulin, beta2- adrenergic activity and alkalosis also cause hypokalaemia by similar mechanism [10].

Clinical manifestation of a typical attack consists of muscular weakness mostly affecting the proximal muscles of leg. Decreased muscle tone with diminished reflexes is typical. Bulbar paralysis though rare, has been reported in few cases [11,12]. Sensory system, level of consciousness and bowel and bladder are spared. These episodes usually occur in morning or evening hours, precipitated by strenuous exercise or high carbohydrate meal. Other precipitating factors include emotional stress, drugs such as insulin, steroids and diuretics. The paralysis may last few hours to few days. Findings of thyrotoxicosis may or may not be present.

The management includes treatment of acute attack of hypokalaemic paralysis followed by definitive therapy for hyperthyroidism. The acute attack can be managed by oral or intravenous potassium chloride [13]. Care must be taken to avoid overcorrection of potassium. A non-selective beta blocker like propranolol should be used. It acts by blunting the hyper adrenergic stimulation of Na+/K+ ATPase and thus preventing the intracellular shift of potassium [14]. The definitive therapy includes anti thyroid medications, surgical thyroidectomy or radioiodine therapy depending on the aetiology.

CONCLUSION

Physicians must be aware about this disorder for early diagnosis and treatment. Thyrotoxic periodic paralysis should be considered in any patient presenting with acute paralysis and hypokalaemia even in the absence of clinical features suggestive of hyperthyroidism.

REFERENCES

[1] McFadzean AJ, Yeung R. Periodic paralysis complicating thyrotoxicosis in Chinese. *BMJ*. 1967;1(538):451–55.
[2] Kalitha J, Goyal G, Bhoi SK, Chandra S, Mishra UK. Comparative study of thyrotoxic periodic paralysis from idiopathic hypokalaemic periodic paralysis: An experience from India. *Ann Indian Acad Neurol*. 2012;15(3):186–90.
[3] Nilachandra L, Joy Singh AK, Akoijam B, Singh NB. Hypokalaemic Thyrotoxic Periodic Paralysis in Female - A Report of 2 Cases with review. *IJPMR*. 2004; 48-51.
[4] Satam N, More V, Shanbag P, Kalgutkar A. Fatal thyrotoxic periodic paralysis with normokalaemia. *Indian J Pediatr*. 2007;74(11):1041-43.
[5] Balakrishnan RK, Chandran SR, Thirumalnesan G, Doraisamy N. Thyrotoxic periodic paralysis. *Indian Journal of Endocrinology and Metabolism*. 2011;15(Suppl2):S147-49.
[6] Sanyal D, Bhattacharjee S. Thyrotoxic hypokalaemic periodic paralysis as the presenting symptom of silent thyroiditis. *Annals of Indian Academy of Neurology*. 2013;16(2):218-20.
[7] Chakrabarti S. A rare case of thyrotoxic periodic paralysis precipitated by hydrocortisone. *J Nat Sci Biol Med*. 2015;6(1):271-74.
[8] Chakrabarti S. Normokalaemic Thyrotoxic Periodic Paralysis with Preserved Reflexes - A Unique Case Report. *Journal of Clinical and Diagnostic Research*. 2015;9(2):OD05-06.
[9] Heong Goh S. Thyrotoxic periodic paralysis: report of seven patients presenting with weakness in an Asian emergency department. *Emerg Med J*. 2002;19:78-79.
[10] Kasper D, Fauci A, Hauser S, Longo D, Jameson L, Loscalzo J. Harrison's principles of Internal Medicine, 19th edition. New York, Mc Graw Hill: 2015; 304-312.
[11] Mathew B, Devasia AJ, Ayyar V, Thyagaraj V, Francis GA. Thyrotoxicosis presenting as acute bulbar palsy. J Assoc Physicians India. 2011;59:386–87.
[12] Bertola G, Ausenda C, Bocchia M, Grassi F, Ciani M, Sassi L. Bulbar paralysis and flaccid tetraparesis in thyrotoxicosis. *Recenti Prog Med*. 2002;93:169–71.
[13] Lin SH. Thyrotoxic periodic paralysis. *Mayo Clin Proc*. 2005;80(1):99–105.
[14] Yeung RT, Tse TF. Thyrotoxic periodic paralysis: effect of propranolol. *Am J Med*. 1974;57:584-90.

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