Doi: 10.32677/IJCR.2015.v01.i03.004 **Case Report**

Thyrotoxic Hypokalemic Periodic Paralysis – A Case Report

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ABSTRACT

Thyrotoxic hypokalemic periodic paralysis is a medical emergency which presents with muscular weakness, hypokalemia and features of thyrotoxicosis. Urgent treatment is very important; since, very low potassium levels can lead to cardiac complications. Here, we present a case where features of hyperthyroidism were missed on initial assessment and were picked up subsequently in the second presentation of paralysis. This emphasizes the importance in recognizing the subtle features of hyperthyroidism from both history and clinical examination. Acute management consisted of administering intravenous potassium chloride and then treatment of thyrotoxicosis.

Key words: *periodic paralysis, thyrotoxicosis, hypokalemia, emergency department*

Thyrotoxic hypokalemic periodic paralysis (THPP) is an acquired form of periodic paralysis that presents occasionally to the emergency department. It encompasses of hypokalemia, features of thyrotoxicosis and periodic paralysis [1-3]. Immediate correction is very imminent since it can predispose to cardiac arrhythmias, precipitated by low potassium levels. There is a paucity of reports on this disorder from India; therefore, we present such a case of THPP in a 38 year old man [4]. Because of its rare presentation, the emergency physician should be well versed in identifying the condition and treating it, thus preventing complications.

CASE REPORT

A 38-year-old man presented to the emergency department (ED) in the morning with complaints of weakness in both the lower limbs, inability to walk, mild breathing difficulty and lack of sleep. The night prior to presentation, he had consumed alcohol along with a carbohydrate diet and went to bed. Early morning around 2.00 AM, he woke up and observed that he was unable to flex his knees but could roll his lower limbs from side to side. He noticed mild breathing difficulty and he was able to move his upper extremities well. He also gave a history of heat intolerance and loss of weight since the past one month. His wife also told that she has been noticing his eyeballs to be prominent since the last weeks.

There was no history of fever, trauma, family history or any new drug intake. He had recently returned from abroad, where he used to work as a manual laborer. He had a similar history back there. He was taken to the hospital and was told his condition is due to low potassium levels. Correction was given and discharged with oral potassium chloride (KCl) tablets. On reaching his home here in India, noticing improvement and no further deficit, he had stopped taking the KCl tablets since the last few days.

On physical examination, he was conscious, afebrile, anxiously looking and had exophthalmos bilaterally. His pulse rate was 90/min, regular and good volume, respiratory rate was 22 breaths /min and blood pressure was 140/70 mm Hg in the right arm in supine position. Rest of the respiratory and cardiovascular system examinations was normal. On neurological examination,

higher mental functions and cranial nerves were normal. He had fine tremors of both hands with proximal weakness in both the lower limbs and diminished tendon reflexes bilaterally. Powers at all joints in upper limb were 5/5 while it was grade 3/5 at both hip and grade 4/5 at knee and ankle joints. Sensory system and cerebellar functions were normal. Examination of neck revealed diffuse goitre. His laboratory profile at presentation is shown in Table 1.

Table 1 - Results of Initial Laboratory Studies

Laboratory Tests	Results	Normal
pH	7.368	7.35-7.45
pO_2(mmHg)	79.5	83.0-108.0
pCO_2 (mmHg)	38.0	35.0-48.0
$cHCO_3^-$ (mmol/L)	21.9	21.0-28.0
Sodium (mmol/L)	141	138-146
Potassium (mmol/L)	2.1	3.5-4.5
Calcium (mmol/L)	1.3	1.15-1.33
Hematocrit (%)	42	38-51
Hemoglobin (g/dL)	14.3	12-17
Lactate (mmol/L)	1.21	0.56-1.39
WBC Count (cells/mm^3)	10300	4000-10000
Platelets (lakhs/cumm)	3.1	1-3

Spot urine potassium was 9 mmol/L (a low urine potassium level is <20 mmol/L). Thyroid profile was suggestive of hyperthyroidism i.e. free T3 - 11.5 pmol/L (normal 4-8.3 pmol/L), free T4 - 24.7 pmol/L (normal 9-20 pmol/L) and TSH - 0.03 µIU/ml (normal 0.2-5 µIU/ml). X-ray chest was normal. Ultrasonography of thyroid revealed enlarged thyroid gland with mild increase in color flow which could represent toxic goitre. ECG showed flattened T waves in lateral leads. In view of documented hypokalemia, lower limb weakness and features of thyrotoxicosis, a diagnosis of thyrotoxic hypokalemic periodic paralysis was made.

The patient was started on intravenous potassium chloride (KCl) and he improved dramatically in the ED itself. The patient was then shifted to the ward, where repeat blood test showed improved potassium level to 4.26 mmol/L (normal value – 3.5 to 4.5 mmol/L). He was also started on Propranolol and Carbimazole. With improvement in symptoms, he was discharged after three days on Propranolol and Carbimazole to review after two weeks.

DISCUSSION

Thyrotoxic hypokalemic periodic paralysis is a rare but potentially lethal manifestation of hyperthyroidism which is characterized by muscular weakness due to intracellular shift of potassium and subsequent hypokalemia [1]. Patients with this type of paralysis have an inherent defect in Na^+-K^+ ATPase activity which is sensitive to thyroid hormone. The thyroid hormone alters the cell membrane permeability to potassium through Na^+-K^+ ATPase pump leading to intracellular shift of potassium resulting in hypokalemic periodic paralysis without depleting the total body K^+ (potassium) stores [2].

THPP is more commonly reported in Oriental Asians in the age group of 20-40 years. Paralysis occurs only when the patient is at rest, and usually in bed at night. The attacks of paralysis vary widely in severity and range from weakness of the muscles of the pelvic girdle, lasting several hours, to a total paralysis of all the muscles from the neck downward, lasting up to 48 hours [3]. Proximal muscles are affected more severely than distal muscles [3].

Various precipitating factors include trauma, infection, emotional upset, menstruation, epinephrine, thyroid hormone, steroids, carbohydrate intake and unaccustomed exercise [5]. The majority of cases of hyperthyroidism associated with THPP are due to Grave's disease although other conditions including thyroiditis, toxic multi-nodular goitre, toxic adenoma, TSH secreting pituitary tumor, ingestion of T4 and inadvertent iodine excess have also been implicated [6].

Lin et al advocated the use of spot urine for K^+ excretion rate and evaluation of blood acid-base status in the diagnosis and management. A very low rate of K^+ excretion coupled with the absence of a metabolic acid-base disorder suggests HPP, whereas a high rate of K^+ excretion accompanied by either metabolic alkalosis or metabolic acidosis favors non-HPP. The presence of features of hyperthyroidism along with low potassium excretion and normal acid base status points towards THPP [7].

During periodic paralysis and marked hypokalemia, immediate supplementation with KCl is warranted to prevent major cardiac complications. KCl is given slow IV or orally or both. While treating with KCl, it should be kept in mind that the hypokalemia seen in THPP is due to

sudden intracellular shift of K^+ and not due to K^+ deficiency. Therefore, clinicians should exercise caution when administering high-dose K^+ supplementation as rebound hyperkalemia can also occur [8].

Oral or IV propranolol, a nonspecific-adrenergic blocker, has also been proposed as an alternative treatment to ameliorate symptoms. The common recommendation is that KCl supplementation dose should be <10 mEq/hr with close monitoring of serum potassium levels to avoid rebound hyperkalemia [9]. Use of potassium supplements is not useful for prophylaxis against further paralytic attacks and should not be given to patients between attacks. Because THPP does not recur once the patient is euthyroid, adequate control of hyperthyroidism is the mainstay of therapy. The cause for the hyperthyroidism should also be identified [10].

CONCLUSION

The diagnosis of THPP is often delayed partially due to the subtle features of thyrotoxicosis and partially due to unawareness of this condition because of its relative rarity. Hypokalemia with paralysis has got many differential diagnoses. Excluding family history and including features of hyperthyroidism and also checking for low urine potassium excretion will give a definite diagnosis of THPP. The aim of treatment is to correct hypokalemia and later to achieve a euthyroid state.

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