*Case Report*

# Thyrotoxic Periodic Paralysis: A Rare Presentation of Muscle Weakness

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**Abstract:**
*Thyrotoxic periodic Paralysis is a rare metabolic disorder that is seen mostly in men of the Asian origin. Here we reported a case of 35 years old female patient who had presented with the complaints of sudden onset of weakness of both lower limbs. Hypokalemia was identified during investigation and treated with resolution of symptoms accordingly. Additional investigation identified that the patient is being thyrotoxic. So, it is important to consider the diagnosis of both hypokalemia and thyrotoxicosis simultaneously when a patient is presented with acute onset of the muscle weakness. In this case of the female patient, therapy was started according to the diagnosis and she was responded well to the treatment and recovered soon.*

**Keywords:** Thyrotoxic periodic paralysis, Hypokalemic paralysis, Sudden limb weakness

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**Introduction:**
Hypokalemic periodic paralysis is one of the rare complications of hyperthyroidism[1]. It has been described in different racial and ethnic groups, mostly in young Asian males[2]. Thyrotoxic Periodic Paralysis (TPP) can be defined as a disorder that is characterized by concurrent presentation of thyrotoxicosis, hypokalemia and progressive symmetrical weakness leading to paresis or paralysis of extremities and other muscle groups[1,2]. Hypokalemia is defined as a deficiency of plasma potassium below 3.5 mEq/L. Homeostasis of this cation is tightly regulated and achieved mainly via alteration in renal excretion[3,4].

Although the incidence of TPP in Asian countries (2%) is approximately 10-20 times higher than the incidence in non-Asian ethnic population (0.1%-0.2%), TPP is increasingly reported in western countries because of globalization and immigration[4]. The pathogenesis of TPP has long been thought related to increased $Na^+$-$K^+$ ATPase activity stimulated by thyroid hormone and/ or hyper adrenergic activity & hyperinsulinemia[5]. Although it is relatively uncommon, yet potentially life-threatening condition because of development of cardiac arrhythmias and arrest[6].

Here we reported a case of TPP in Bangladeshi female predominantly presented as acute quadriparesis without impairing brain function and sensory loss.

**Case report:**
A 25 years old lady rushed into the emergency department with the complaints of bilateral lower limb weakness which developed over last 1 day. According to her statement she was relatively well 2 days back. Initially she developed cramping pain involving both lower limbs after a heavy carbohydrate meal. Within next morning she developed difficulty in walking.

After primary management in emergency department she was sent to Medicine ward where she was evaluated further. She had no H/O trauma, drugs, vomiting, diarrhoea, fever preceding this event. She experienced such weakness two years back. Within next couple of hours she developed quadriparesis. After doing neurological examination it was found that she was alert, oriented and all cranial nerves were intact. There was quadriparesis and muscle power in all four limbs was 2/5 without affecting sensory function and bowel & bladder involvement. Pulse, blood pressure and other vital signs were normal.

Other relevant examination revealed that there was diffuse enlargement of thyroid gland and borderline exophthalmos. All routine investigations including serum electrolytes and ECG were sent immediately. Her serum $K^+$ level was 2.3 mEq/L (hypokalemia) and there was U-wave and flattened T-wave in chest leads associated with atrial fibrillation in ECG. Later on thyroid function test including FT3, FT4, TSH and thyroid antibody were done which was

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suggestive of primary thyrotoxicosis. She was given intravenous potassium as well as oral potassium supplement and oral anti-thyroid drug was introduced from next day. Her condition improved accordingly and her biochemical reports became normal within next two days. After the management she was diagnosed as a case of Thyrotoxic Periodic Paralysis (TPP).

**Discussion:**

Thyrotoxic periodic paralysis is although rare, but important cause of hypokalemic paralysis (HP). HP is a condition characterized by muscle weakness associated with changes in potassium level. HP can occur either due to transient shift (Hypokalemic periodic paralysis or HPP) or reduction in absolute potassium levels[7].

Familial causes are inherited in an autosomal dominant manner. Mutation in CACNA1S and SCN4 gene adversely affect the function of calcium and sodium ion channels serving muscle cells respectively[8]. Young oriental men are more likely to be affected with a prevalence of around 2% of all Thyrotoxic case compared to 0.2% in Thyrotoxic Caucasian patients[9]. However this condition can affect individuals from different parts of the world[9,10].

Our case was Bangladeshi middle aged female but most of the case reports were Asian young males. So our case report contradicts with their reports. This may be due to less reported case in this area.

Most of the cases are typically transient and last hours to days and may be triggered by number of conditions. Neurological symptoms develop within couple of hours sparing the central nervous system (CNS). Our case had the similar attack. Our patient was alert and oriented and trigger factor was rich Carbohydrate diet although Thyrotoxicosis was found later on.

Any weakness is common yet non-specific presentation of various neurological and non-neurological conditions in which hypokalemic periodic paralysis is an important cause of acute flaccid paralysis. There may be clinical differential diagnosis like Guillain-Barre syndrome, Acute transverse myelitis, Poliomyelitis. These neurological diseases should be ruled out first by doing extensive neurological examinations.

The underlying hyperthyroid state has to be addressed in order to definitively rectify the condition, however initially potassium replacement may be needed to hasten muscle recovery and prevent cardiac complications[11].

The $Na^+$-$K^+$ ATPase pump located on the sarcolemma of muscle cells which prevent intracellular accumulation of sodium. It pumps out 3 $Na^+$ for every 2 $K^+$ ions into the cell. The influx of $K^+$ into the cell alters the resting potential of the muscle cell having unable to depolarize because it is already hyperpolarized. The pump is activated by catacholamines of susceptible patients and the Thyroid hormones also sensitize the circulatory catacholamines. Thyroid hormones can also directly stimulate the $Na^+$-$K^+$ ATPase enzyme activity and thyrotoxicosis has been shown to increase pump number[11,12].

In one study among Asian people Mohapatra *et al*. found that hypokalemic periodic paralysis is more common in young Asian male patient without any family history. These cases mostly occurred in summer and after waking up from the sleep[13].

Paiboonpol S and Neki NS in their case series found that TPP was the common cause for flaccid paralysis[14,15]. Our case report was also related with their findings but for genetic conformation we could not identify which channelopathies are responsible in our limited hospital settings.

**Conclusion:**

Although rare but a clinician should have higher degree of clinical suspicion when a young adult presented with flaccid paralysis. Failure to quickly identify the diagnosis as TPP can be fatal, but efficient correction of potassium as well as the hyperthyroid state can resolve symptoms rapidly to prevent complication.

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