An Unusual Presentation of Thyrotoxicosis

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Abstract

Thyrotoxic periodic paralysis (TPP) is a rare complication of thyrotoxicosis, that constitutes an endocrine emergency. Periodic paralysis without a familial history in a patient with hyperthyroidism is a strong clinical clue to the diagnosis, especially when accompanied by concomitant hypokalemia. We present a case of 28 year old male admitted with recurrent history of weakness of all four limbs with hypokalemia which turned out to be a thyrotoxicosis induced hypokalemic periodic paralysis.

Key Words

Periodic paralysis, Thyrotoxicosis, Hypokalemia.

Case History

28 year old male admitted with history of weakness of all four limbs of one day duration. He noticed weakness, while getting up from bed in the morning, associated with retention of urine. History of muscle cramps was present in the previous night. No history suggestive of cranial nerves, sensory or cerebellar involvement.

There was no preceding history of trauma, fever, or recent vaccination. He had similar two episodes in the recent past which recovered spontaneously within a day. No history of similar illness in the family.

On examination there was flaccid quadriparesis with predominant proximal muscle involvement, power was 2/5 in both upper and lower limbs.

Deep tendon reflexes were diminished, plantar was flexor response.

Proceeded with provisional diagnosis of periodic paralysis. Routine blood investigations were within normal limits. Serum electrolytes report was K⁺ -2.3 meq/l, Na⁺ -134meq/l, serum calcium was 8.2mg/dl. So diagnosis of hypokalemic periodic paralysis probably of secondary causes was made.

Next step was to find out the cause of hypokalemia. From the history, gastro intestinal loss and chronic diuretic use was ruled out. Urine electrolytes were done to rule out renal tubular acidosis, which came as normal. Ultrasound abdomen, chest xray were within normal limit, ECG showed sinus tachycardia.

Even though there were clinically no features of thyrotoxicosis except for tachycardia we proceeded with a thyroid function test which shown TSH of 0.02 µIU/L, total T4 68µg/L, total T3 700ng/L. So diagnosis of hypokalemic periodic paralysis secondary to thyrotoxicosis was made.

Patient was treated with oral potassium about 60 meq over 24 hrs and recovered very well next day without residual paralysis. and on discharge serum k⁺ was 3.8 meq/l. He was started on

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carbimazole 10 mg tds and propranolol 20 mg bd for the thyrotoxicosis.

**Discussion**

Periodic paralysis associated with the thyrotoxic state is a rare and peculiar disorder that affects only skeletal muscles. The degree of paralysis varies from one attack to another. Severe episodes can cause paralysis of all skeletal muscles, including those controlling respiration. Smooth muscles are seldom affected, and cardiovascular dysfunction is not observed in this disorder. The cardinal feature of a typical attack is muscle weakness, usually symmetrical paralysis beginning in the proximal muscles of the legs. Hypokalemia (potassium level <3.0 mmol/L) associated with hyperthyroidism is also common.

A typical attack of thyrotoxic periodic paralysis lasts from a few hours to several days. Most attacks occur in the morning or evening. In our patient, the first two attacks lasted for about few hours each and weakness recovered spontaneously but the current attack lasted about 24 hours and recovered with oral potassium treatment. All the three attacks occurred in the early morning hours. In contrast to familial periodic paralysis, in which the first attack occurs before the age of 10 years, patients with thyrotoxic periodic paralysis are usually aged 20 to 40 years.

Asians are disproportionately affected by this condition, with one study showing Asians were at 159-fold higher risk compared with white Europeans. There is an overwhelming male predominance, with a male-to-female ratio of 77:20. The condition has been reported worldwide, including in the United Kingdom, the United States (in whites and African Americans), and South America. A low serum potassium level has been noted in the majority of patients with thyrotoxic periodic paralysis. Thyrotoxicosis is associated with normal regulation of body sodium, potassium, and cell volume. Administration of thyroid hormone has been shown to increase the fractional excretion of potassium. Compared with other body tissues, human renal tissue contains a higher concentration of messenger RNA for the thyroid receptors alpha-1, alpha-2, and beta. An alteration of thyroid-receptor expression may be an important mechanism that controls the tissue effects of the hormone, as the thyroid hormone levels in the serum increase.

Asian and Hispanic patients with thyrotoxicosis are particularly susceptible to periodic paralysis. It is also important to remember that although hypokalemia is common during the acute paralytic episode, between attacks plasma potassium and total body potassium stores remain within normal limits. Precipitating factors of hypokalemic thyrotoxic periodic paralysis include strenuous exercise followed by rest, excessive ingestion of carbohydrate-rich food, administration of insulin or epinephrine, trauma, exposure to cold temperatures, infection, menstruation, and emotional stress.

Definitive treatment of thyrotoxic periodic paralysis consists of the management of thyrotoxicosis, medical therapy, surgery or radioactive iodine therapy. Treatment for an acute attack is potassium administration, but excessive doses of potassium can lead to hyperkalemia once potassium shifts to the extracellular space. No correlation between potassium dose administered and recovery time was observed. To prevent attacks until euthyroid state is achieved, a useful therapy is the administration of a ß-adrenergic blocker like propranolol. Other preventive measures that may be effective include a low-carbohydrate diet and potassium-sparing diuretics. Use of potassium supplements is not useful for prophylaxis against further paralytic attacks, and it should not be given to patients between episodes.
Case Report

Summary

Thyrotoxic periodic paralysis is a rare complication of thyrotoxicosis that must be regarded as an endocrine emergency. Clinical clues to this condition include a presentation of periodic paralysis without a family history, along with a history of hyperthyroidism. Episodes of periodic paralysis usually precede the diagnosis of thyroid dysfunction and do not recur once euthyroidism is achieved. Therefore, it is necessary that an early diagnosis of TPP is made to administer definitive treatment and prevent morbidity and mortality, mainly due to fatal arrhythmias. The presence of acute paralysis, especially with hypokalemia, should prompt the clinician to consider TPP as a cause and evaluate thyroid function.

References