

Thyrotoxic Hypokalaemic Periodic Paralysis, a Rare Cause of Acute Muscle Weakness

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Thyrotoxic periodic paralysis is a rare neuromuscular disorder related to a defect in muscle ion channels and manifest by episodes of painless muscle weakness. Periodic paralysis is classified as hypokalaemic when episodes occur in association with low potassium levels or as hyperkalaemic when episodes can be induced by elevated potassium. Thyrotoxic hypokalaemic periodic paralysis may present as a life threatening emergency and unfamiliarity with the syndrome could result in a fatal outcome. Compliance with therapeutic management plays an important part in the treatment of this condition. Although the incidence is higher in the Asian population, it has been reported in other ethnic groups. This case report is of an Omani male presented to Buraimi hospital with acute muscle weakness.

A 36 year old Omani male presented to Buraimi hospital on 14.08.2008 with history of sudden onset of lower limb weakness and inability to walk after he woke up from sleep on the same day. There was no history of trauma, fever, diarrhoea, weight loss, heat intolerance or palpitations. There was no significant past illness or family history. On examination his pulse was 80/mt, blood pressure 140/80. Neurological examination revealed reduced muscle tone in lower limbs with 3/5 power at the hip and 4/5 power distally. Power in the upper limb was 4/5 proximally and 5/5 distally. He had reduced deep tendon reflexes with no sensory loss and the cranial nerves were intact. Systemic examination was normal without any signs and symptoms suggestive of thyrotoxicosis.

The laboratory studies revealed potassium of 2.6 mmol/litre with sodium 141 mmol/litre and chloride 103.3 mmol/litre. Creatinine was 30 umol/litre and urea 41 mmol/litre. Haemoglobin was 11.2 gram/deciliter and ESR 10 mm/hour. Thyroid function test showed Free T4 -60.4 pmol/litre and TSH-<0.005 mIU/litre. Liver function test was normal and urinary spot potassium was 67.0 mmol/litre. Blood sugar was normal. Other biochemical investigations were unremarkable. ECG showed normal sinus rhythm.

The patient was admitted and treated with intravenous potassium and oral potassium supplements. Once his serum potassium normalized, his symptoms resolved. He was discharged on complete resolution of symptoms. He was started on carbimazole, propranolol and oral potassium and advised regarding follow up and compliance.

Thyrotoxic hypokalaemic periodic paralysis (ThPP) is a rare complication of thyrotoxicosis, more common in Asians between second and fourth decades of life. However it has been reported in other ethnic groups as well. Most cases tend to occur in men, despite the higher incidence of hyperthyroidism in women. A high carbohydrate meal, warm weather, increased physical exertion, insulin, adrenalin and potassium sparing diuretics are usual precipitants. The later age of onset is helpful to distinguish thyrotoxic periodic paralysis from the familial periodic paralysis which presents at a younger age.

The flaccid paralysis more prominent in the lower extremities than in the upper limbs and is usually bilateral. Proximal muscles are affected more often than distal muscle groups. Decreased muscle tone with hyporeflexia or are flexia is typical. Mild tachycardia is common. Rarely cases of respiratory and bulbar muscle weakness have been reported in ThPP, as well as cases of severe arrhythmias, including sinus arrest and ventricular fibrillation. Although attacks of weakness may occur at any time of the day, high frequency of attacks at night or early in the morning has been reported in Thyrotoxic hypokalaemic periodic paralysis (ThPP). Attacks vary in frequency and duration. Intervals of weeks to months are common, but some patients experience several attacks per week. Duration of several hours is typical, but can range from minutes to days.

The primary defect in ThPP is an intracellular sequestration of potassium with normal potassium stores in the body. The main biochemical abnormalities during the paralysis are high levels of thyroid hormones and hypokalaemia. Thyroid hormone changes the plasma membrane permeability to potassium by increasing the Na/K ATP ase activity. There is an increase in the beta adrenergic receptors in the skeletal muscles, which increases the Na/K ATP ase activity. These factors along with the insulin and testosterone increase the intracellular shift of potassium. Electron microscopic section reveal a dilatation of sarcoplasmic reticulum. In contrast in familial periodic paralysis the intracellular shift of potassium is Na/K ATP ase independent.

The acute attack needs to be distinguished from other causes of acute quadriplegia, such as myasthenic crisis, Guillain-Barre syndrome, acute myelopathy etc. The finding of hypokalaemia generally alerts the clinician to the diagnosis of periodic paralysis.

A higher index of suspicion should be exercised when there is a symmetrical muscle weakness affecting the proximal muscles more than the distal.

Treatment of ThPP requires urgent correction of potassium levels; however rebound hyperkalaemia can be problematic. Patients with potassium concentrations below 2.5 mmol/litre or with symptoms of paralysis should be treated with intravenous potassium. Patients with potassium level above 2.5 mmol/litre may be treated with oral potassium, as aggressive therapy may cause rebound hyperkalaemia.

Propranolol is found to be useful to reverse paralysis, hypokalaemia, and hypo phosphataemia. Propranolol down regulates the Na/K ATP ase activity.

The definitive therapy of ThPP is the achievement of euthyroid state by medical, surgery or radioactive iodine therapy. The resolution of periodic paralysis occurs with the restoration of euthyroidism. After initiating the definite treatment for thyrotoxicosis, patient should be advised to avoid the known precipitating factors.

Thyrotoxic hypokalaemic periodic paralysis should be considered as one of the differential diagnosis of patients presenting with muscle weakness soon after awakening. Hypokalaemia is the most constant electrolyte abnormality. Although the pathogenesis of this condition is still not clear, there is strong evidence that complete resolution occurs following successful treatment of thyroid dysfunction, which is the definitive treatment. Propranolol and spironolactone can be used to prevent the paralytic attacks. Oral potassium is not effective in preventing the relapse. Potassium is useful mainly to hasten the recovery and prevent cardiac arrhythmias during the attack.

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