INTRODUCTION

Thyrotoxic periodic paralysis is a sporadic form of periodic paralysis which can occur in association with thyrotoxicosis, hence it should be differentiated from familial causes which have an autosomal dominant inheritance. The condition is more common in males, and the incidence is highest in Asian population. It manifests as episodes of neuromuscular weakness which can be precipitated by heavy exercise or carbohydrate rich meal.

This report describes the uncommon condition.

CASE REPORT

A fit and well gentleman of Asian origin aged 36 years presented to the medical admission unit with a few days history of progressive painless leg weakness making him bedbound over the last one day. He was known to have Graves disease which was treated medically about 5 years ago and had been in remission. There was no history of any other neuromuscular symptoms, preceding illness, backache or incontinence. There was no family history of a similar presentation. Clinical examination showed paraparesis with power of 1/5 in the proximal and 2/5 in the distal muscles of both legs. Reflexes and sensations were intact, coordination and gait could not be assessed due to severe weakness. Neurological examination of arms and cranial nerves was normal and there were no signs of respiratory compromise. Clinically, patient appeared euthyroid.

Routine biochemistry results were within the reference range apart from marked hypokalemia (2.5 mmol/L).

Potassium was replaced intravenously with cardiac monitoring and patient made a swift recovery. He was mobilising independently after 12 hours of being admitted to hospital and was reviewed by neurology and endocrine team. Propranolol and propylthiouracil were commenced to treat his hyperthyroidism and prevent further episodes of periodic paralysis with a clinical diagnosis of thyrotoxic periodic paralysis. He remains euthyroid on treatment with no further episodes of paralysis.

DISCUSSION

In contrast to other forms of thyroid disease, which are more common in females, thyrotoxic periodic paralysis is commoner in males. Thyroid hormone increases tissue responsiveness to beta-adrenergic stimulation, which increases sodium-potassium ATPase activity in the skeletal muscle membrane. This tends to drive potassium into cells, perhaps leading to hyperpolarization of the muscle membrane and relative inexcitability of the muscle fibers. In this way, excess thyroid hormone may predispose to paralytic episodes by increasing the susceptibility to the hypokalemic action of epinephrine or insulin.

Patients by definition, have attacks in the hyperthyroid state. During an acute attack, it must be distinguished from other common causes of acute quadriparesis, such as myasthenic crisis, Guillain-Barré syndrome, acute myelopathy and botulism.
Attacks of weakness occur with generalized weakness and preserved consciousness. Thyrotoxic symptoms usually precede the onset of paralysis. Attacks vary in frequency and duration. Intervals of weeks to months are common. Duration of symptoms can range from minutes to days. Attacks can be precipitated by events that are associated with an increased release of epinephrine or insulin. Most commonly, the precipitating factor is strenuous physical activity, stress, or a high-carbohydrate load. In most instances, no obvious precipitant is identified.

Neurologic examination during an attack demonstrates weakness, usually affecting proximal more than distal muscles, and the legs more than the arms. Decreased muscle tone with hyporeflexia or areflexia is typical. Rarely bulbar and respiratory weakness requiring ventilatory support can occur. Hypokalemia can cause heart block and ventricular arrhythmias.

Usually, the severity of weakness corresponds to the degree of hypokalemia. Other common laboratory findings include mild hypophosphataemia and hypomagnesaemia. Creatine kinase may be normal but has been reported to be mildly elevated in two-thirds of patients. Rhabdomyolysis can occur. Electrocardiogram may show hypokalemic changes, blocks and arrhythmias. Diagnosis is clinical although electromyography, provocative testing, and muscle biopsy can aid in diagnosis.

Potassium supplementation leads to complete improvement of weakness. Propranolol presumably reverses excessive drive of potassium into cells and has also been shown to decrease the frequency and severity of attacks. It may be used to prevent further attacks until the patient is rendered euthyroid with thionamides. Precipitating factors should be avoided.

The finding of hypokalemia in a patient presenting with neuromuscular paralysis should alert the clinician to the diagnosis of periodic paralysis, in which the possibility of thyrotoxicosis must always be evaluated.

REFERENCES