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Severe lower limb weakness and thyrotoxicosis: A case of periodic thyrotoxic paralysis

Ritienne Debono; David Coppini; Sarah Craus; Deborah Balzan; Jack Vella*

Department of Medicine, Mater Dei Hospital, Msida, Malta.

***Corresponding Author: Jack Vella**
Department of Medicine, Mater Dei Hospital, Msida, Malta.
Email: jack.vella@gov.mt

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Abstract

Introduction: Thyrotoxic periodic paralysis is an uncommon clinical entity which may present with dramatic, sudden onset of neuromuscular weakness in the absence of any previous known neurological, musculoskeletal, or metabolic disease. This report presents a case of rapid onset paraplegia, found to be due to severe thyrotoxicosis, and also aims to explain the known pathophysiology of the disorder and make recommendations as to it's identification and treatment.

Case report: We report the case of a 39 year old Albanian man who presented to the Emergency Department with severe lower limb weakness and gait instability. Investigations revealed a profound hypokalaemia and thyrotoxicosis; his symptoms resolved upon correction of his hypokalaemia. He is now controlled on carbimazole with no recurrence of symptoms.

Conclusion: Thyroid function tests should be taken in cases of unexplained weakness and hypokalaemia. Correction of thyroid function, and not just hypokalaemia, is necessary to prevent a recurrence of symptoms.

Keywords: Thyroid; Thyrotoxicosis; Hypokalaemia; Paraplegia; Periodic paralysis.

Introduction/background

Acute lower limb paralysis is a distressing clinical entity which most commonly occurs secondary to spinal cord pathology. Metabolic causes, such as Thyrotoxic Periodic Paralysis (TPP) ought to be considered, particularly once urgent neurosurgical causes have been ruled out. Here, we present a case of a previously healthy 39 year old, who woke up unable to move his legs. He was found to be profoundly hypokalaemic, secondary to biochemical thyrotoxicosis. This occurred despite the absence of any symptoms of hyperthyroidism. Rapid but controlled correction of his hypokalaemia, and control of his underlying Graves' disease, brought around total remission of his symptoms with no residual deficit.

Case description

A 39 year old Caucasian Albanian male presented to the Emergency Department with acute onset lower limb weakness and gait instability, which started on waking up. The patient denied any problems with his upper limbs, had no history of low back pain, sphincter disturbance, or sensory disturbance. He claimed to have had two episodes of loose stools the day prior to presentation, but denied nausea or vomiting, or febrile episodes. The patient was on carbimazole 10 mg daily, which his general practitioner had initiated back in Albania few weeks before after presenting with unintentional weight loss and having been reportedly found to be hyperthyroid. The patient was a smoker, and had no relevant surgical or family history. At his initial as-

sessment, he was clinically stable, with no haemodynamic compromise. Cardiovascular, respiratory and abdominal systems were unremarkable on examination. Neurological examination revealed reduced proximal power proximally in the lower limbs, with symmetrical weakness (⅗ on MRC Power Scale) in the iliopsoas, quadriceps, and hamstring muscle groups. Sensation and tone were preserved. Cranial nerves' examination was normal throughout. Urgent blood tests and venous blood gases were sent for analysis (Table 1). These found the patient to be severely hypokalaemic, with a potassium (K^+) level of 2.1 mmol/L. A provisional diagnosis of hypokalaemia related muscle paralysis was made, and the patient was started on standard potassium replacement regimes with combined Intravenous (IV) and oral replacement. This consisted of intravenous administration of 40 mmol of 15% potassium chloride, diluted in 1 litre of 0.9% normal saline, administered at 160 mls/hour. He was also given oral potassium supplementation (20 mmol every 8 hours). The patient improved considerably once potassium replacement was commenced and weakness resolved completely within a few hours, once potassium levels normalised. No clear cause for the hypokalaemia was at first apparent. The patient had only had one low volume bout of loose stools the day before presentation, was on a healthy diet, and had no medications that could precipitate hypokalaemia. Urinary electrolytes showed no renal potassium loss. He had an elevated serum creatinine kinase (517 U/l, reference 39-308 U/l) with a normal aldolase. His morning serum cortisol was also within range. Further testing including thyroid function tests showed that the patient was biochemically hyperthyroid (Table 2) despite being compliant to Anti-Thyroid Drugs (ATD). He was reviewed an endocrinology team, who doubled the carbimazole dose. The history, presence of biochemically severe hyperthyroidism, and lack of other causes for hypokalaemia was consistent with a diagnosis of Thyrotoxic Periodic Paralysis (TPP) was put forward. TSH receptor antibodies were strongly positive at 33.7IU/L (reference 0.1-1.0 IU/L), suggesting autoimmune Graves' disease as an underlying pathology. The patient was discharged after one further night of observation, and is being regularly followed up at Endocrine out-patients. He remains well and has no residual weakness. Biochemically, his thyroid function tests have also improved on a daily dose of 20 mg carbimazole.

Table 1: Venous blood gases.

		Reference range
pH	7.32	7.35-7.45
Na^+	146	135-145 mmol/L
K^+	2.1	3.5-5.1 mmol/L
Lactate	1.5 mmol/L	
Glucose	6.1 mmol/L	
HCO_3^+	22.1	18-22 mmol/L

Table 2: Thyroid function tests.

	At time of diagnosis	At presentation	Reference range
TSH		<0.008	
Free T4	190	40.59	45-117 ng/ml
Free T3	8.43	17.62	0.69-2.15 ng/ml

Discussion

TPP most commonly affects adolescent males, with an incidence of around 2% in the Asian population [1]. The characteristic features include thyrotoxicosis (and related symptoms such as weight loss), acute painless paralysis and hypokalaemia. Attacks commonly initially involve the lower limbs, without sensory involvement [2], with the severity of weakness being correlated to the degree of hypokalaemia. Most cases of TPP are found in patients with Graves' disease. Other associated conditions include thyroiditis and toxic multinodular goitre. The exact mechanism of TPP is still not well understood. Skeletal muscles contain a large amount of potassium and are crucial in maintaining extracellular potassium homeostasis through sodium-potassium-adenosine triphosphate (Na/K ATPase) pumps (which regulate intracellular potassium shifts) and potassium channels which shift K^+ extracellularly [3]. It is thought that thyroid hormones sensitise the Na/K-ATPase pump activity in skeletal muscle, liver and kidney via both transcriptional and post-transcriptional methods [4,5]. This translates in an increase in the genetic transcription of genes coding of the Na/K^+ pump, augmenting its intrinsic activity. Enhanced sympathomimetic and beta-2 adrenergic stimulation also potentiates the hypokalaemic effects of adrenaline and insulin, which explains why triggering factors include stress, exercise or large meals [4,5]. The goals of treatment are rapid, but safe, normalisation of potassium. Any underlying cause should ideally be identified and treated. Rebound hyperkalaemia may occur if potassium is supplemented hastily. Patients should have cardiac monitoring whilst potassium is being supplemented IV, at a dose that usually varies between 40-200 mmol. Oral or IV beta-adrenergic blockers such as propranolol can be beneficial. Anti-thyroid drugs are useful in the setting of Graves' disease. Radioactive iodine or surgery might be required in some scenarios. Patients should avoid precipitating factors such as intensive exercise or heavy carbohydrate meals.

Conclusion

TPP is a rare condition characterised by acute painless lower limb paralysis in patients with thyrotoxicosis. Patients should be educated about precipitating causes such as strenuous activity and high carbohydrate meals, since these lead to a rise in epinephrine levels and resultant hypokalaemia through shift of K into the cells. Early identification of TPP is crucial, since hypokalaemia may be life-threatening. The mainstay of treatment involves identification and treatment of the underlying cause, with urgent K^+ supplementation to avoid arrhythmias, with possible addition of IV beta blockers.

We suggest the following take-away points:

1. TPP represents a rare metabolic cause for acute flaccid paralysis.

2. The mainstay of treatment involves identification and treatment of the underlying cause, with urgent K+ supplementation to avoid arrhythmias, with possible addition beta-blockers.

3. Patients should be educated about precipitating causes such as strenuous activity and high carbohydrate meals, since these lead to a rise in epinephrine levels and resultant hypokalaemia through shift of K^+ into cells.

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