

THYROTOXIC PERIODIC PARALYSIS AFTER ADMINISTRATION OF METHYL PREDNISOLONE

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Case report

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Abstract

Thyrotoxic periodic paralysis (TPP) is a rare complication of thyrotoxicosis. Common precipitating factors include high carbohydrate diet, alcohol ingestion, strenuous exercise followed by rest, emotional stress, and treatment with insulin or acetazolamide. There are not much reports of steroid treatment precipitating TPP. In this particular patient TPP was manifested after treatment with long acting methyl prednisolone for symptomatic Graves ophthalmopathy. Diagnosis was made after ECG and laboratory confirmation of hypokalemia and was treated with potassium supplementation upon which he showed full recovery. This case is reported for the awareness about the possibility of precipitation of TPP while treating Graves's ophthalmopathy with high dose steroid pulse therapy.

Case summary

55 year old man presented with features of thyrotoxicosis of six months duration and the biochemistry showed elevated Free T3 {6.02 pg/ml (1.7-4.2)} and Free T4 {2.61 ng/dl (0.7-1.8)} along with suppressed TSH {< 0.01 μ IU/mL (0.3-5.5)}. He also had features of Grave's ophthalmopathy with Class 3 of NOSPECS classification by American Thyroid Society. He was counseled on modalities of treatment options available to him and started on Methimazole 20 mg/day along with propranolol 40 mg /day. He was reviewed 2 months later with relief in symptoms, reduction in Free T3 and Free T4 {3.8pg/ml, 1.9ng/dl respectively} and TSH < 0.01 μ IU/mL. He showed concerns over his symptomatic ophthalmopathy and wanted treatment for it. It was decided to administer high dose intravenous steroid pulse therapy initially and to be followed by oral steroid therapy. He was given injection methyl prednisolone 1 gram in 100 ml of normal saline intravenously over one hour. He was stable after the injection for about 8 hours after which he

developed acute onset flaccid paralysis worse in the lower limbs; the rest of the clinical examination was unremarkable. Laboratory results showed serum potassium of 2.1 (N 3.5-4.5 Meq/L) and the ECG revealed signs of hypokalemia (Fig 1). He was treated with potassium supplementation initially intravenous followed by oral upon which patient recovered completely. Repeat serum potassium was 4.8 and the ECG changes reversed to normality the next day (Fig 2). Thus a diagnosis of TPP was made.

Discussion

TPP is a condition characterized by transient localized or generalized muscle weakness or flaccid paralysis which may be of sudden onset and recurrent. It is a rare complication of thyrotoxicosis. It is more common in Asian men, but it can occur in men of other racial background¹. Male to female ratio of incidence is 20:1². The special susceptibility of people of Asian descent is so far unexplained. The patient may even present with subtle signs and symptoms of thyrotoxicosis³.

In patients with TPP, the Na⁺/K⁺-ATP ase pump activity is considerably increased by excess thyroid hormones, resulting in an increased intracellular potassium shift. The common precipitating factors include high carbohydrate foods, alcohol ingestion, emotional stress, exposure to cold, strenuous exercise followed by rest, and treatment with insulin, acetazolamide or glucocorticoids⁴. Glucocorticoids have been shown to increase the number of Na⁺/K⁺-ATP ase molecules in skeletal muscle. They also increase insulin secretion both basal and first phase which in turn activates Na⁺/K⁺-ATP ase pump.

The weakness is usually symmetrical and more obvious in the lower limbs than the upper limbs. The condition is customarily associated with hypokalemia but plasma potassium may be normal⁵. The mainstay of treatment is correction of potassium, thyrotoxicosis, and addition of beta-blockers to reduce the frequency and severity of the attack. In the patient described, he was already on treatment with antithyroid medications and beta-blocker. TPP was probably precipitated after administration of high dose methyl prednisolone for Grave's ophthalmopathy. He recovered after correction of hypokalemia and the dosage of beta- blocker was increased. This case is reported to show the

importance of awareness regarding the possibility of TPP being precipitated by steroid therapy in patients with Grave's ophthalmopathy.

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