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Case Report

An interesting case of thyrotoxic periodic paralysis

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ABSTRACT

Hyperthyroidism rarely cause paralysis by redistributive hypokalemia, with periodic attacks of paralysis termed as thyrotoxic periodic paralysis (TPP). It is more commonly seen in the Asian or Hispanic populations, genetic variation in Kir2.6, a muscle specific thyroid hormone responsive potassium channel cause predisposition of TPP. It typically presents in the early morning with limb weakness. Hyperthyroidism signs and symptoms are need not to be present in all patients. TPP attacks can be triggered by factors such a high-carbohydrate diet, sternous activity, mental stress, and steroids. TPP should be suspected in young patients presenting with acute muscle weakness and severe hypokalemia. Arrhythmias can be noted but most of these will resolve with normalization of potassium.

Keywords: Hyperthyroidism, Hypokalemia, Periodic paralysis

INTRODUCTION

Periodic paralysis is a group of muscle disorders characterized by episodes of painless muscle weakness, usually precipitated by exercise, fasting, or a high carbohydrate meal. The most common causes of periodic paralysis are often electrolyte disturbances, particularly of potassium. Both hypo and hyperkalemia may induce periodic paralysis. Of the rarer causes comes hyperthyroidism/thyrotoxicosis. Thyrotoxic periodic paralysis (TPP) is pretty uncommon in hyperthyroid patients, but is nevertheless very dangerous. There is associated hypokalemia with symmetrical proximal lower-limb weakness, which may progress to respiratory muscle paralysis and death. Owing to its lethal nature, early detection and prompt treatment is necessary.[1]

CASE REPORT

A 43-year-old male patient came to medicine outpatient department (OPD) with complains of sudden onset bilateral lower limb weakness. He was unable to stand up from a sitting position, and also complained of a history of fever for the past 2 days. He did not have any other complaint but on probing, he revealed that he has been a known case of hyperthyroidism for 1 year, and was not on any medication for the same.

On general examination, diffuse swelling present on the anterior aspect of the neck, on palpation there was no tenderness and nodules, his cardiovascular system (CVS) and respiratory system (RS) examination were normal. However, his central nervous system (CNS) examination revealed that bilateral lower limb the tone was flaccid and power was reduced to 3/5. There was no loss of reflexes in any of his limbs. His pulse rate (PR) was 110/min and his sleeping heart rate was 92/min. The electrocardiography (ECG) revealed that he had QT prolongation and the presence of U waves. A complete electrolyte and blood panel was advised and it was found that his serum potassium was only 1.8 mEq/l.

The patient was given a provisional diagnosis of hypokalemic periodic paralysis, patients TFT values are (FT3–10.3 pg/ml, FT4–6.72 ng/dl, TSH–<0.015 mIu/ml), the patient was found to be in a hyperthyroid state, serum

magnesium (1.2 mg/dl) and phosphorus (1.6 mg/dl) values are also low, then injection potassium chloride 60 mEq IV correction was given then oral supplements also given, and serum electrolyte profile was monitored at regular intervals, after potassium chloride correction also serum potassium is not improved to normal, then IV propranolol was given, on the next day serum potassium was came to normal. After the potassium correction, patient complaints were reduced and he recovered. For hyperthyroid state, carbimazole was started.

Figure 1: Diffuse swelling over the neck.

DISCUSSION

Thyrotoxic periodic paralysis is common among the Asian population, and can be due to hyperthyroidism from any cause including Grave's disease, multinodular goiter, toxic nodule, and inflammatory conditions of the thyroid. Though classified as a separate entity, TPP is most often due to the associated hypokalemia.[2]

It is common knowledge that skeletal musculature play a major role in the potassium homeostasis in the body. There are two major channels/pumps contributing to this - the Na-K ATPase pump and the inward-rectifying K channels, which are each responsible for inward and outward transfer of potassium respectively. Thyrotoxicosis causes an increased transcription of the Na-K-ATPase channel along with an increase in its intrinsic activity thereby letting potassium inside skeletal myocytes. Further, there may also be a loss-of-function mutation of the Kir2.6 channels. The entry of potassium into the myocyte causes a paradoxical depolarization leading to inactivation of existing sarcolemmal sodium channels thus causing paralysis.

Additionally, patients with TPP have been found to have high insulin response during the oral glucose challenge tests. It is highly possible that this insulin response may play a permissive role in the inward shift of potassium. This hyperinsulinemia associated response may also explain why TPP episodes are common after carbohydrate rich meals.

The usual symptoms are episodes of predominantly proximal muscle weakness, myalgias, and other features of hyperthyroidism like palpitations, tachycardia, and dyspnea. Typical features of hyperthyroidism like the presence of a visible or palpable nodule, exophthalmos, tachycardia and loss of reflexes may be seen on examination. The patients usually present with history of muscle weakness precipitated by exercise or high-carbohydrate meal.

Preliminary investigation should include a complete blood test panel with T3, T4 and TSH levels and serum electrolytes. The serum magnesium and phosphate may help distinguish TPP from other causes of periodic paralysis including familial periodic paralysis as hypomagnesemia and hypophosphatemia may be present in the former. An ECG is mandatory to check for rhythm abnormalities owing to low serum potassium. If resources are available, an electromyogram may be carried out to confirm the presence of myopathy.[3]

The first priority when it comes to therapy would be to correct the serum potassium levels with potassium chloride. KCl is either given orally and/or IV. Care should be taken to avoid excessive potassium supplementation as it may lead to rebound hyperkalemia during recovery.

Alternative treatment options include oral or IV propranolol to reduce the paralysis rapidly, especially in patients who do not respond to potassium. High dose oral propranolol of 3-4 mg/kg has been found to rapidly ameliorate paralysis.[4,5]

Further, it is now known that TPP cannot occur if the patient is euthyroid. Hence, control of hyperthyroidism will remain the mainstay of therapy over the long run.

Steps should be taken to identify the cause of hyperthyroidism and definitive treatment with radioactive iodine or surgery should be carried out. Patients should also be advised to avoid triggering factors like strenuous exercise or a high-carbohydrate meal.

CONCLUSION

For a long time, it was unknown that thyrotoxicosis can be a cause of periodic paralysis. However, now that we've recognized TPP as a separate entity, we also know that it is dangerous, and needs prompt management to prevent mortality. Goals of treatment should be to ameliorate the paralysis and to prevent further such episodes by maintaining a euthyroid state.

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