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Research paper

Thyrotoxic periodic paralysis mimicking hypokalemic periodic paralysis

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Abstract

Thyrotoxic periodic paralysis is a unique disorder that causes episodic proximal lower extremity muscle weakness in a patient who already has a hyperthyroid state and is not treated for the underlying disease state. The affected population mostly includes Asian in origin specifically the male gender (vs most thyroid disorder affects the women gender). Precipitating factors include in addition to a hyperthyroid state are strenuous exertion, a high-carbohydrate meal, and drugs like diuretics, insulin, high dose of steroids, antiretrovirals, and interferon therapy. Other genetic causes include the decreased activity of Kir2.6 mutation increased activity of Na+/K+ ATPase causing an intracellular shift of potassium that leads to hypokalaemia. The other challenging part for the physician is to differentiate it from Familial periodic paralysis an autosomal disorder seen in Caucasians and Western countries because both the disorders present similarly and differentiate between them by the presence of hyperthyroid state with hypokalaemia in Thyrotoxic periodic paralysis. The mortality is associated with respiratory paralysis hence the acute intervention includes giving nonselective beta-blockers followed by anti-thyroid medications. We present a case of an Indian adolescent who presented to us with lower muscle weakness with an underlying thyrotoxicosis state. This help physician with early diagnosis and appropriate treatment.

Keywords: *Endocrinology, Hyperthyroidism, Hypokalaemia, Hypokalemic periodic paralysis, Reversible cause of muscle weakness*

1. Introduction

The spectrum of muscle weakness to paralysis induced by hypokalaemia is called hypokalaemia paralysis and is the leading cause of hypokalaemia-related medical emergencies [1].The etiology of hypokalaemia paralysis can be generally classified into two groups: hypokalaemia periodic paralysis, due to shifting of potassium into the intracellular space without a total potassium deficit, and non-hypokalaemia periodic paralysis, due to a large potassium deficit via gastrointestinal or renal loss [2]. Among the hypokalaemia periodic paralysis, familial hypokalaemia periodic paralysis is the most common cause in Caucasians and Western countries, and thyrotoxic periodic paralysis, is characterized by the triad of acute hypokalaemia without total body potassium deficit, muscle paralysis, and thyrotoxicosis, is the most common cause in Asia population.

2. Case report

A 34-year-old Indian male patient who comes to the emergency department of our hospital presents with an extended body muscle weakness with sudden onset of lower proximal extremities muscle weakness unable to walk from early this morning. The patient has had a recurrent same episode two times in the last year. The patient does not misuse addictive substances or any drugs such as diuretics, insulin, or a high dose of steroids. The patient also complains of weight loss despite increases in intake of food, palpitation, tremor, and diarrhoea. The patient does not have any family history of lower muscle weakness and radiation exposure but a maternal history of diabetes mellitus. The patient's past medical history of hyperthyroidism was taking his drug regularly but does not consistent with his drug for the last 1 year due to his low social-economic status.

On physical examination in the emergency department, the patient appeared according to his age but does not able to move his lower extremity with a blood pressure of 90/54 mmHg, a pulse of 105 beats per minute, a temperature of 99.8°F, respiratory rate: of 18 breaths per minute, and oxygen saturation: 99% on room air has sinus tachycardia, fine tremors with hand extension, heat intolerance, mild exophthalmos, lid lag. Neck examination show enlargement of the thyroid gland with tenderness. Neurological examination shows decreases in weakness in the upper extremities by 3/5 in muscle strength and lower extremities by 2/5 in muscle strength and positive brisk and symmetric patellar reflexes. There are no other neurological deficits are found.

In the emergency department, Laboratory evaluations show serum potassium of 1.3 mEq/L, serum phosphorus of 1.5 mg/dL, magnesium 1.9 mg/dL. An electrocardiogram showed atrial fibrillation. On the same day, thyroid function tests show a marked increase in total T3 (296 ng/dL) and T4 (11 mcg/dL) with a significant decrease in thyroid-stimulating hormone (<0.05 mIU/mL) The patient was diagnosed with Thyrotoxicosis induced Thyrotoxic periodic paralysis. The patient immediately received an Intravenous KCl solution with normal

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saline and an oral KCl solution. In addition to that, the patient has also been provided a non-selective beta-blocker as well as anti-thyroid medication for thyrotoxicosis.
After three hours the patient was able to move his lower extremities and his potassium was 4.2 mEq/L and significant improvement in muscle strength of the lower extremities by 5/5. After one day of observation in the emergency, ward patient was discharged with non-selective beta-blockers and anti-thyroid medication.

3. Discussion

Thyrotoxic periodic paralysis is a type of reversible flaccid paralysis that is induced by hypokalaemia. The weakness is more commonly seen in the lower extremities muscle group compared to the upper extremities, especially in the proximal muscle group in contrast to the distal muscle group. The diseases can range from mild muscle weakness to quadriplegia to total paralysis [3, 4]. Precipitating factors include in addition of a hyperthyroid state as seen in this patient along with that strenuous exertion, a high-carbohydrate meal, and drugs like diuretics, insulin, high dose of steroids, antiretroviral, and interferon therapy.

Pathophysiology :Increases in T4 levels due to a hyperthyroid state cause increased secretion of insulin as well as increases in the adrenergic tone that stimulate the Na+/K+-ATPase which causes the potassium ion to shift intracellularly this leads to hypokalaemia and hyperpolarization of the cell membrane and result into periodic paralysis [5]. Predominantly affecting the male gender compared to the female gender due to the increased ratio of muscle to body mass index, they have more Na+/K+-ATPase pumps that result in Thyrotoxic periodic paralysis [5]. The physician must be able to differential Familial periodic paralysis an autosomal disorder is seen in Caucasians and Western countries due to a defect in L-type calcium channel 1-subunit (CACNA1S) on chromosomes 1q31-32 whereas, Thyrotoxic periodic paralysis is more commonly seen in Asian countries [6]. Familial periodic paralysis has no relation to thyrotoxicosis as well as it affects more first two decades of life and equally affects the male and female gender in contrast to that Thyrotoxic periodic paralysis affects the third to fifth decade of life male dominate disorder [7,8].

4. Conclusion

Thyrotoxic periodic paralysis is always put into differential diagnosis when considering hypokalaemia paralysis. It is always predominantly shown in the Asian population associated with the hyperthyroid state [9]. This increases awareness among physicians about this disorder because it is quick and reversible cause of hypokalaemia paralysis. This helps the physician with early diagnosis, management, and prevention of rebound hyperkalaemia [10].

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