

Thyrotoxic Periodic Paralysis

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Abstract

Thyrotoxic periodic paralysis (TPP) is characterized by abrupt onset of hypokalemia and paralysis. This condition primarily affects the lower extremities but may affect both upper and lower extremities and is secondary to thyrotoxicosis. It is most commonly seen in Asian men and it has been increasingly reported in USA due to the rise in the immigrant population. Hypokalemia in TPP results from an intracellular shift of potassium induced by the thyroid hormone sensitization of Na⁺/K⁺-ATPase rather than depletion of total body potassium.

Keywords: Thyrotoxicosis; Upper and Lower extremities; Paralysis

Introduction

Thyrotoxic periodic paralysis (TPP) is characterized by abrupt onset of hypokalemia and paralysis. This condition primarily affects the lower extremities but may affect both upper and lower extremities and is secondary to thyrotoxicosis [1]. It is most commonly seen in Asian men (Chinese, Vietnamese, Filipino and Koreans). In North America the incidence is 0.1% to 0.2%. Sporadic cases of TPP have also been reported in non-Asian population such as Caucasians, African-Americans, American Indians and Hispanics. The number of TPP cases reported in Western countries has increased recently in past decade. It has been increasingly reported in USA due to the rise in the immigrant population [2]. Hypokalemia in TPP results from an intracellular shift of potassium induced by the thyroid hormone sensitization of Na⁺/K⁺-ATPase rather than depletion of total body potassium. Treatment includes prevention of this shift of potassium by using nonselective beta-blocker, correcting the underlying hyperthyroid state and replacing potassium. TPP is curable once an euthyroid state is achieved. Early recognition of TPP is vital to initiate appropriate treatment and to avoid the risk of rebound hyperkalemia that may occur from high dose potassium replacement.

Case Report

A 31 year old Hispanic male arrived to Emergency Room by Emergency Medical Service with complaint of lower and upper Extremities weakness that started about 5 days prior to admission, which progressively worsened. On the day of admission, he was unable to ambulate. Apart from the weakness the patient denied dyspnea, fever, neck pain, no recent history of travel, no recent upper respiratory tract infection or gastrointestinal infection or sick contacts. His past medical history includes Grave's disease which was previously treated with methimazole but he discontinued the medication a few weeks ago. Patient denied using any recreational drugs, over the counter medications, prescription medications including laxatives or diuretics. He did however admit to recent tobacco abuse. He reported intermittent palpitations and diarrhea and had no family history of periodic paralysis.

On physical examination, patient was found to have 2/5 upper extremity and 1/5 lower extremity motor strength with hypoactive reflexes. He had no enlarged thyroid, no exophthalmoses, no sensory or cranial nerve deficit. Vitals signs were: Blood pressure 123/84, Heart rate 80 beats per min, pain 0/10, Respiratory rate 17, oxygen Saturation 98% in Room air. Initially in the emergency room, Potassium level was 1.8 mEq/L (reference range 3.6-5.0 mEq/L) and TSH was found to be 0.0 MU/L (Reference range 0.50-6.80 Mu/L).

EKG showed sinus rhythm with a prolonged Q-T interval; other labs showed sodium 139 mmol/L, chloride 108 mmol/L, CO₂ 21

mEq/L, BUN 13 mg/dl, Cr 0.7 mg/dl, anion gap of 10, Glucose 158, AST 21, ALT 24, Alkaline phosphates 169, Total Bilirubin 0.2, lipase 27, CPK 322, total thyroxin 18.5, thyroid peroxides antibody positive, CBC and CRP were within normal limits. Due to his past medical history and current symptoms, patient was diagnosed with thyrotoxic hypokalemic periodic paralysis (TPP). TPP is characterized by diffuse muscle weakness caused by transient shift of potassium into the cells. The Thyrotoxic periodic paralysis has a high prevalence in young Asian males compared to any other ethnicity. It is of utter importance to recognize these cases since the clinical condition may appear at any Emergency room because of the influx of the immigrant population. The condition primarily affects the lower extremities, with proximal muscle affected more than distal muscles and characterized by acute onset of severe hypokalemia.

He was given 40 mEq of intravenous potassium chloride in the emergency department and then was started on a normal saline infusion with 20 mEq/L of potassium chloride. He was also placed on an oral potassium replacement protocol with close monitoring of serum potassium level and EKG in the intensive care unit, which resulted in normalization of serum potassium level and resolution of his upper and lower extremities paralysis. Patient could not be started on propranolol at that time due to positive urine drug screen for cocaine which was found later on and patient was started on methimazole. Serial measurements of his serum potassium level in the hospital remained within normal limits and repeat TSH and FT4 level improved. The patient was discharged home with the diagnosis of TPP secondary to thyrotoxicosis/Grave's disease.

Discussion

One of the main goals of medical management is to replenish the potassium level as imbalance in potassium can lead to arrhythmia and cardiovascular complications. The guideline to replace potassium is: A) Critical deficit – serum potassium less than 2.0 mmol/L, which is a critical emergency and requires ICU/CCU admission and administration of potassium under continuous EKG monitoring by using Intravenous and oral potassium replacement if oral route possible. Intravenous potassium chloride 40 mmol in 100 ml IV Fluid over 1 hour. Oral

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potassium 28-42 mmol every 2 to 4 hours if tolerated and continue to measure serum potassium every 1 to 2 hours and replace until potassium more than 2.8 mmol/L. B) Severe deficit- serum potassium 2.0-2.5 mmol/L, requires ICU admission and administration of intravenous KCL 20-30 mmol in 100 ml IV fluid over 1 hour, continue to measure serum potassium every 1-2 hours until potassium more than 2.8 mmol/L. Two potassium tablets by mouth (28 mmol) per hour if tolerated. C) Moderate deficit- serum potassium (2.5-3.0) mmol/L, requires oral (preferred) or IV replacement, two potassium tablets by mouth (28 mmol) three times a day or Intravenous KCL 30 mmol/L at rate of 5-10 mmol/hr, repeat until serum potassium more than 3.2 mmol/L. D) Mild deficit-serum potassium 3.0-3.5 mmol/L, requires two oral potassium tablets (28 mmol) three times a day. Potassium Chloride is a good choice for supplementation. Rebound hyperkalemia occurs in approximately 40% of patients with TPP, especially if they receive more than 90 mEq of potassium chloride within the first 24 hours [3]. There is a positive correlation between the dose of potassium chloride administered and the degree of rebound hyperkalemia. Judicious replacement of Potassium is necessary because potassium can undergo a transcellular shift from extracellular to intracellular space.

Oral or IV preparations of Propranolol, a non-selective beta-adrenergic blocker (10-40) mg three times daily to four times daily is beneficial in aiding in potassium homeostasis and this is the initial treatment of choice along with potassium replacement [4]. It prevents the intracellular shift of potassium by blunting the hyper adrenergic stimulation of Na⁺/K⁺-ATPase and therefore allows for increase in Potassium levels and resolves the paralysis without the rebound effect of hyperkalemia [5,6].

The combination of nonselective beta-blocker and low dose potassium is the initial treatment of choice for facilitating recovery and reducing rebound hyperkalemia. Once patient is clinically stable and serum potassium level normalizes then antithyroid medication should be initiated. Without prompt treatment thyrotoxic periodic paralysis can progress to complete extremities paralysis without full recovery of motor strength. The definitive therapy and the corner stone for the treatment of TPP is the treatment of hyperthyroidism with antithyroid drug therapy, radioactive iodine ablation therapy or surgical thyroidectomy [7]. The underlying cause of a patient's hyperthyroidism should be identified and treated accordingly. Patients with Grave's disease, thyroid adenoma and multinodular goiter should undergo radioactive iodine ablation or thyroidectomy [8]. It is also

important to exclude precipitating factors such as high carbohydrate meals, excessive alcohol intoxication and vigorous physical activities. After initiation of definitive therapy, patient should avoid precipitating factors and continue propranolol until an euthyroid state is achieved to prevent recurrence. Once the euthyroid state is achieved, TPP is curable.

Conclusion

There are other differential diagnosis that could present with extremity paralysis such as Guillain-Barre syndrome, meningitis, Stroke, medication side effects or even malignancy. But TPP is one of the diagnoses often missed due to its low occurrence and also because of unawareness about this disorder. The combination of paralysis with hypokalemia in a patient with hyperthyroidism should prompt to any physician the possibility of this disease. The prompt diagnosis of this condition will help to avoid expensive laboratory studies, imaging studies and invasive procedures. It will also help to soon provide the patient with the appropriate treatment such as judicious potassium replacement in addition with propranolol to correct the hypokalemia, thus improving the Extremities weakness. Increased awareness among physicians regarding this condition will also result in early diagnosis, appropriate management and prevention of thyrotoxic periodic paralysis.

References

1. Lam L, Nair RJ, Tingle L (2006) Thyrotoxic periodic paralysis. Proc (Bayl Univ Med Cent) 19: 126-129.
2. McFadzean AJ, Yeung R (1967) Periodic paralysis complicating thyrotoxicosis in Chinese. Br Med J 1: 451-455.
3. Kung AWC (2006) Clinical Review: Thyrotoxic Periodic Paralysis: a diagnostic challenge. J Clin Endocrinol Metab 91: 2490-2495.
4. Shayne P, Hart A (1994) Thyrotoxic Periodic Paralysis terminated with intravenous propranolol. Ann Emerg Med 24: 736-740.
5. Lin SH, Lin YF (2001) Propranolol rapidly reverses paralysis, hypokalemia and hypophosphatemia in thyrotoxic periodic paralysis. Am J Kidney Dis 37: 620-623.
6. Lu KC, Hsu YJ, Chiu JS, Hsu YD, Lin SH, et al. (2004) Effects of potassium supplementation on the recovery of thyrotoxic periodic paralysis. Am J Emerg Med 22: 544-547.
7. Okinaha S, Shozume K, Lino S (1957) The association of periodic paralysis and hyperthyroidism in Japan. J clin Endocrinol Met 17: 1454-1459.
8. Lin SH (2005) Thyrotoxic Periodic Paralysis. Mayo Clinic Proc 80: 99-105.