ISSN: 2474-3674

Samad et al. Int J Crit Care Emerg Med 2021, 7:117
DOI: 10.23937/2474-3674/1510117
Volume 7 | Issue 1
Open Access

International Journal of
Critical Care and Emergency Medicine

CASE REPORT

Thyrotoxic Periodic Paralysis- An Uncommon Disease in the Western World: A Case Report

Sara Samad, DO, Joshua M Samuel, BSc* and Frida Popilevsky, MD

Coney Island Hospital, USA

***Corresponding author:** *Joshua M Samuel, BSc, Coney Island Hospital, 2601 Ocean Parkway Brooklyn NY 11235, USA*

Abstract

Thyrotoxic Periodic Paralysis (TPP) is a rare diagnosis in the western world; however, its incidence has been rising in recent years due to globalization. This condition has the potential to be deadly if not detected and treated in a time appropriate manner. This case discusses our management of a patient who came into our hospital with complaints of total body numbness and weakness for several hours. He was subsequently diagnosed with TPP and was treated with Methimazole and aggressive potassium supplementation and had an overall good outcome. This case report highlights an unusual disease that needs to be considered as a differential when a young man reports numbness and weakness to the clinician.

Introduction

Thyrotoxic periodic paralysis (TPP) is a disease categorized as weakness and paralysis due to hypokalemia in the setting of hyperthyroidism. TPP is rarely seen in North America, with an incidence of 1/100,000 [1], and it affects only 1%-2% of thyrotoxic patients in the United States. The disease is more common among Asian and Polynesian descent, affecting men more than women (17:1-70:1) and tends to affect those between the ages of 20 to 40 [2,3]. We recently treated a patient who was subsequently diagnosed with this disease. We report our findings in this manuscript in accordance with the guidelines found in the CARE checklist.

Case Presentation

This is a 38-year-old male with a past medical history of hyperthyroidism, non-compliant on Methimazole, who was admitted to the ER with the complaint of painless whole body numbness and weakness for five hours. He stated that a few months ago, he had a similar episode, which prompted him to go to a neurologist and was prescribed medication. His symptoms were associated with a dry throat, headaches in the occipital region, rated 4/10, and that extended to his bilateral shoulders. He denied any other symptoms such as nausea, vomiting, and chills. The patient endorsed that he recently emigrated from Uzbekistan one year ago and has lost 15 pounds unintentionally since then. He denied recreational drug use, tobacco, and alcohol usage. On physical exam, vitals were the following; BP: 120/83 mmHg, RR: 14, HR: 110 BPM, and T: 98.6 °F. He reported full sensation over the bilateral upper and lower extremity as well as the face. He was unable to lift his hands and feet against gravity, strength 2/5, his tone was flaccid on passive movement, and he presented with frequent hand and leg motion, although the patient stated he was not moving his extremities. CT scan of the head and neck showed no acute pathology.

Labs were significant for a potassium level of 1.3 mmol/L and a TSH of < 0.00 uIU/mL. He was sent to the Medical Intensive Care Unit for continued care. He received aggressive supplementation of potassium via the intravenous and oral route. Free T4 levels were later checked and were found to be elevated at 3.99 ng/dL and T3 at 295 ng/dL. Endocrinology was consulted for hyperthyroidism. Upon further workup, thyroglobulin level was 180 ng/ml, TSH receptor antibody: 2.83 IU/L, thyroid peroxidase antibody: 32.8 IU/mL, and thyroid-stimulating immunoglobulin: 3.04 IU/L. Our patient's findings were consistent with hyperthyroidism. He was started on Methimazole 20 mg daily, propranolol for heart rate control, and continued electrolyte supple-

Citation: Samad S, Samuel JM, Popilevsky F (2021) Thyrotoxic Periodic Paralysis- An Uncommon Disease in the Western World: A Case Report. Int J Crit Care Emerg Med 7:117. doi.org/10.23937/2474-3674/1510117

Accepted: March 29, 2021: **Published:** March 31, 2021

mentation. The rest of the patient's hospital course was uncomplicated, and symptoms resolved after aggressive potassium correction. The patient was discharged home with schedule follow-up appointment in the endocrinology clinic.

Discussion

Thyrotoxic Periodic Paralysis is a rare disease that was first documented by Dr. Rosenfeld back in 1902. The essential issue in this pathology is that the weakness and paralysis are in response to potassium being moved into the intracellular compartment, making the patient look hypokalemic. This hypokalemia is misleading because the body stores of potassium are normal. Potassium levels within the cells are regulated by the antagonistic function of the NA/K pump and Kir channel. These two channels are under control by several chemicals, such as thyroid hormones, catecholamines, androgens, and insulin. In TPP, these hormones are increased, and thereby modifies the activity of the channels [4].

The pathophysiology behind the thyroid hormone causing hypokalemia starts at the genetic level. Thyroid hormone enters the nucleus and binds to the thyroid response element upstream of the NA/K pump gene. This hormone works on the NA/K pump to synergistically create more of these channels with increased activity. This change occurs by both transcriptional and post-transcriptional modification. These Na/K pumps cause more potassium to be shuttled into the cells causing hypokalemia in most presentations. This hormone is the reason why hyperthyroid disorders are so closely associated with hypokalemia. While Grave disease is the most common thyroid disorder causing TPP, there have been reports that other thyroid disorders being associated as well. Examples include TSH-secreting pituitary adenoma, toxic adenoma, toxic nodular goiter, subacute thyroiditis of de Quervain, radiation thyroiditis with Graves disease, amiodarone therapy, and drugs containing tiratricol [3].

In contrast to thyroid hormones, catecholamines and insulin work by increasing the amount of potassium transported into the cell by working in two ways. One, they both inhibit the Kir channel activity, and secondly, they increase the NA/K pump activity. Catecholamines, more specifically, upregulates the B_2 receptor. Thus, beta-blockers such as propranolol are an essential component of treatment in TPP [3,5].

Androgens, on their own, have a unique setting in this disease. Throughout the literature, hyperthyroidism is usually a disease that affects women. TPP, on the other hand, is a disease that primarily targets men. Estrogen inhibits the Na/K pump, while testosterone increases this pump activity. A study conducted showed that men who were affected with TPP had a higher level of testosterone than men who did not have TPP. Another class of steroids that is linked to TPP would be the corticosteroids. Medications that we administer, such as dexamethasone and methylprednisolone, also upregulate NA/K pump. Unfortunately, an adverse effect of corticosteroids would be hyperglycemia, which leads to an insulin spike and further shuttles more potassium into the cells. Insulin levels can further rise after a large carbohydrate meal and have been an initiating event that causes TPP in some patients [6-8].

Before diagnosing TPP, it would be wise to think about other more common reasons for hypokalemia. Typical examples are diarrhea, vomiting, as well as renal pathologies. When considering renal function, a 24-hour urine collection would provide useful information. If the urine potassium to creatinine ratio is high, that indicates a renal pathology. A possible cause could be Renal tubular acidosis such Gitelman syndrome. For most clinicians, a 24-hour collection is not feasible, and an alternative method is necessary to assess the urine potassium/creatinine ratio from a urine sample. If it is under 13 and the patient is hypokalemic, then consideration that potassium loss can be either through its movement into the cell or loss of potassium from the gut [6].

After the more common differentials for hypokalemia are ruled out, the disease Familial Hypokalemic Paralysis (FPP) should be considered before TPP. FPP is a disease that looks almost identical in clinical presentation to TPP and is, in fact, more common in the western world. Two things can help the astute clinician to differentiate TPP from FPP. One is that this disease is an autosomal dominant disorder that equally affects men and women, doesn't have a predilection for Asians, and the first episode of paralysis usually occurs before the age of ten. Here, taking a good history from the patient would be vital. Also, there are different genetic associations with FPP when compared to TPP. FPP association includes CACN1AS and SCN4A [9], while the TPP association includes HLAB5, HLABW46, KCNE3 [10]. FPP must be ruled out because the drug Acetazolamide which is a vital component of treatment in FPP, is a drug that can initiate a TPP episode.

Early diagnosis of this disorder is critical to protect the cardiovascular system. Although it wasn't done in this case, a possible high sensitive test that can be done in the ED is a spot urine sample to determine the calcium/phosphate ratio. Usually, this should be performed early, when the patient is first admitted into the ED. If it is above 1.7, this is indicative of TPP. This test has a sensitivity of 100% and a specificity of 96% [11].

When it comes to the treatment of TPP, aggressive potassium replenishment should not be initiated because the risk of rebound hyperkalemia is very high. Also, all patients should receive an electrocardiogram. During this disease, findings include QT-U that is prolonged, a prolonged PR, and resting sinus tachycardia. Although nerve stimulation studies are not necessary

for diagnosis, if performed during the paralytic episode, there is a decrease in both compound muscle action potential and the amplitude of the evoked muscle action potential [12].

In conclusion, TPP has the potential to be a very deadly disease for the patient. Although rare, physicians need to have TPP as a possible differential when a young male patient gets admitted for weakness or paralysis. Fortunately, when identified early and treated appropriately, outcomes are excellent as they were in this case.

Authors Declarations

Reporting checklist

The authors have completed the CARE reporting checklist.

Conflict of interest

The authors report no conflict of interest.

Ethical statement

This report was in accordance with the institutional ethical standards and in accordance with the Helsinki Declaration. This case report has non-identifiable clinical data of our patient.

References

1. Kumar S, Sonkar S, Singh N (2018) Thyrotoxic hypokalemic periodic paralysis. Indian J Crit Care Med 22: 378-380.
2. Elston MS, Orr-Walker BJ, Dissanayake AM, Conaglen JV (2007) Thyrotoxic, hypokalaemic periodic paralysis: Polynesians, an ethnic group at risk. Intern Med J 37: 303-307.
3. Oh SB, Ahn J, Oh MY, Choi BG, Kang JH, et al. (2012) Thyrotoxic periodic paralysis associated with transient thyrotoxicosis due to painless thyroiditis. J Korean Med Sci 27: 822-826.
4. Chang C-C, Cheng C-J, Sung C-C, Chiueh T-S, Lee C-H, et al. (2013) A 10-year analysis of thyrotoxic periodic paralysis in 135 patients: focus on symptomatology and precipitants. Eur J Endocrinol 169: 529-536.
5. Garla VV, Gunturu M, Kovvuru KR, Salim SA (2018) Thyrotoxic periodic paralysis: Case report and review of the literature. Electronic Physician 10: 7174-7179.
6. Affram KO, Reddy TL, Osei KM (2018) A rare case of thyrotoxic periodic paralysis after epidural steroid injection: A case report and literature review. Am J Case Rep 19: 1453-1458.
7. Meseeha M, Parsamehr B, Kissell K, Attia M (2017) Thyrotoxic periodic paralysis: A case study and review of the literature. J Community Hosp Intern Med Perspect 7: 103-106.
8. Yao Y, Fan L, Zhang X, Xiao Z, Long Y, et al. (2013) Episodes of paralysis in chinese men with thyrotoxic periodic paralysis are associated with elevated serum testosterone. Thyroid 23: 420-427.
9. Ng WY, Lui KF, Thai AC, Cheah JS (2004) Absence of ion channels CACN1AS and SCN4A mutations in thyrotoxic hypokalemic periodic paralysis. Thyroid 14: 187-190.
10. Bhattacharjee S, Sanyal D (2013) Thyrotoxic hypokalemic periodic paralysis as the presenting symptom of silent thyroiditis. Ann Indian Acad Neurol 16: 218-220.
11. Lin SH, Chu P, Cheng CJ, Chu SJ, Hung YJ, et al. (2006) Early diagnosis of thyrotoxic periodic paralysis: Spot urine calcium to phosphate ratio. Crit Care Med 34: 2984-2989.
12. Thethi TK, Parks R, Katalenich B, Chhabra P, Mc Caw J, et al. (2014) Case of thyrotoxic periodic paralysis in a caucasian male and review of literature. Case Rep Med 2014: 314262.