

CAS CLINIQUE/CASE REPORT

THYROTOXIC PERIODIC PARALYSIS

A Case Report

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Atallah P, Dib ER, Khoury M. Thyrotoxic periodic paralysis. A case report. *J Med Liban* 2007 ; 55 (3) : 167-169.

ABSTRACT : Thyrotoxic hypokalemic periodic paralysis (TPP) – a rare complication of thyrotoxicosis and a medical emergency – is characterized by recurrent episodes of muscle weakness and hypokalemia associated with hyperthyroidism.

We report a case of TPP in a 38-year-old white Lebanese male. The patient suffered from severe muscle weakness of the upper and lower limbs. His blood tests revealed hypokalemia (k : 2.4 mEq/L), low thyroid-stimulating hormone TSH (0.001 µIU/mL) and normal levels of thyroid hormones. The thyroid scan showed a hot nodule.

His paralysis resolved with IV potassium. The patient was treated with propranolol and radioactive iodine with complete remission of the hyperthyroidism and the paralysis. A discussion of the clinical and pathophysiological features and treatment of TPP is presented.

INTRODUCTION

Periodic paralysis is a rare complication of hyperthyroidism. It has been described in diverse racial and ethnic groups, predominantly in Asian and Latin-American young males.

Thyrotoxic hypokalemic periodic paralysis (TPP) is a disorder characterized by concurrent thyrotoxicosis, hypokalemia and progressive symmetrical weakness leading to paralysis of extremities and other muscle groups. In TPP, hypokalemia is typically associated with hypophosphatemia and mild hypomagnesaemia [1]. The pathophysiology of TPP remains largely unexplained and controversial [1].

In this report, clinical and biochemical findings in a young man with hyperthyroidism who experienced an acute attack of periodic paralysis are described.

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Atallah P, Dib ER, Khoury M. Paralyse périodique thyrotoxique. A propos d'un cas. *J Med Liban* 2007 ; 55 (3) : 167-169.

RÉSUMÉ : La paralysie périodique thyrotoxique (PPT) est une complication rare de la thyrotoxicose et une urgence médicale. Elle est caractérisée par des épisodes paralytiques récidivants associés à une hypokaliémie et une hyperthyroïdie. Un cas de PPT chez un Libanais de 38 ans est rapporté. Le patient s'est présenté aux urgences pour une faiblesse sévère des quatre membres. Il avait une hypokaliémie (k : 2,4 mEq/L), une TSH très basse (0,001 µIU/mL) et des hormones thyroïdiennes normales. La scintigraphie thyroïdienne a montré un nodule toxique à l'origine de l'hyperthyroïdie.

Sa paralysie a régressé après l'administration de potassium par voie intraveineuse. Le patient a ensuite été traité par du propranolol et de l'iode radioactif avec une rémission complète de l'hyperthyroïdie et des accès paralytiques. Une discussion des caractéristiques cliniques, physiopathologiques et du traitement de la PPT est présentée.

CASE REPORT

A 38-year-old Lebanese male presented himself to the ER for severe weakness of the four limbs upon awakening with incapacity to stand upright. He had a recent history of diffuse myalgias and cramps at night that used to resolve spontaneously in the morning. He complained of severe lower back pain two days prior to his admission. Two weeks earlier he had cramps at the shoulders, lower limbs and epigastric region with some weakness. Six months earlier he had cramps and weakness at both lower limbs. He had lost 5 kg the previous two months even though he had increased his food intake and complained about excessive sweating, intolerance to heat and nervousness.

The patient is a smoker with no other medical history. No similar familial history was recognized.

The physical examination showed a blood pressure of 130/80 ; the heart rate was 95/mn and the temperature was 37°C.

The neurological exam showed normal cranial nerves and visual fields, no nystagmus, no diplopia, the motricity was 1/5 over both lower limbs and 2/5 over both upper limbs, he had hyperreflexia of both upper and lower limbs, plantar reflex on flexion and he had normal sensation all over.

The rest of the physical exam revealed no other

abnormalities. The thyroid gland was not palpable.

The blood tests showed : WBC 11600 : 75% PN/24% L ; Ht 45% ; Creatinine 0.64 mg/dL (0.6-1.2) ; Ca 9 mg/dL (8.6-10.2) ; Ph 3.6 mg/dL (2.7-4.5) ; Alb 3.8 mg/dL (3.5-4.8) ; Mg 2 mg/dL (1.8-2.4) ; Alkaline phosphatase 143 U/L (32-104) ; SGOT 24 U/L (0-37) ; SGPT 50 U/L (0-31) ; Na 144 mEq/L (133-148) ; K 2.4 mEq/L (3.5-5) ; Bicarbonate 22.9 mEq/L (22-32) ; CPK 340 U/L (24-167).

The EKG shows a prolonged QT of 520 msec. The cervical MRI was normal. EMG was not done neither was HLA study.

The patient's paralysis resolved in 6 hours with IV potassium infusion of 30 meq KCL over 3 h and the serum potassium levels rose to normal ranges K 5.1 meq/L ; Na 142 meq/L ; CPK level decreased to 234 U/L.

The thyroid function tests revealed :

TSH 0.001 μ IU/mL (0.35-4.9) ; FT4 1.84 ng/dL (0.70-1.85) ; FT3 2.9 pg/mL (1.71-3.71).

The radioiodine thyroid scan showed a hot nodule in the left thyroid lobe with extinction of the right lobe.

The diagnosis of TPP due to a toxic nodule was confirmed and the patient was treated with 25 mci of radioactive iodine and propranolol 10 mg twice daily.

One month later the patient called from Europe where he works complaining of nausea, fatigability and palpitations. The thyroid hormones were as follows : TSH : 0.001 μ IU/mL ; FT4 : 4 ng/dL (0.8-1.85), and an anti-thyroid drug (methimazole) 15 mg daily was added to the previous treatment for two months. Later on he became euthyroid and no further episodes of paralysis occurred two years after his presentation to the ER.

DISCUSSION

TPP is often unrecognized when first encountered because of its relative rarity. It has been confused with other similar clinical problems such as familial periodic paralysis, Guillain-Barré syndrome, spinal cord compression, polyneuropathy, or acute muscular strain [1]. It is an endocrine emergency, and it is important to recognize and diagnose promptly to prevent repeated admissions to the hospital and to decrease morbidity and mortality [2].

TPP is a complication of thyrotoxicosis that is found most commonly in men. The incidence was greater in Asia than in the West until recently with an increase in reported cases of TPP in Western countries [2-3]. In the North American Caucasian population, the incidence is estimated to be between 0.1% and 0.2% of thyrotoxic patients, and the onset is typically between the ages of 20 and 40 years [2].

It has been associated with some human leukocyte antigens : HLA B46, DR9, DQB1 0300 in Hong Kong Chinese TPP patients, HLA A2, BW22, AW19, B17 in Singapore Chinese patients and DRW8 in Japanese patients [2].

Pathophysiologically, various attempts have been

made to explain TPP, e.g., by the intracellular shift of potassium due to thyroxin-stimulated enhancement of sodium potassium adenosine triphosphate (Na/K ATP ase) pump activity on cellular membrane. This increases the transcellular influx of potassium, leading to a shift of potassium ions from the extracellular space to the intracellular space [2, 4].

Electromyographic studies during a paralysis showed myopathic changes and normal peripheral nerve function with absence of myopathy between attacks [3, 5].

TPP occurs most commonly in patients with Graves' disease, however, any cause of hyperthyroidism including excessive use of a substitute, iodine induced thyrotoxicosis [6], thyrotoxicosis factitia [7] or thyrotropin-secreting pituitary adenoma [8], toxic adenoma [5] may lead to this disorder.

Some patients experience prodromal muscle pains, stiffness, or cramps in the thighs one hour to three days before the onset of paralysis as was the case with our patient. The paralysis is sporadic and occurs suddenly, and its onset typically occurs during sleep or in the morning immediately upon awakening, often after a day of strenuous exercise [3]. Severity varies from paraparesis to severe flaccid tetraplegia. The absence of sensory deficit and preservation of deep tendon reflexes help to differentiate TPP from other causes of acute paraplegia.

Factors that can precipitate an attack of TPP include high-carbohydrate meals, elevated insulin levels, rest after heavy exercise or strenuous activity and alcohol consumption [3]. Any hyperadrenergic state can also precipitate an attack [1]. Other triggering factors include acetazolamide use, trauma, cold exposure, infections, menses, emotional stresses [3] and, recently, one case has been reported after prednisone use [9].

Laboratory studies reveal an elevated thyroid hormone level, and decreased serum TSH level [1, 3]. Hypokalemia is generally present at less than 3 mmol/L. Mild hypophosphatemia and hypomagnesemia occur in two thirds of the cases [3] but were normal in our patient. Serum creatinine phosphokinase of muscle origin is elevated in two thirds of patients [3], it was elevated in our patient and decreased 3 hours after K perfusion. The mild elevation of serum alkaline phosphatase and liver enzyme in our patient are probably related to his hyperthyroidism [3]. Two cases of normokalemic periodic paralysis with hyperthyroidism have been reported [10].

Electrocardiogram abnormalities include sinus tachycardia, diffuse ST changes, flattened T waves, prolonged QT intervals, and U waves [1]. Second-degree AV block, ventricular fibrillation, and ventricular tachycardia may also be present [1, 3].

Definitive treatment for TPP is correcting the hyperthyroidism [2-3]. Once a euthyroid state is achieved, paralytic attacks rarely occur [1, 3].

As in a hospital setting, close observation of serum potassium levels is a general rule [2-3]. It has been reported that recovery from paralysis may be hastened

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by administration of potassium, intravenously or orally. Recovery can be spontaneous after three to thirty-six hours, and serum potassium and phosphate levels can normalize without replacement [1-2]. However, it is important to monitor the patient for rebound hyperkalemia [3].

Our patient was living in Europe since his childhood. With increasing immigration, TPP is likely to occur more frequently in both Europe and North America and awareness of the condition is vital [2].

CONCLUSION

Thyrotoxic hypokalemic periodic paralysis should be considered in any acute episode of motor paralysis in young patients, especially males.

Although the pathogenesis of this cumbersome disorder has not yet been fully elucidated, and the reason for sexual and racial predisposition remains unknown, there is strong evidence that successful therapy of hyperthyroidism leads to regression of all clinical and laboratory parameters of the disorder, as was the case with our patient.

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شلل بفترة الانسمام الدرقي. سيرة حالة

موجز : الشلل بفترة الانسمام الدرقي اختلاط نادر الحدوث وهي حالة طارئة طبية تتصف بفترات شلل معاودة مشتركة مع نقص البوتاسيوم وفرط الدرقية. نذكر حالة شلل بفترة الانسمام الدرقي عند لبناني عمره ٢٨ عاماً. راجع المريض الطوارئ لضعف شديد بالاطراف الاربعة. تبين عنده نقص البوتاسيوم $k+2,4$ مللي مساوي / ليتر $2,4 \text{ mEq}$ وهورمون منشط الدرغ متدني $0,001 \mu\text{IU/mL}$ والهورمونات طبيعية الدرقية. تصوير الدرغ الومضاني اظهر وجود عقدة سمية هي اساس الانسمام الدرقي. تراجع الشلل بعد اعطاء البوتاسيوم وريديا. بعد ذلك عولج المريض بالبروبرانولول واليود المشع فتراجعت الاعراض كليا لفرط الدرقية ونوبات الشلل. قدمت ونوقشت الخصائص السريرية والامراضية والعلاج.