

Thyrotoxic hypokalemic paralysis: case report

Alberto Kurzbaum MD^{*}; Maria Gershovich MD[^], Lucy Lieberman MD^{*}; Safori Gassan MD^{*}; Joseph Reifler MD^{*}

Unit of Emergency Medicine ^{} and Internal Medicine [^] Poria Government Hospital; Tiberias, Israel*

Abstract

This article describes a case of acute flaccid paralysis and severe hypokalemia in a young Thai man as the first manifestation of thyrotoxicosis. The patient demonstrates the salient manifestations of this endocrine emergency. We discuss the pathophysiology, diagnosis and treatment. The emergency physician is often the first to see these patients and deserves a high index of suspicion to avoid a missed diagnosis.

MeSH words: Periodic Paralysis; Thyrotoxicosis, Complications

Introduction

Thyrotoxic periodic paralysis (TPP) is a rare endocrine emergency associated with thyrotoxicosis. Any cause of thyrotoxicosis can cause an attack of TPP in susceptible patients. (1). This condition has three characteristic symptoms: elevated thyroid hormones, hypokalemia and episodic muscle weakness or paralysis. This condition has a clear racial and sex distribution for males of Asian descent and is rarely reported in Israel (2). We present a case of TPP as the first manifestation of thyrotoxicosis and a brief discussion of the pathophysiology and treatment of this emergency.

Case Report

A 31-year-old male, a Thai agricultural worker, arrived to the Emergency Department in the early morning hours because he was unable to stand and walk. He stated that the previous day he had done heavy physical work during banana harvesting. He stated that he had eaten many bananas.

He awoke with diffuse muscular aches. On rising from rise bed, he collapsed. Systems review was limited by a language barrier. However it was clear that this was the first time he had such an episode.

On examination in the emergency department the patient was immobile due to tetraparesis. His vital signs were as follow: blood pressure, 120/80 mmHg; pulse 100/min; respiratory rate 18/min; temperature 36.8°C. The pertinent findings were a mild diffusely enlarged and not tender thyroid gland. There was no exophthalmos or other obvious peripheral findings suggesting hyperthyroidism.

The neurological examination revealed an alert and oriented man with diffuse, severe, symmetric, muscle weakness. The deep tendon reflexes were absent and no muscle wasting or fasciculations were seen. Cranial nerve functions were normal and no pathological reflexes were

elicited. Sensation and consciousness were normal.

The electrocardiogram revealed a sinus tachycardia of 100/minute, PR segment of 0.18 sec, T wave flattening and prominent U waves especially evident in V2-V5. The ECG changes suggested hypokalemia before the initial laboratory results arrived.

The laboratory results were notable for a potassium level of 1.8 mEq/L (normal 3.5-5.3 mEq/L). The urinary potassium was 6.8 mEq/L. The remainder of electrolytes levels, plasma glucose, urea nitrogen, creatinine levels were normal. The calcium, phosphorus and magnesium levels were within normal limits. The blood count showed mild leukocytosis of $12,500/\text{mm}^3$ with neutrophilia (87%). The muscle enzyme assays, total creatine phosphokinase and CKMB fraction, were normal.

Because the age of presentation and no previous personal or relevant family history, the clinical diagnosis of thyrotoxic periodic paralysis was suspected, rather than the more frequent familial and nonfamilial subtypes of Hypokalemic periodic paralysis. A blood sample for thyroid function tests was sent. The results confirm the clinical suspicion. The TSH level was 0.002mIU/ml (normal 0.5-6 mIU/ml) and the FT4 4.5 ng/dl (normal 0.8-1.8 ng/dl). The aldosterone level was 73 pg/ml (normal recumbent 10-160 pg/ml).

Because the very low levels of potassium and notable ECG findings the I.V. route of replacement was chosen. The patient received 26 mEq of potassium chloride over a 2 hour period in the emergency department. He was admitted to the Internal Medicine service for monitoring and potassium replacement.

Within 6 hours the patient demonstrated improvement. The ECG showed resolution of the hypokalemic changes when the potassium levels reached 5.6 mEq/L.

While in the hospital he was treated with propranolol and mercaptazole. He was discharged after 3 days and was scheduled for outpatient follow up. He was advised to continue

medication, to avoid physical efforts and high carbohydrate intake.

Two days after discharge he returned to the hospital with a similar but milder episode of muscle weakness that occurred when the potassium level was 2.7 mEq/L. He received potassium replacement and improved rapidly. Because ambulatory evaluation was not feasible a thyroid isotopic scan was performed. The scan showed a normal gland that supported the diagnosis of Grave's disease.

After his second discharge was lost for follow up.

Discussion

Hypokalemic periodic paralysis may be primary (familial) or secondary. The primary type is more common and has an autosomal dominant inheritance. The primary type is associated with normal serum potassium during the asymptomatic period and usually mildly reduced during the period of muscle weakness. It has usually precipitating factors as high carbohydrate intake and cold exposure (3).

Secondary hypokalemic paralysis is less common. In patients who have their first attack of weakness in adulthood a secondary cause must be searched for (4). The secondary hypokalemic paralysis lacks a family history. The usual causes are: thyrotoxicosis, barium poisoning, primary aldosteronism, licorice ingestion gastrointestinal wasting disorders and renal tubular acidosis (3). Serum potassium levels are usually less than 3 mEq/L in the asymptomatic period and decreased further during the attacks (4).

Thyrotoxic hypokalemic paralysis (TPP) is a rare endocrine emergency associated with thyrotoxicosis. Any cause of thyrotoxicosis can provoke an attack in susceptible patients (1) including secondary causes as in thyrotoxicosis due to amiodarone (5). TPP has 3 characteristics: elevated thyroid hormones, hypokalemia and episodic muscle weakness or paralysis that may last for a few hours or persist for days. The frequency of the attacks is variable. It has a predilection for males of Asian descent. It has a prevalence between 1 and 8% among Chinese

and Japanese with thyrotoxicosis compared with a prevalence of 0.1 to 0.2 % of Caucasians in North Americans with thyrotoxicosis (6). An increased incidence is also seen in Hispanics and Native Americans and may be underestimated. Male sex is a characteristic of TPP and usually presents in the third to fifth decade of life. It was rarely described in Israel (2).

The attacks classically start while in bed at night or early in the morning following a day of strenuous work or exercise and increased carbohydrate intake. The attacks are also more frequent in summer, possibly relating to increased sweating in summer and increased ingestion of drinks rich in carbohydrates. The muscular weakness is variable in severity from mild to paralysis. Proximal muscles, especially in the lower limbs are most affected. Early symptoms may include aching stiffness or cramps. Cranial nerves and respiratory muscles are usually not affected. No sensory or mental symptoms are found. In general, deep tendon reflexes are decreased to absent (1).

Body stores of potassium are at normal levels. The hypokalemia results from an intracellular shift. In TPP hypokalemia is usually associated with hypophosphatemia and hypomagnesemia (7). Serious cardiac arrhythmias can occur. Rebound hyperkalemia may occur in 40% of patients (1) and mandates cautious potassium replacement during the attack.

Thyrotoxicosis must be present to invoke these episodes of paralysis and almost always hypokalemia is present during the attack. The etiology of the thyrotoxicosis is less critical and may not be clinically evident. The pathogenesis is not clear but the intracellular shift of potassium has been attributed to enhance of Na^+ , K^+ -ATPase activity (1).

Definitive treatment of this condition is the correction of the thyrotoxic state along with cautious potassium replacement during the attack. The potential cardiac effects of hypokalemia can indicate parenteral potassium treatment. Until the resolution of the thyrotoxicosis, the use of propranolol and avoiding usual precipitating factors are advisable.

TPP must be included in the differential diagnosis of acute muscle weakness. When it was determined that the patient is hypokalemic, (the ECG may be the first clue), the differential diagnosis narrows to include TPP, familial periodic paralysis, barium poisoning and a variety of potassium deficits disorders (3).

Because the clinical manifestations of thyrotoxicosis may be subtle it is mandatory to check thyroid function tests in patients with the acute onset of muscle weakness and/or hypokalemia. Our patient, a young Asian man, demonstrated the salient features of TPP. The age of presentation and the fact that he does not have a family history of periodic paralysis prompted a rapid evaluation of thyroid functions tests.

The emergency physician should be aware of this condition and should consider that population groups other than Asians may be affected.

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Correspondence:

A. Kurzbaum. Emergency Medicine Department.
Poria Government Hospital, Tiberias, Israel.
E-mail: akurzbaum@poria.health.gov.il
Fax: (972)4-6738478