Thyrotoxic Hypokalemic Periodic Paralysis in a Philippine Man

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In recent years, many East Asians seeking work have begun to arrive in western countries, including Israel. With the influx of this new population, new diseases have emerged that are unfamiliar to western medicine. It is important that we learn to recognize and understand these conditions, which are common knowledge to the practitioner in the East.

We report the case of a 28 year old Philippine man who presented with severe hypokalemia and profound muscle weakness. The patient had no history of hyperthyroidism or family history of periodic paralysis. Blood tests revealed hyperthyroidism and a diagnosis of thyrotoxic periodic paralysis was reached.

Serum potassium returned to normal and muscle weakness ceased after supplementation with potassium alone. Treatment was instituted for hyperthyroidism. This case illustrates how diseases well known to the clinician can present in unfamiliar forms in patients of different races. Here, periodic paralysis was the presenting symptom of hyperthyroidism.

Patient Description

A 28 year old Philippine man was admitted to the Wolfson Medical Center's emergency department with severe proximal muscle weakness, more pronounced in the legs, and vomiting. He reported that the previous evening he had participated in a dinner where he consumed large quantities of carbohydrates and alcoholic beverages. On the morning of admission he was unable to stand or raise his arms. He reported three episodes of vomiting earlier in the morning. The patient did not have a history of similar episodes. He denied taking any medications, including laxatives, diuretics, anti-mycotics or corticosteroids. The patient also denied any weight loss, diarrhea, licorice consumption, or exposure to paints. His social history, past medical history and family history were unremarkable.

The physical examination revealed a well-developed man in mild distress. Pulse was 110 beats/min, respiratory rate 18 breaths/min, blood pressure 144/76 mmHg, and temperature 37°C. Physical examination revealed a smooth, non-tender, slightly enlarged thyroid gland without bruit. Chest, heart and abdominal examination was normal. Neurologic examination revealed a lower extremity strength of 1/5 and an upper extremity strength of 2/5. Reflexes were slightly decreased. The patient did not have any sensory deficits.

Laboratory studies performed in the emergency department revealed the following: glucose 179 mg/dl, serum sodium 137 mmol/L, potassium 1.9 mmol/L, phosphate 3.6 mg/dl, chloride 98 mmol/L, blood urea nitrogen 31 mg/dl, creatinine 0.9 mg/dl, hemoglobin 14.2 g/dl, white blood cell count 6.7 x 10^3/µl. A single random urine sample for electrolytes showed sodium 78.5 mmol/L and potassium 41.6 mmol/L. Electrocardiographic recording showed sinus tachycardia with first-degree atrioventricular block.

The patient was admitted to our ward with initial blood potassium of 1.9 mmol/L and received 10 mEq of potassium chloride intravenously per hour over 12 hours to obtain a total of 112 mEq. A few hours after initiation of the therapy, a repeat measurement showed 2.1 mmol/L. On the second day of hospitalization serum potassium was 5.2 mmol/L and 5.4 mmol/L. Following administration of potassium, there was a marked amelioration in symptoms. A repeat neurologic examination was normal and a repeat ECG revealed normal sinus rhythm without first-degree atrioventricular block

Laboratory studies performed on admission to the ward were as follows: total thyroxin 280 ng/dl (normal 80–200 ng/dl), free thyroxin index 4.03 nd/dl (normal 0.90–1.90 ng/dl) and thyroid-stimulating hormone < 0.00 mU/L (normal 0.25–4 mU/L.). Three days later, a thyroid scan with technetium 99 was performed and the diagnosis of Graves' disease was made. The patient was started on propylthyouracil 100 mg three times a day. His serum potassium remained normal and he was discharged.

Comment

Thyrotoxic periodic paralysis is an uncommon disorder characterized by acute and reversible attacks of severe muscle weakness associated with low serum potassium. This endocrine emergency, caused by excessive thyroid hormones, can lead to respiratory failure, cardiac arrhythmia, and death. It is seen primarily in East Asian males, and is uncommon in Caucasians, blacks or Hispanics. In recent years attempts have been made to find the genetic basis of the disease [1] and genetic polymorphisms have been identified, though no clear conclusions have been reached.

Many patients with TPP have no obvious symptoms of hyperthyroidism. In the case of our patient, for example, TPP preceded overt symptoms and signs of hyperthyroidism and could therefore have been misdiagnosed. Primary factors reported to precipitate attacks of paralysis in these patients include excessive physical activity, excessive carbohydrate ingestion and alcohol consumption. Other factors include trauma, emotional stress, exposure to cold, and the use of medications such as diuretics, corticosteroids, insulin, adrenalin and pilocarpin.

Hypokalemia with paralysis can be the result of hypokalemic periodic paralysis caused by movement of potassium into cells, or non-HPP, due to potassium loss. It is important to distinguish between HPP and non-HPP [2]. In this case the laboratory results indicated HPP and more specifically, TPP. An intracellular shift of potassium may be responsible for the neuromuscular symptoms that are the hallmark of this syndrome. Although the mechanism of this rapid shift of potassium has not been clearly defined, several investigations have postulated an acute increase in sodium/potassium ATPase activity. Insulin may play a role in precipitating hypokalemia in this group of patients by activating the sodium/potassium ATPase pump. In our patient, a large carbohydrate meal on the day before admission to our ward was the trigger for the attack of paralysis. The rapid correction of serum potassium with potassium supplements also supports an intracellular shift of potassium as the mechanism of hypokalemia in our patient. In treating TPP with intravenous potassium, it is important to remember the phenomenon of rebound hyperkalemia [3]. We recommend supplementation in small doses and frequent monitoring of potassium levels both during and after cessation of the therapy to prevent possible complications, though later on non-selective beta-blockers such as propanolol may help to prevent paralysis attacks once patients are on anti-thyroid medications [4].

The cardiac presentation of tachycardia with first-degree atrioventricular block is more common in TPP than non-TPP patients [5], a fact that further supported the diagnosis. Since many patients with thyrotoxic periodic paralysis present with few or no symptoms of thyrotoxicosis, it is important to evaluate the thyroid function activity in any patient with unexplained hypokalemia and paralysis, especially patients of East Asian origin. This relatively uncommon but potentially dangerous clinical syndrome, if not recognized and treated appropriately, can lead to severe and life-threatening sequelae. Once a correct diagnosis is reached, a relatively simple intervention, i.e., correction of the hypokalemia, will resolve the clinical presentation, and correction of the thyrotoxic state is the definitive treatment of this disorder. In a typical case of TPP, normalization of thyroid function leads to complete cessation of paralytic attacks.

TPP = thyrotoxic periodic paralysis
HPP = hypokalemic periodic paralysis

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