Thyrotoxic Periodic Paralysis: an under-recognized condition

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Background

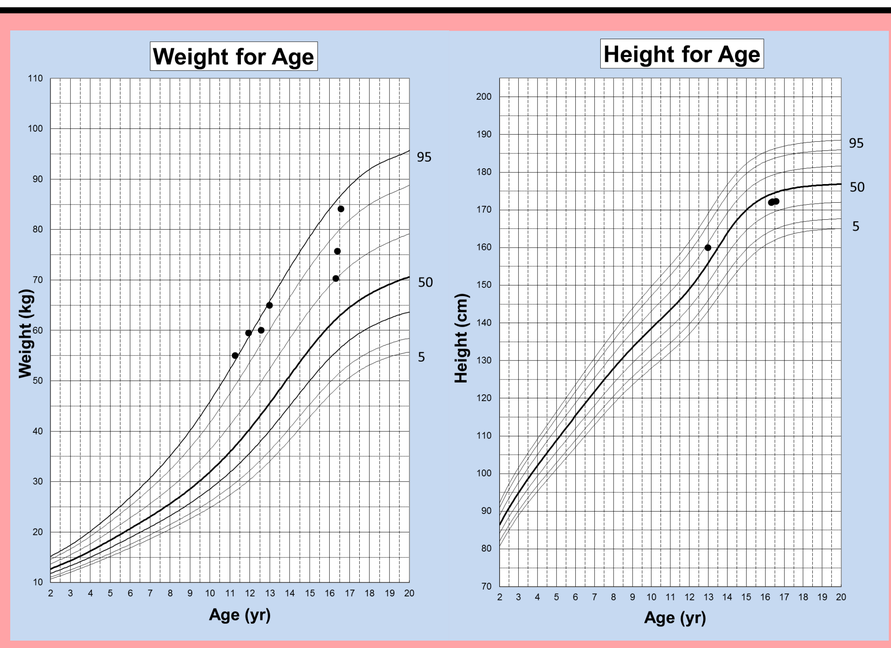
Thyrotoxic periodic paralysis (TPP) is a rare and potentially lethal complication of hyperthyroidism, commonly seen in Asian males and rarely in children. The condition is characterized by the triad of acute hypokalemia without total body potassium deficit, episodic muscle paralysis and thyrotoxicosis. Patients often present with sudden onset muscle weakness associated with severe hypokalemia. Many affected patients do not have obvious symptoms and signs of hyperthyroidism.

Clinical Case

A 16-year-old Filipino male, without significant past medical history, presented with acute onset bilateral lower extremities weakness. He was asymptomatic the day prior without any previous trauma/injury. He woke up at night but was unable to move his legs. His upper limbs were not affected. He denied any recent viral infections, ingestion of canned foods, and past history of paralysis.
He only recalled experiencing occasional palpitation for more than a year and more frequent bowel movements recently to 2 to 3 times a day. He also had a 2.5-kg unintentional weight loss despite increased appetite. He otherwise denied tremors, diaphoresis, mood lability or goiter. Family history was non-contributory.

Physical Exam

Vital signs: **BP 133/66mmHg, pulse 120/min**, temperature 36.8C. He was alert and calm. He had mild **exophthalmos** and **subtle hand tremors**. No lid lag or retraction. He had a **small goiter with audible bruits**. Chest was clear and abdomen was soft, no organomegaly. He had **bilateral lower extremity muscle weakness, 1/5 motor strength in proximal muscles with normal sensation. Reflexes were decreased.** Pubertal development was appropriate for age.



Investigations

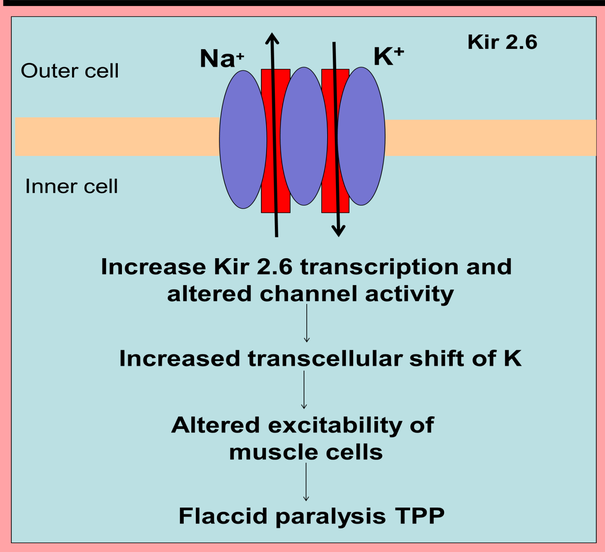
Labs	
Potassium	**2 mEq/L (3.5-5.5)**
Sodium	139 mEq/L (135-145)
Magnesium	**1.6 mg/dL (1.8-2.4)**
Calcium	8.7 mg/dL (8.5-10.2)
Creatine kinase	**795 IU/L (39-308)**
AST (SGOT)	22 IU/L (5-41)
ALT (SGPT)	**79 IU/L (10-50)**

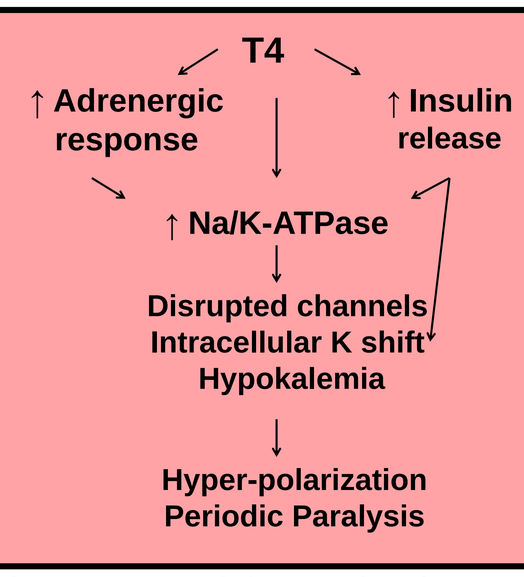
Labs	
TSH	**<0.02 mcIU/ml (0.5-4.5)**
Free T4	**>7 ng/ml (0.8-2)**
Total T3	**>7.8 ng/ml (1-2.1)**
Thyroperoxidase Ab	**353 IU/ml (<35)**
Thyroglobulin Ab	**>3000 IU/ml (<40)**
Thyroid-Stimulating Immunoglobulin	**6.6 (<1.3 TSI index)**

Clinical course

His initial thyroid function tests confirmed Graves' disease and TPP. In addition to hypokalemia, laboratory findings can include hypophosphatemia, hypomagnesemia and elevated creatine kinase.
He was managed with IV potassium and magnesium. Potassium normalized to 4.6 mEq/L and paralysis resolved upon discharge. Methimazole and Propranolol (non-selective β blocker) treatment was started and thyroid function tests improved. He remains euthyroid and without neuromuscular symptoms more than six months after initiation of therapy and normalization of thyroid hormone levels.

Discussion





The pathogenesis of thyrotoxic periodic paralysis remains unclear.
Hypokalemia is the consequence of exaggerated potassium influx. This is believed to be related to increased Na/K ATPase activity in skeletal muscles which causes increased intracellular shift of extracellular potassium.
Attacks can be precipitated by high carbohydrate meal, strenuous exercise and thyroid hormone excess, which stimulates the upregulation of Na/K ATPase.

Patients with TPP can have subtle signs and symptoms of thyrotoxicosis on presentation and is easily under-recognized. High index of suspicion is crucial in patients who present with acute paralysis associated with hypokalemia. It is important to exclude other causes of paralysis, including familial hypokalemic periodic paralysis, Guillain-Barré syndrome, transverse myelitis, spinal cord compression and other causes of hypokalemia such as renal tubular acidosis, hyperaldosteronism or diarrhea.
Early diagnosis and treatment of the hyperthyroid state prevent life-threatening complications of hypokalemia, such as cardiac arrhythmias and recurrence of paralysis.
Because TPP does not recur once the patient is euthyroid, adequate control of hyperthyroidism is the mainstay of therapy.

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