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LOWER-EXTREMITY WEAKNESS IN A TEENAGER DUE TO THYROTOXIC PERIODIC PARALYSIS

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□ **Abstract—Background: Thyrotoxic hypokalemic paralysis is the hallmark of thyrotoxic periodic paralysis (TPP). TPP is a potentially deadly complication of hyperthyroidism that occurs because of rapid and dramatic intracellular shift of potassium. This transference results in severe hypokalemia and clinically manifests itself as muscle weakness or paralysis. This condition predominantly affects males of Asian descent, and its presentation can range from mild to severe, as seen in our case. Case Report: We present the case of a 15-year-old Asian-American male who presented to a tertiary-care pediatric emergency department complaining of generalized weakness and flaccid paralysis of his lower extremities. The differential for such a complaint is extremely broad, and the symptoms can result from etiologies arising from the cerebral cortex, the spinal cord, peripheral nerves, the neuromuscular junction, or even the muscles themselves. Our patient was found to have an extremely low serum potassium concentration, as well as an electrocardiogram that revealed a prolonged QT interval and right bundle branch block. The etiology of these abnormalities and the patient's symptoms was found to be undiagnosed and uncontrolled hyperthyroidism from Grave's disease, which resulted in this dramatic presentation of thyrotoxic hypokalemic paralysis. Why Should an Emergency Physician Be Aware of This?: This entity is common in Asia but still somewhat rare in the United States and other Western countries. Our case illustrates that careful history taking and a focused diagnostic evaluation, in conjunction with having an awareness of this disease, can help expedite diagnosis and management, as well as avoid unnecessary and potential harmful testing in the emergency department setting.**

□ **Keywords—thyrotoxic hypokalemic paralysis; thyrotoxic periodic paralysis; hypokalemia; hyperthyroidism; Grave's disease; paralysis; weakness**

INTRODUCTION

Thyrotoxic hypokalemic paralysis is the hallmark of thyrotoxic periodic paralysis (TPP). TPP is a potentially deadly complication of hyperthyroidism that occurs because of rapid and dramatic intracellular shift of potassium. This transference results in severe hypokalemia and clinically manifests itself as muscle weakness or paralysis. This condition predominantly affects males of Asian descent, and its presentation can range from mild to severe. We present the case of a teenager who presented with acute-onset flaccid paralysis of his lower extremities that was the result of TTP from previously undiagnosed Grave's disease and severe hypokalemia.

CASE REPORT

A 15-year-old Chinese-American boy presented to a tertiary-care pediatric emergency department (PED) with complaint of weakness, more pronounced in the lower extremities than the upper extremities. The patient stated that

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he started having minor muscle aches and weakness approximately 2 weeks before presentation. On the day of presentation, the patient stated that his "joints felt sore" at 1 AM. Upon waking in the late morning, he was unable to move his legs, and had to ask his father to reposition his legs in bed. Due to this drastic change from the patient's baseline, he was brought to the PED.

On review of systems, the patient complained of weakness and fatigue, and stated that he was having intermittent difficulty breathing. He also endorsed nausea and mild abdominal pain, which he attributed to not eating. He denied any recent travel, vomiting, diarrhea, fevers, chills, weight or appetite changes, cough, headaches, vision changes, palpitations, or rashes.

The patient had a history of asthma, which was well controlled and for which he used only albuterol as needed. After detailed probing for previous neurologic issues, the patient stated that he had a resting tremor, which he described as mild, longstanding, and unchanged.

The patient's social history revealed that he lives at home with his mother and father. He attends a technical high school. He has never been sexually active. He does not smoke cigarettes or marijuana. He does admit to drinking approximately one beer per week and states that he used Ritalin recreationally approximately 1 week before presentation. He denied other drug use.

On physical examination the patient was unable to ambulate on his own. He was brought by wheelchair to the treatment room and helped onto the stretcher. He was awake and alert, in no apparent distress. Vital signs revealed a temperature of 36.9°C, pulse of 96 beats/min, respiratory rate of 22 breaths/min, blood pressure of 130/60 mm Hg, and oxygen saturation 100% on room air. His head, eyes, ears, throat, and neck examinations were normal, with no obvious facial asymmetry or neck masses. His heart and lung examinations were unremarkable. The patient's neurologic examination revealed normal cranial nerve function. Strength in the upper extremities was 4/5 bilaterally, and he had normal biceps and brachioradialis reflexes. Upper-extremity sensation was intact. Examination of his lower extremities showed that he was able to wiggle his toes and could move his feet against gravity. Strength was 0/5 in both legs, and he was unable to move either leg. He refused to attempt to bear weight. He had 1+ ankle reflexes but absent patellar reflexes. He had normal sensation to pain and light touch, as well as normal proprioception. His back was not tender to palpation.

Complete blood count showed white blood cell count 8.9 × 1,000/μL (76% neutrophils, 17% lymphocytes, 4% monocytes, 2% eosinophils), hemoglobin 15.4 g/dL (11.2–14.8 g/dL), hematocrit 45.3% (34%–43.9%), platelets 315 × 1,000/μL. Erythrocyte sedimentation rate was 9 mm/h (0−20 mm/h) and C-reactive protein was 0.6 mg/L (0.1–3.0 mg/L). Creatine kinase was 172 U/L (24−195 U/L), and creatine kinase MB was 2.7 ng/mL (<5 ng/mL). Urine and serum toxicology screens were negative. Results of negative inspiratory force testing performed in the PED were normal.

Serum chemistries showed sodium of 139 mmol/L, potassium of 1.7 mmol/L (3.5–5.0 mmol/L), chloride of 104 mmol/L, bicarbonate of 23.0 mmol/L, blood urea nitrogen of 12 mg/dL, creatinine of 0.7 mg/dL, and a glucose of 118 mg/dL (70−100 mg/dL). Calcium was 9.4 mg/dL (8.8–10.2 mg/dL), magnesium was 1.6 mg/dL (1.7–2.6 mg/dL), and phosphorus was 1.5 mg/dL (3.1–4.7 mg/dL). Liver enzymes were normal, other than an alanine aminotransferase of 66 U/L (0−34 U/L). An electrocardiogram was performed due to the severe hypokalemia and it revealed a rate of 84 beats/min with prolonged QT interval and right bundle branch block, but no T wave changes.

Careful questioning about the patient's family history revealed "thyroid issues." His mother had radioablation of a thyroid nodule, although she was uncertain why it was ablated. His father stated that he believes he had hyperthyroidism, but that he is not currently on any medications and did not have any ablation or other procedures. He did report one episode similar to his son's presentation, for which he received potassium supplementation. Given the patient's hypokalemia, strong family history of hyperthyroidism, and Chinese nationality, we had a high suspicion for thyrotoxic hypokalemic paralysis.

Thyroid studies confirmed our suspicion, revealing a thyroid stimulating hormone level of 0.008 uIU/mL (normal range 0.3–4.3 uIU/mL), total thyroxine level of 12.6 μg/dL (normal range 5.0–10.6 μg/dL), free thyroxine level of 2.8 ng/dL (normal range 1.0–2.2 ng/dL), thyroxine binding capacity of 20.7 μg/dL (normal range 19.0–28.0 μg/dL), and total triiodothyronine level of 240 ng/dL (normal range 79−149 ng/dL).

For his severe hypokalemia, the patient was given 40 mEq potassium chloride (KCl) orally, and was started on maintenance i.v. fluids with 20 mEq/L KCl. He was admitted to the pediatric intensive care unit for electrolyte repletion and careful cardiac monitoring. Repeated negative inspiratory force testing was normal. Three hours after oral KCl, the patient's potassium level was found to be 1.5 mmol/L, so he was given an i.v. run of 20 mEq KCl. A repeat potassium level 2 h later was 2.5 mmol/L, so he received a second 20 mEq KCl. Follow-up levels 2 and 6 h later revealed serum potassium of 4.3 and 4.8 mmol/L, respectively. Physical examination returned to baseline, with normal lower-extremity strength and reflexes by the time his serum potassium was 4.3 mmol/L. The maintenance fluids with KCl were discontinued at that point and the patient was transitioned to a high-potassium diet.