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Clinical Communications: Adult

CHANGING MANAGEMENT GUIDELINES IN THYROTOXIC HYPOKALEMIC PERIODIC PARALYSIS

Matthew Correia, MD, Mark Darocki, DO, and Eva Tovar Hirashima, MD, MPH

Department of Emergency Medicine, University of California, San Diego, San Diego, California
Reprint Address: Matthew Correia, MD, Department of Emergency Medicine, University of California, San Diego, 200 W. Arbor Dr. MC8767, San Diego, CA 92103

☐ **Abstract—Background: Periodic paralysis is a rare complication of hyperthyroidism. Patients of East Asian descent are most commonly affected. Presentation is characterized by recurrent episodes of painless, abrupt-onset weakness, with laboratory evaluation characterized by profound hypokalemia. Underlying hyperthyroidism may not be clinically evident, but differentiation from the familial variant is critical due to differing treatment pathways. Case Report: We describe the presentation of a 22-year-old man with recurrent relapsing-remitting weakness with undiagnosed hyperthyroidism. Why Should an Emergency Physician Be Aware of This?: In patients with acute-onset paralysis with significant hypokalemia, or relapsing-remitting symptoms, hyperthyroidism should be suspected. Obese patients are at an especially increased risk due to underlying insulin resistance, which enhances basal sodium-potassium ATPase function. Hypokalemia is functional in nature. Nonselective β-blockers (such as propranolol) should be considered first line, as they simultaneously decrease ATPase activity, limit insulin secretion, and address the underlying disorder. Administration of > 50 mEq of exogenous potassium places patients at risk of dysrhythmias from rebound hyperkalemia.** © **2018 Elsevier Inc. All rights reserved.**

☐ **Keywords—hypokalemic; periodic paralysis; thyrotoxic periodic paralysis; hypokalemia; hyperthyroidism**

INTRODUCTION

Periodic paralysis is a rare complication of hyperthyroidism. Patients of East Asian descent are most commonly affected. Presentation is characterized by recurrent episodes of painless, abrupt-onset proximal extremity weakness with laboratory evaluation characterized by profound hypokalemia. Underlying hyperthyroidism may not be clinically evident, but differentiation of this disease process from the familial variant is critical due to differing treatment modalities.

CASE PRESENTATION

A 22-year-old Hispanic man presented to the Emergency Department (ED) with persistent acute-onset weakness since awakening the previous morning. He described his weakness as symmetric and so severe that he had been unable to bear weight or lift his arms higher than his shoulders. He denied localized paresthesia or numbness, however, he complained of mild muscle soreness and difficulty mustering a strong cough. This was his sixth such episode in the last 2 months, and each subsequent event became more severe than the prior. In the past, his symptoms had typically abated within a few hours of awaking, but this episode was persistent. He had visited his primary doctor twice and two other local

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EDs without a clear cause for his symptoms. Diagnostic work-up had included a lumbar spine magnetic resonance imaging scan that was unremarkable.

The patient was unaware of any precipitating factors; symptoms were always present upon awakening and never started midway through the day. Findings from a pertinent review of systems noted persistent warmth, insomnia, trouble concentrating on tasks, and loose stools.

The patient had no significant past medical or surgical history. His family reported that he regularly ate candy and other "junk food." There were no family members with similar symptoms.

On physical examination, his vital signs revealed a heart rate in the low 100s (beats/min), a blood pressure of 117/65 mm Hg, a respiratory rate in the low teens (breaths/min), and normothermia. His general appearance was notable for an obese, young, alert man who required assistance to sit up in bed. There was no exophthalmos nor goiter. He had tonsillomegaly without erythema or exudates. Cardiorespiratory examination was notable for tachycardia. The abdomen was unremarkable. His skin was warm and diaphoretic; his posterior neck exhibited acanthosis nigricans. Strength was decreased symmetrically: 3/5 in his proximal upper extremities, 4+/5 distally with grip, 2/5 proximally at hips, and 4/5 with plantar and dorsiflexion at his ankles. He denied sensory deficits. Cranial nerve examination was unremarkable.

The electrocardiogram was notable for sinus tachycardia with a manually derived corrected QT of 478 ms (the computer calculated 284 ms) (Figure 1). Laboratory samples were notable for a potassium of 1.7 mmol/dL (ref 3.5–5.4 mmol/dL), magnesium of 1.8 mmol/dL, and calcium of 9.3 mmol/dL. Thyroid-stimulating hormone was undetectable, < 0.01 μIU/mL; creatine phosphokinase was 190 U/L; C-reactive protein was mildly elevated to 0.9 mg/dL (ref < 0.5 mg/dL), erythrocyte sedimentation rate was 11 mm/h (ref < 25 mm/h). White blood count was 16,600/mm^3 with 80% neutrophils. His presentation was deemed consistent with thyrotoxic hypokalemic periodic paralysis. He was started on potassium chloride replacement, receiving 90 mEq over the first 4 h (p.o. [by mouth], i.v. [intravenously] combined). He had only minimal improvement in weakness, and repeat potassium was 2.1 mmol/dL, so he was given an additional 40 mEq p.o. and 50 mEq i.v. potassium over the following 4 h. He had significant improvement in his weakness. Potassium normalized to 4.4 mmol/dL. He was admitted to the Medicine floor and Endocrinology was consulted. He was started on propranolol at 20 mg three times per day the following afternoon. Potassium the following morning returned at 4.5 mmol/dL; Free T_4 returned at 3.11 ng/mL (upper limit of normal 1.70); and T_3 at 3.2 ng/mL (upper limit of normal 2.0). Thyroid-stimulating immunoglobulins were 112% of baseline basal activity (ref $\leq$ 122%). Thyroid ultrasound was remarkable for a slightly hypoechoic segment within the left thyroid lobe, measuring 1.9 cm in its largest dimension. It was unclear if it was a true nodule or a hypoechoic region within an overall heterogeneous thyroid lobe. FT_4 down-trended over the course of his hospital stay and he did not have any recurrent weakness or paralysis. During his inpatient stay, he developed progressive odynophagia and was noted to have a positive rapid group A strep screen and was treated for pharyngitis with penicillin VK.

At his follow-up appointment later that month, symptoms remained controlled on 20 mg propranolol three times a day. At the time of writing this, he has a follow-up appointment pending to determine if repeat ultrasound or radioiodine uptake scan are necessary to evaluate whether he has Graves' Disease, thyroiditis, or a toxic nodule.

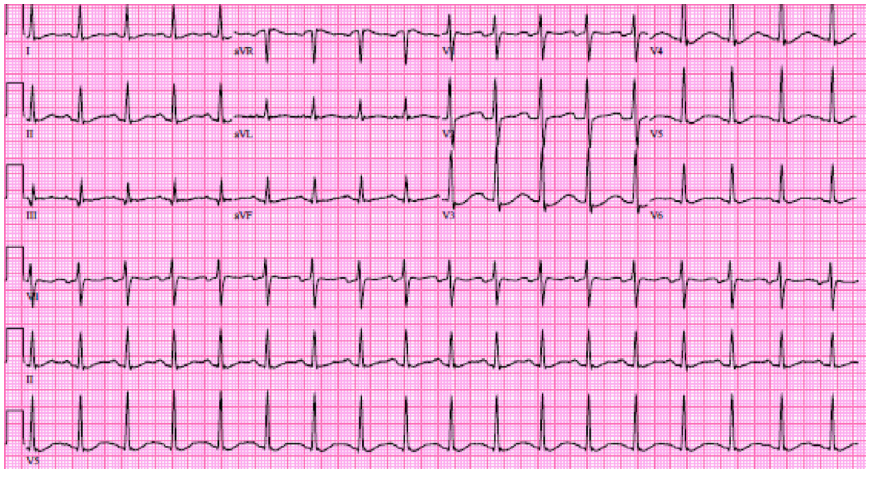

Figure 1. Electrocardiogram shows a heart rate of 106 beats/min, PR interval of 164 ms, QRS duration of 108 ms, and a QTc interval of 284 ms (computer) and 478 ms (calculated).