Electrocardiogram changes in thyrotoxic periodic paralysis

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INTRODUCTION

The overall incidence of thyrotoxic periodic paralysis (TPP) is 0.1-0.2% in North America, but varies dramatically among certain populations. In Chinese and Japanese patients, the incidence is 1.8-1.9%, the highest among any one population. Attacks are characterized as recurrent, transient episodes of muscle weakness that range from mild weakness to complete flaccid paralysis. Episodes of weakness are often accompanied by hypokalemia, and treatment of the underlying thyrotoxicosis will resolve symptoms of muscle weakness. Aggressive replacement of potassium can cause a drastic rebound hyperkalemia, but left untreated, severe hypokalemia can also cause life-threatening arrhythmias.

In this case, we report the presence of 3 distinct arrhythmias in a single patient as his potassium level changed during an episode of acute TPP.

CASE

A 29-year-old Asian male presented to the emergency department (ED) with symmetric paralysis of his lower extremities and weakness of his upper extremities that developed overnight. He had been recently diagnosed with hyperthyroidism 10 days prior, after presenting with 4 months of palpitations, muscle pain, cramping, and stiffness. He was started on propranolol and methimazole at that time. After resolution of his original symptoms, he stopped taking the medications and presented to the ED with complete paralysis of his lower extremities.

On further history, it was discovered that over the preceding 4 months the patient had clinical features of TPP that had been subtle, with reports of transient lower extremity weakness occurring that resolved without medical intervention.

On physical exam, the patient had symmetrical weakness and was unable to move his legs, but able to move his upper body, with sensation still intact. Upon initial laboratory evaluation, the basic metabolic panel (BMP) demonstrated a potassium level of 1.7 milliequivalent (mEq/L). The free thyroxine (T₄) level was elevated at greater than 8.8 ng/dL, and thyroid-stimulating hormone (TSH) was decreased at...
0.010 µU/mL. The initial electrocardiogram (ECG) showed 1\textsuperscript{st} degree heart block with prominent U waves (Figure 1). The PR interval was prolonged to greater than 0.2 sec as seen in leads V1 & V5, and U waves were present in all leads. There were no prior ECGs to compare these findings. Treatment was initiated with slow potassium repletion, but the potassium continued to drop to 1.6 mEq/L and the ECG showed 2\textsuperscript{nd} degree heart block, Mobitz Type I, best seen in Lead II and V1 (Figure 2).

The development of this 2:1 AV block coincided with increased weakness of more proximal muscles and decrease in reflexes. However, the patient’s airway was never compromised because his respiratory muscles were always intact. The paralytic attack was aborted with a combination of cautious potassium repletion, methimazole and parenteral propranolol. The patient received initial doses of potassium chloride 10 mEq intravenously and 40 mEq orally. A repeat BMP 3 hours later revealed a potassium level of 3.1 mEq/L and magnesium level of 1.8 mmol/L. Subsequently, the PR interval was noted to shorten and the rhythm returned to normal sinus (Figure 3).

Our patient exhibited numerous ECG changes due to hypokalemia secondary to thyrotoxicosis, with associated thyrotoxic periodic paralysis. Symptoms resolved, along with the seen ECG changes, with cautious potassium repletion and control of the underlying thyrotoxicosis.

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**REFERENCES**