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COMPLETE HEART BLOCK DURING POTASSIUM THERAPY IN THYROTOXIC PERIODIC PARALYSIS

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□ **Abstract—Background: Although cardiac dysrhythmia is common in patients with thyrotoxic periodic paralysis (TPP), high-degree atrioventricular (AV) block complicated by cardiogenic shock, even under KCl supplementation, is rarely described. Objectives: To present a case of TPP in a patient who developed complete AV block with severe consequences due to paradoxical hypokalemia during KCl therapy. In addition, the management of acute hypokalemia in TPP is reviewed. Case Report: A 41-year-old Chinese man with TPP presented to the Emergency Department with a 2-day history of paralysis in the extremities. He developed complete AV block with cardiogenic shock and respiratory failure, necessitating ventilatory support when plasma K^+ level decreased from 1.7 mmol/L to 1.3 mmol/L during KCl replacement of 30 mmol in 2 h. The administration of another 60 mmol KCl over 3 h achieved a plasma K^+ level of 2.1 mmol/L, resulting in the resolution of AV block and successful weaning. However, rebound hyperkalemia (K^+ 5.6 mmol/L) upon recovery was evident and uneventfully corrected. Conclusion: A paradoxical fall in serum K^+ concentration with potentially life-threatening complication is still underappreciated in patients with TPP on KCl supplementation. Early recognition and prompt therapy prevent untoward consequences.** © 2013 Elsevier Inc.

□ **Keywords—heart block; atrioventricular block; hypokalemia; thyrotoxicosis; thyrotoxic periodic paralysis; TPP**

INTRODUCTION

Hypokalemic paralysis (HP) is an electrolyte and metabolic disorder commonly encountered in the emergency department (ED). Patients with HP can be simply divided into two groups: one with hypokalemic periodic paralysis (HPP) due to an acute shift of K^+ into the cells, and another with non-HPP, where there is a large total body deficit of K^+ (1). Within the HPP subgroups, the most common conditions are: familial periodic paralysis caused by Ca^{2+} or Na^+ channelopathies (CACNA1S and SCN4A genes) of skeletal muscle, in Western countries; and thyrotoxic periodic paralysis (TPP) in Asia (2–4). With globalization and immigration, TPP has been increasingly reported worldwide (5).

In TPP, the cardiovascular system is very sensitive to thyrotoxicosis and plasma K^+ concentration (6). The classic triad of tachycardia, high QRS voltage, and first-degree atrioventricular (AV) block on electrocardiography (ECG) are very common in addition to typical hypokalemic findings (6). To prevent fatal cardiac dysrhythmia and to foster muscle recovery, the administration of KCl has been advocated (2). However, a further fall in plasma K^+ concentration due to a persistent and stronger driving force to shift K^+ into the cells during KCl therapy is still underappreciated in TPP. We describe

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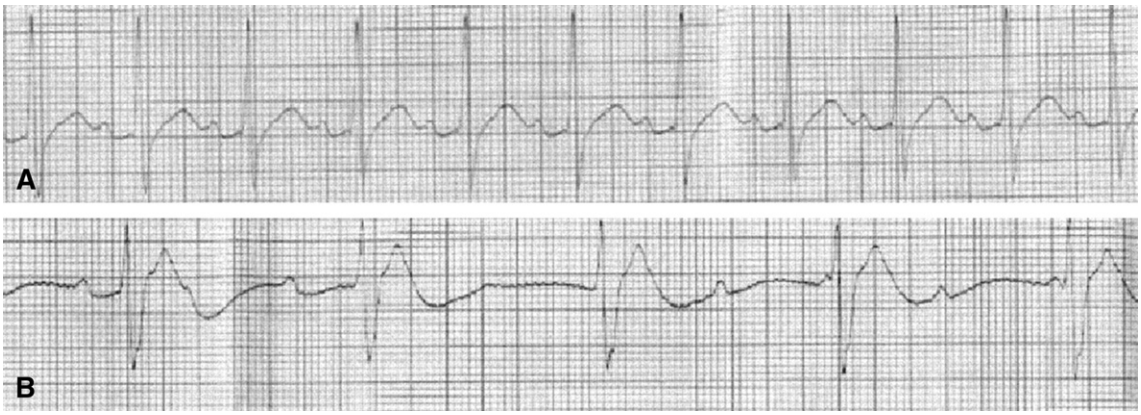

Figure 1. Electrocardiogram in lead II showing sinus tachycardia with first-degree atrioventricular (AV) block (plasma K^+ 1.7 mmol/L) in the emergency department (A) and complete AV block (plasma K^+ 1.3 mmol/l) 2 h later (B).

a Chinese man with TPP who developed complete AV block with cardiogenic shock and respiratory failure necessitating ventilatory support as a result of severe paradoxical hypokalemia during KCl supplementation.

CASE REPORT

A 41-year-old Chinese man presented to the ED with 2-day duration of muscle weakness progressing to paralysis in the extremities. He denied use of any medications, vomiting, diarrhea, palpitations, strenuous exercise, binge drinking, or binge eating in the prior days. His family history was non-contributory.

On arrival at the ED (hour 0), the physical examination revealed a blood pressure (BP) of 194/86 mm Hg, heart rate (HR) of 101 beats/min, and respiratory rate of 20 breaths/min. The thyroid gland was enlarged. Neurologic examination disclosed symmetrical paralysis of the four extremities. The remainder of the physical examination was unremarkable. Laboratory examinations showed profound hypokalemia (K^+ 1.7 mmol/L) accompanied by a normal blood acid-base state (pH 7.38, PCO_2 32.6 mm Hg, PO_2 102 mm Hg, and HCO_3^- 22.1 mmol/L). Other biochemical studies, including renal function, liver profiles, sodium, chloride, calcium, and magnesium, were within normal limits. An ECG revealed sinus tachycardia with first-degree AV block (Figure 1A).

After receiving 30 mmol of KCl orally and intravenously, the patient developed nausea, chest tightness, and cold sweating by the third hour (at hour 2). With central venous pressure of 13 mm Hg, the BP and HR decreased to 84/46 mm Hg and 60 beats/min, respectively. The ECG showed complete AV block (Figure 1B) while serum K^+ level dropped to 1.3 mmol/L (Figure 2). Despite immediate replacement of 20 mmol intravenous KCl, bradycardia (HR 30 beats/min) with profound shock (BP 70/30 mm Hg) and shallow breathing developed over 30 min. His arterial blood gas under O_2 mask (10 L/min) revealed pH 7.361, PCO_2 36.6 mm Hg, PO_2 70.8 mm Hg, and HCO_3^- 20.9 mmol/L. Therefore, the patient was transferred to the intensive care unit with dopamine (8 μg/kg/min) and non-invasive pressure preset ventilation support. With the additional administration of intravenous KCl 40 mmol within the next 2.5 h (at hour 5), his plasma K^+ concentration rose to 2.1 mmol/L, with accompanying normal sinus rhythm on ECG and improved muscle strength. Then the KCl infusion rate was

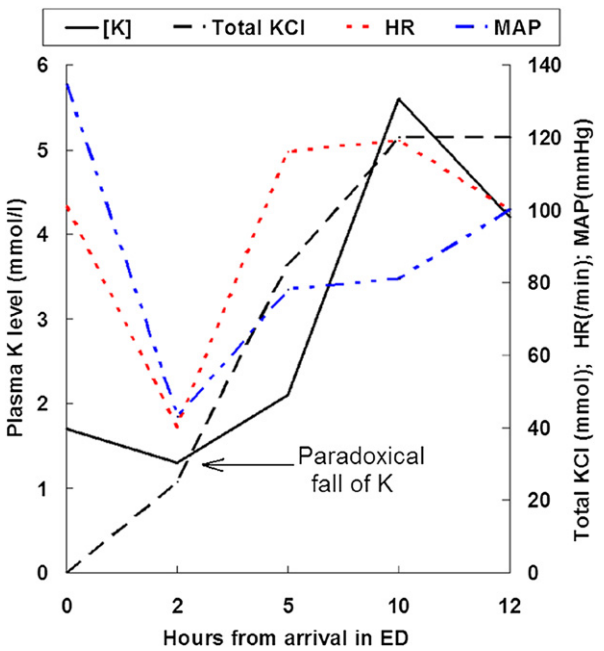

Figure 2. A paradoxical fall in the plasma K^+ concentration accompanied a drop in heart rate (HR) and blood pressure (BP) even after KCl supplementation of 30 mmol, followed by rebound hyperkalemia. Black solid line is plasma K^+ level; black dashed line is total dose of KCl supplemented; red dashed line is HR; blue dashed line is mean arterial pressure (MAP). ED = emergency department.

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