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TETRAPARESIS AND FAILURE OF PACEMAKER CAPTURE INDUCED BY SEVERE HYPERKALEMIA: CASE REPORT AND SYSTEMATIC REVIEW OF AVAILABLE LITERATURE

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☐ **Abstract—Background: In severe hyperkalemia, neurologic symptoms are described more rarely than cardiac manifestations. We report a clinical case; present a systematic review of available literature on secondary hyperkalemic paralysis (SHP); and also discuss pathogenesis, clinical effects, and therapeutic options. Case Report: A 75-year-old woman presented to the emergency department complaining of tetraparesis. Her serum potassium level was 11.4 mEq/L. Electrocardiogram (ECG) showed a pacemaker (PMK)-induced rhythm, with loss of atrial capture and wide QRS complexes. After emergency treatment to restore cell membrane potential threshold and lower serum potassium, neurologic and ECG signs completely disappeared. An acute myocardial infarction subsequently occurred, possibly linked to tachycardia induced by salbutamol therapy. We reviewed 99 articles (119 patients). Mean serum potassium was 8.8 mEq/L. In most cases, ECG showed the presence of tall T waves; loss of PMK atrial capture was documented in 5 patients. In 94 patients, flaccid paralysis was described and in 25, severe muscular weakness; in 65 patients, these findings were associated with other symptoms. Concurrent renal failure was often documented. The most frequent treatments were dialysis and infusion of insulin and glucose. Eighty-seven percent of patients had complete resolution of symptoms. Why Should an Emergency Physician Be Aware of This?: Severe hyperkalemia is always a life-threatening medical emergency, as it can precipitate fatal dysrhythmias and paralysis. SHP should be considered in the differential diagnosis of neurologic signs and symptoms of uncertain etiology, especially in a subject with kidney failure or who is taking medications that may worsen renal function. The presence of a PMK does not necessarily impede hyperkalemic cardiac toxicity.**

☐ **Keywords—hyperkalemia; paralysis; pacemaker capture failure; kidney failure**

INTRODUCTION

Severe hyperkalemia is a well-known life-threatening event that can lead to fatal cardiac dysrhythmias or neurologic derangements, such as muscle weakness and paralysis. Paralysis related to high serum potassium levels may be a recurrent and predictable syndrome due to a genetic disease (familial periodic paralysis) or an isolated, acute, and often undiagnosed event; the latter condition is known as secondary hyperkalemic paralysis (SHP). In clinical practice, neurologic symptoms are rarely seen, perhaps because cardiac manifestations begin earlier and are more frequently thought of and managed. We report a case where a patient presented with the chief complaint of hyperkalemia-induced paralysis and subtle, though very serious, cardiac abnormalities. In addition, we present a systematic review of available literature, discussing this condition together with the cardiac and neurologic effects of hyperkalemia, as well as its pathogenesis and therapeutic options.

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CASE REPORT

A 75-year-old woman was sent to the emergency department by her general practitioner, who diagnosed "Transient ischemic attack. Drop attack. Patient unable to keep a standing position." Her history revealed an acute myocardial infarction several years earlier, hypertension, sick sinus syndrome managed with a dual-chamber pacemaker (PMK), and mild chronic kidney disease. Her medications included acetylsalicylic acid (300 mg/d), benazepril (10 mg/d), amiloride/hydrochlorothiazide (5/50 mg/d), lercanidipine (10 mg/d), atorvastatin (20 mg/d), betahistine (8 mg/d), transdermal nitroglycerin, and occasional piroxicam and alprazolam. The patient was alert, oriented, and collaborative. She complained of progressive muscular weakness of a week's duration, initially in both lower limbs and spreading to the upper limbs in the last 12 h. At the time of admission, she was unable to walk or stand up. Vital signs showed a blood pressure of 150/70 mm Hg, heart rate of 60 beats/min, respiratory rate of 18/min, peripheral oxygen saturation of 96% on room air, and skin temperature of 36.5°C. She denied injuries, as well as fever, vomiting, change in bowel habit, and use of drugs or medications other than those prescribed. On clinical examination, skin and oral mucosa were pale and dry. Heart, lung, and abdominal examinations were normal. On neurologic examination, the lower limbs were completely flaccid, while muscles of the upper limbs had some residual power and greatly decreased tone; she had extreme difficulty moving her fingers and was able to move the upper limbs on the plane, but was unable to raise them against gravity. She was areflexic. There was no sensory deficit and plantar responses were bilaterally absent. Facial and lingual motility were normal but speech was difficult and apparently dysarthric. Bladder catheterization revealed 200 mL dark amber urine. Blood tests showed 201 mg/dL urea, 4.4 mg/dL creatinine, 110 mEq/dL chloride, 136 mEq/L sodium, and 11.4 mEq/L potassium. Arterial blood gas analysis showed high anion gap (AG) metabolic acidosis (pH 7.30, pCO_2 22.1 mm Hg, HCO_3 10.6, base excess −14.0, AG 26.9). At the electrocardiogram (ECG), a PMK firing at 60 beats/min was noted, with loss of atrial capture and wide (about 240 ms) QRS complexes (Figure 1).

The patient was rapidly treated with an intravenous bolus of 10% calcium chloride (10 mL), infusion of 25 g glucose, and 10 IU insulin over 15 min, followed by nebulization of 15 mg salbutamol. At the same time, an infusion of 80 mEq sodium bicarbonate over 5 min was commenced, followed by an additional 80 mEq and 40 mg furosemide. After excluding ureteral obstruction with renal ultrasonography, a rapid infusion of 1000 mL normal saline was started. Finally, an enteral solution with 15 g polystyrene sulfonate was administered.

After about 40 min, neurologic signs almost completely disappeared, and only moderate generalized weakness persisted. Two hours after hospital admission, serum potassium was 6.6 mEq/L and ECG showed atrial fibrillation with mean heart rate of 105 bpm, ST segment depression, and negative T waves in the inferior and lateral leads. Four hours later, a third ECG (Figure 2) showed spontaneous atrial activity and a PMK spike with regular ventricular capture; an inferioseptal myocardial infarction was now evident and confirmed by

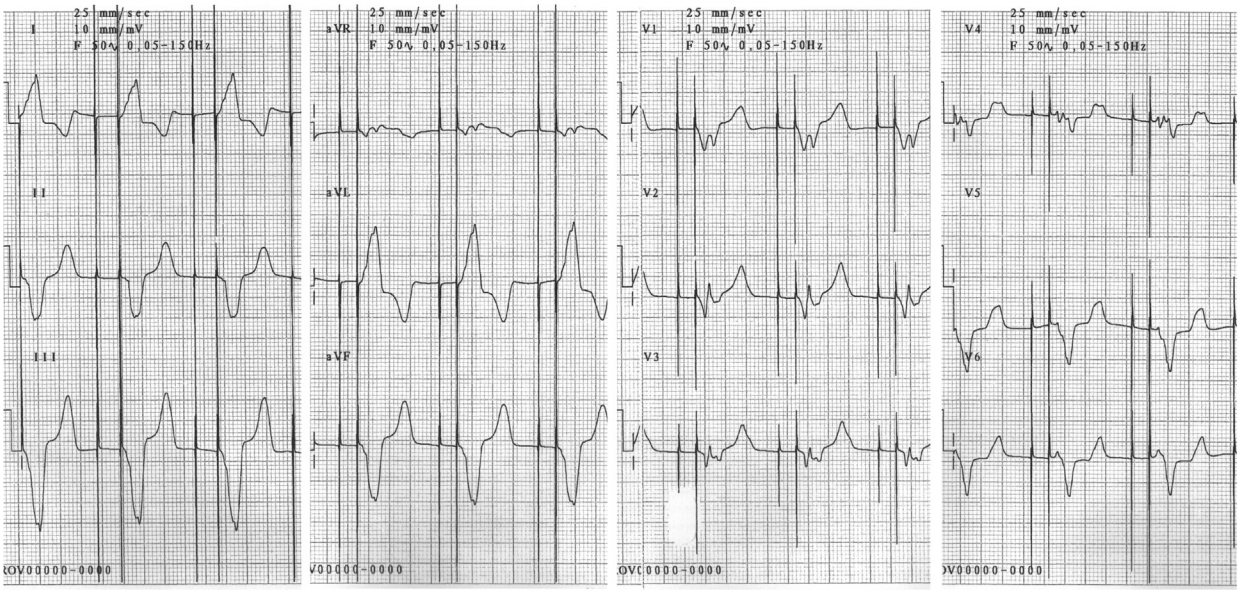

Figure 1. Electrocardiogram on admission (K^+: 11.4 mEq/L). Bicameral pacemaker, 60 beats/min; loss of atrial capture; very wide (about 240 ms) QRS complexes; tall and peaked T waves.