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Case Report
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Hyperkalemia Induced Arrhythmia Reversed by Calcium

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Abstract

Hyperkalemia is a life-threatening electrolyte disorder, most commonly occurring in patients with Chronic Kidney Disease (CKD), diabetes mellitus and heart failure. It usually manifests in nonspecific symptoms but may also cause cardiac arrhythmias including bundle branch blocks, bifascicular block, AV blocks, sinus bradycardia, sinus arrest, slow idioventricular rhythms, ventricular arrythmias and asystole.

Calcium counterbalances the effect of potassium on myocytes such that intravenous calcium antagonizes hyperkalemia induced cardiac membrane excitability and protects the heart against arrhythmias.

In this report we present a hemodynamically unstable patient in cardiac arrhythmia due to hyperkalemia that was stabilized with the sole use of intravenous calcium.

Keywords: Hyperkalemia; Electrocardiogram abnormalities; Calcium; Arrhythmia; Absent p-wave

Introduction

Hyperkalemia is a life-threatening electrolyte disorder, most commonly occurring in patients with Chronic Kidney Disease (CKD), diabetes mellitus and heart failure. It usually manifests in nonspecific symptoms but may also cause cardiac arrhythmias including bundle branch blocks, sinus bradycardia, ventricular arrhythmias and asystole.

Calcium counterbalances the effect of potassium on myocytes such that intravenous calcium antagonizes hyperkalemia induced cardiac membrane excitability and protects the heart against arrhythmias.

In this report we present a patient in cardiac arrhythmia due to hyperkalemia that was stabilized with the use of intravenous calcium. We present the initial and successive ECGs after calcium administration. Furthermore, we briefly review hyperkalemia related ECG changes, the role of calcium in hyperkalemia management and the literature supporting its use.

Case Presentation

A 76-year-old male with a history of end stage renal disease presented to the hospital with chest pressure, shortness of breath, upper back pain and lower extremity edema for the past four days. His last dialysis session was four days prior to this visit. The patient was triaged to a critical care room due to Electrocardiogram (ECG) abnormalities (Figure 1) obtained on arrival.

His initial vital signs were: Blood pressure 154/73 mmHg, pulse of 66, temperature of 98.5 °F, respirations 19 and oxygen saturation of 100% on room air. Physical exam was notable for an alert and oriented Hispanic male in irregularly irregular heart rhythm with a 2/6 systolic murmur. He had labored breathing but breath sounds were clear to auscultation bilaterally. The cardiac monitor showed a wide complex bradycardia with short runs of ventricular complexes.

Due to the ECG changes in the setting of hyperkalemia (serum potassium of 7.6 mEq/L), he was given two grams of calcium gluconate via slow Intravenous (IV) push. He then had a repeat ECG within five minutes which showed normal sinus rhythm.

He reported that he was feeling "a little better". He was also started on ten milligrams of nebulized albuterol, five units of IV insulin and 25 grams of dextrose (D50) to treat hyperkalemia. After 20 min, he again was noted to have runs of wide complex tachycardia prompting administering another two grams of calcium gluconate. A third ECG (Figure 2) two minutes after the second dose of calcium showed a narrow complex sinus rhythm.

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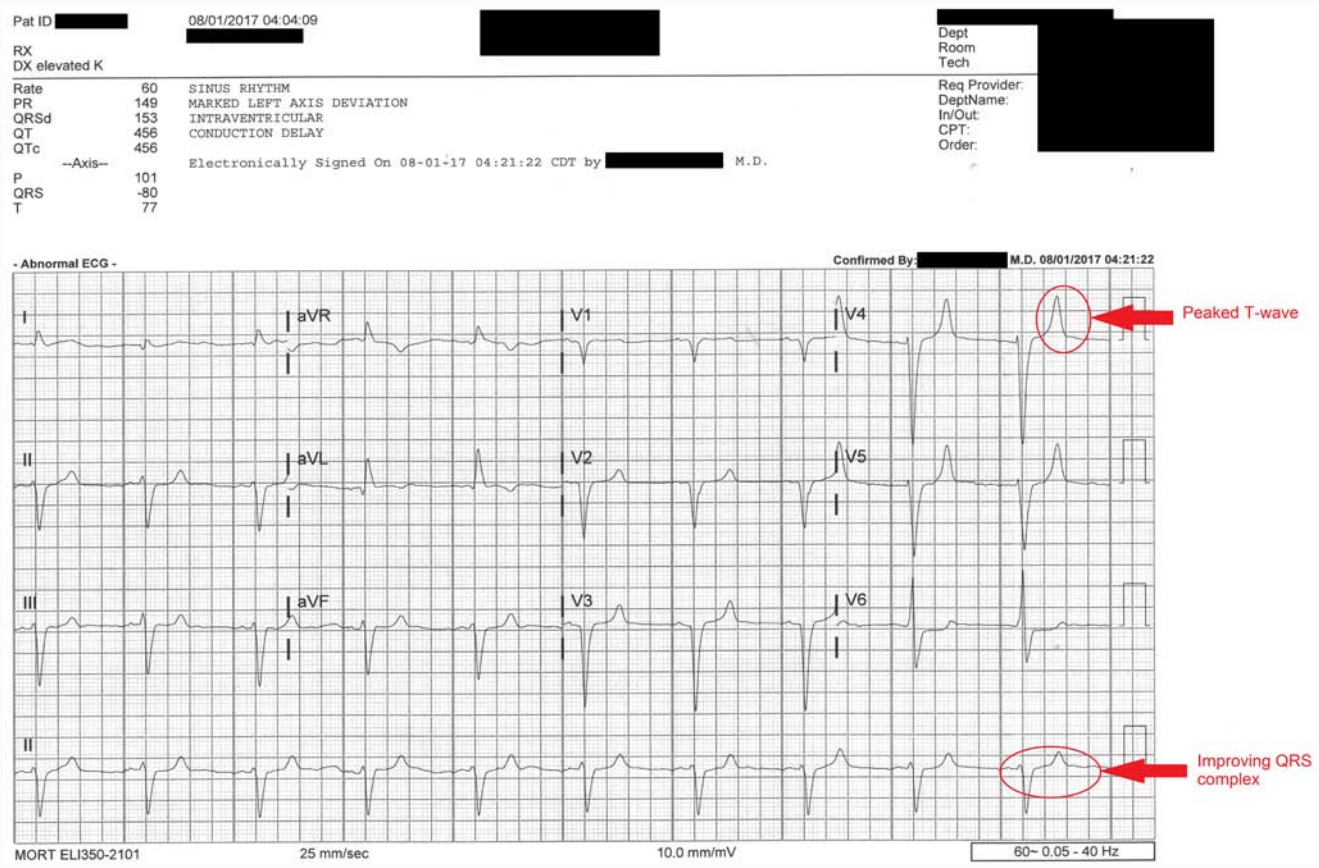

Figure 1: Electrocardiogram (ECG).

The patient was observed on cardiac monitor for several hours. He received hemodialysis in the emergency department and tolerated it well. His presenting complaints resolved after dialysis and he was discharged home with return precautions.

Discussion

This case report describes a potentially fatal cardiac arrhythmia caused by hyperkalemia and its correction by intravenous calcium. This report is unique because sequential electrocardiograms of hyperkalemia related arrhythmia are difficult to capture in the emergent setting, especially of those with life threatening potential. Moreover, this report shows that repeated calcium administration may be necessary to correct ECG abnormalities.

The potassium concentration gradient across the myocardial cell membrane establishes the membrane potential [1]. Hyperkalemia lowers the transmembrane gradient thereby shortening the duration of the action potential and repolarization time [2]. This is responsible for some of the early ECG changes, such as ST-T segment depression, peaked T waves, and Q-T interval shortening [3,4]. As potassium levels continue to rise, PR and QRS prolongation occurs. Other ECG manifestations include a diminished P-wave, QRS widening, and ultimately a wide-complex "sine-wave" that may progress to asystole [5,6].

Calcium antagonizes hyperkalemia induced cardiac membrane excitability and protects the heart against arrhythmias. Although its use for treating fatal arrhythmias is often recommended, data in human subjects are lacking. The toxic effects of potassium on myocytes, and its reversal by calcium, were first demonstrated in animal models [7,8]. Later, Intravenous (IV) calcium was shown to be effective in treating patients with acute kidney injury during the Korean War [8]. Although the bulk of evidence supporting the use of calcium comes from case reports and anecdotal experience [9], there is little doubt that calcium is an important and life-saving agent in the emergency treatment of hyperkalemia.

Some adverse effects of intravenous calcium are peripheral vasodilation, hypotension, bradycardia, and arrhythmias. A more serious adverse effect of IV calcium is tissue necrosis if extravasation occurs [10]. This can be avoided if calcium gluconate is used, which is considered less toxic on peripheral veins. Historically, caution has been advised with administration of IV calcium in patients with known or suspected digoxin toxicity because of case reports of death in this context [11,12]. On the contrary, there are reports showing no adverse effects of IV calcium administration in the presence of digoxin toxicity [13]. Furthermore, a more recent retrospective study of 23 patients receiving IV calcium in the setting of digoxin toxicity has shown no increased risk of arrhythmia or mortality [14].

Lastly, it should be noted that albuterol and insulin were given along with the second dose of calcium. While these treatments may have played a role in correcting serum potassium, we attribute the reversal of ECG abnormalities to calcium administration due to the instantaneous effect noted on the bedside cardiac monitor and the fact that potassium lowering agents can take up to 30 min to take effect [15]. On the other hand, it would not be ethical to withhold medications in unstable patients and so this would have been

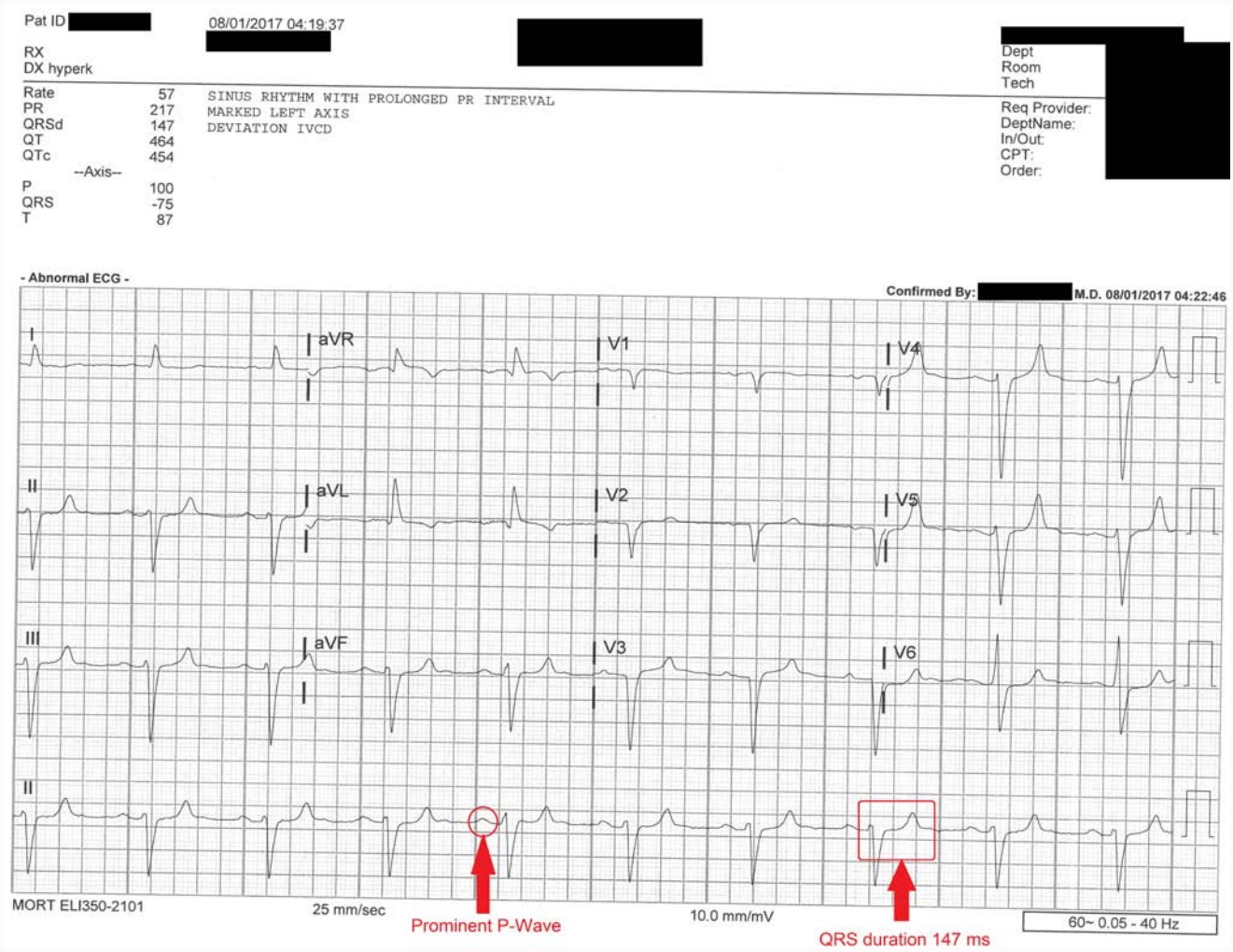

Figure 2: Electrocardiogram (third ECG).

unavoidable.

This case report describes a potentially fatal cardiac arrhythmia caused by hyperkalemia and its correction by intravenous calcium. Moreover, this report shows that repeated calcium administration may be necessary to correct ECG abnormalities.

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