

Case Report:

An Electrocardiographic Sign of Danger: Sinoventricular Rhythm and Sine Wave in Severe Hyperkalemia

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Abstract

Potassium is predominantly an intracellular cation, and it has an important role in determining the resting membrane potential of cells. Disruption of the potassium gradient across the cell membrane can result in impaired cellular functioning. This may affect a number of organs including the cardiovascular system resulting in various symptoms and arrhythmias. Hyperkalemia is a common clinical condition that can induce deadly cardiac arrhythmias and electrocardiographic manifestations vary from the classic sinoventricular rhythm to nonspecific repolarization abnormalities seen with mild elevations of serum potassium. We present a case of severe methamphetamine induced hyperkalemia and the pathophysiology and the treatment is then discussed. This is a case of 29-year-old patient, presenting with methamphetamine use, secondary kidney injury, severe hyperkalemia and sine-wave configuration on electrocardiogram (ECG).

Keywords :Hyperkalemia; Sinoventricular rhythm; Sine-wave; Electrocardiogram

Introduction

Potassium is predominantly an intracellular cation, and it has an important role in determining the resting membrane potential of cells. Disruption of the potassium gradient across the cell membrane can result in impaired cellular functioning. This may affect a number of organs including the cardiovascular system resulting in various symptoms and arrhythmias. Hyperkalemia is a common clinical condition that can induce deadly cardiac arrhythmias and electrocardiographic manifestations vary from the classic sinoventricular rhythm to nonspecific repolarization abnormalities seen with mild elevations of serum potassium. We present a case of severe methamphetamine induced hyperkalemia and the pathophysiology and the treatment is then discussed. This is a case of 29-year-old patient, presenting with methamphetamine use, secondary kidney injury, severe hyperkalemia and sine-wave configuration on electrocardiogram (ECG).

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Case Report

A 29-year-old man presented to the Emergency Department (ED) with nausea, occasional vomiting and progressive weakness over the past 3 days. He denies chest pain, abdominal pain, headache and fever. The patient admitted to recent methamphetamine use, but denied prior cardiac history. On physical examination, vital signs were notable for a pulse of 96 beats/min, blood pressure of 100/60 mmHg, and respiratory rate of 26 breaths/min. Pulse oximetry was 94% oxygen saturation on room air. The patient was in moderate distress. His chest examination was clear to auscultation and heart sounds were normal. The abdomen was soft and nontender, and neurologic examination was notable for mild diffuse weakness. An initial 12-lead electrocardiogram (ECG) demonstrated a regular, markedly widened QRS complexes with a sine-wave configuration and undiscernible P waves (Figure 1).

These ECG findings consistent with the sinoventricular rhythm that is a hallmark of severe hyperkalemia. The suspicion of hyperkalemia was confirmed by a serum potassium level of 9.2 mEq/L, and an arterial blood gas revealed a significant metabolic acidosis (pH 6.98, PO₂ 216, pCO₂ 22). The patient was diagnosed with methamphetamine-induced acute renal failure (creatinine 20.2 mEq/L) and was stabilized with calcium chloride, bicarbonate, insulin/glucose therapy, followed by emergency hemodialysis. We obtained serial ECGs, on which the QRS complex was noted to progressively narrow, and an ECG performed the day after admission revealed a return to sinus rhythm with normal configuration and duration of the QRS complexes (Figure 2). The patient was subsequently discharged in good condition after a 7-day hospitalization.

Discussion

Hyperkalemia triggers a progression of ECG changes, beginning with peaked T waves and PR prolongation.

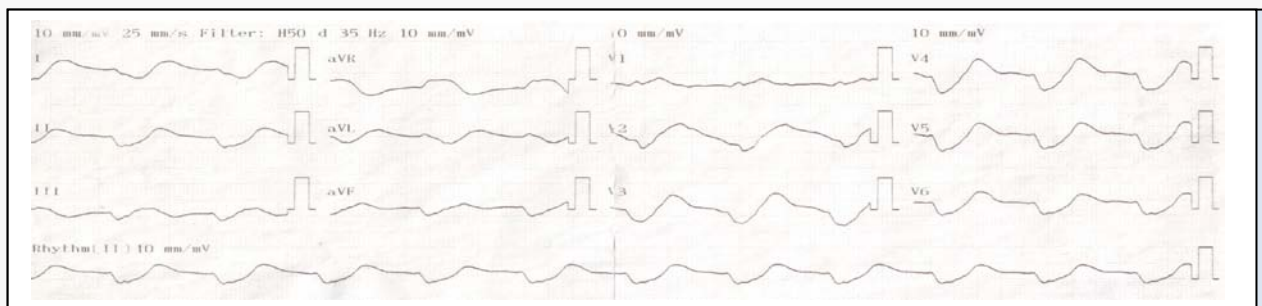


Figure 1: The 12-lead ECG obtained on presentation demonstrated a wide-complex rhythm, P waves are not discernible, and the QRS complexes are markedly widened, with a sine-wave morphology. The T wave has fused with the widening QRS complex to form the sine wave. This is the sinoventricular rhythm of severe hyperkalemia.

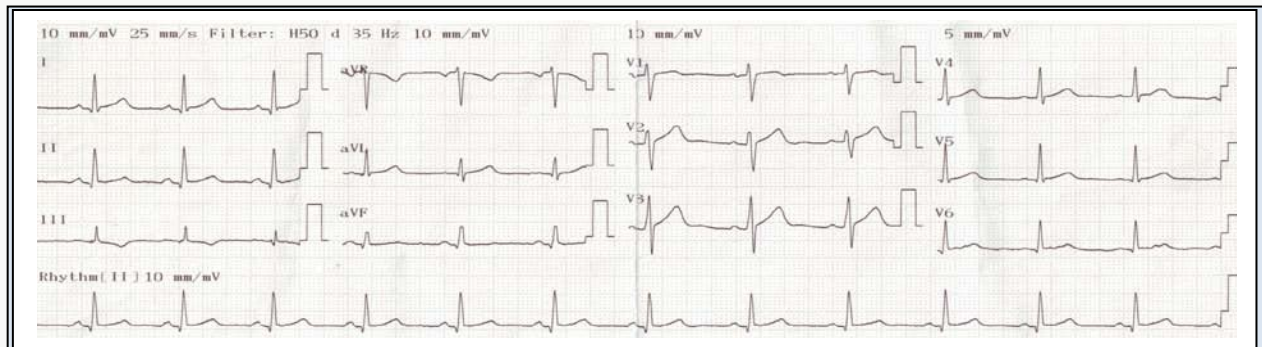


Figure 2: A repeat ECG performed the day after admission revealed sinus rhythm and QRS complexes with normal configuration and duration.

With further elevation of serum potassium, the P waves disappear, and QRS complexes become broad and bizarre in configuration, some-times resembling a sine wave. While the QRS rhythm might suggest a ventricular focus of origin, intracardiac electrocardiographic studies indicate that it might arise from the bundle of His, with marked intraventricular conduction delay, or from sinus node [1-3]. The latter rhythm is termed sinoventricular conduction and represents sinus rhythm, with the sinus impulses being transmitted via intra-atrial conduction tissue to the AV node and thence to the ventricles. Despite transmission of the sinus impulse through the atria, the atrial muscle fails to be depolarized because of the hyperkalemia. Because atrial depolarization does not occur, P waves are not inscribed on the surface ECG. This presentation demonstrated that early recognition of hyperkalemia-induced electrocardiographic sine wave and sinoventricular conduction can provide clues to diagnosis and is essential to successful treatment.

However, patients with severe hyperkalemia commonly have arrhythmias that may rapidly become fatal if unrecognized. Although laboratory tests are the gold standard for diagnosis of the serum potassium concentration, there may be delays in obtaining the results. The electrocardiogram may be a useful tool and can provide clues to the diagnosis, as well as guide for therapeutic interventions. Because hyperkalemia can lead to sudden death from cardiac arrhythmias, any suggestion of hyperkalemia requires an immediate ECG to ascertain whether electrocardiographic signs of electrolyte imbalance are present.

References

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