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**Case Series**

# Hypokalemia-induced arrhythmia: a case series

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## ABSTRACT

Potassium is one of the major intracellular electrolytes in the body and is normally maintained between 3.5 and 5.5 mEq/L. A serum K+ concentration below 3.5 mEq/L is considered hypokalemia. Electrolyte imbalances, particularly potassium disorders, are common in clinical practice. Potassium homeostasis plays a key role in regulating cell membrane excitability. Potassium is a major determinant of the electrophysiologic properties of the myocardial membrane, and it plays an important role in the occurrence of arrhythmia. Hypokalemia can lead to clinically significant, life-threatening arrhythmia. Typical electrocardiographic (ECG) features of hypokalemia include widespread ST depression, T wave inversion, and prominent U waves. However, hypokalemia may present with different types of arrhythmias as well. Herein, we present 3 cases presenting with hypokalemia-induced arrhythmias in different clinical scenarios with documented low potassium levels and treated with timely diagnosis and effective management.

**Keywords:** Hypokalemia, Potassium, Arrhythmia

## INTRODUCTION

The normal serum potassium concentration ranges from 3.5 to 5.3 mmol/L. Hypokalemia is considered severe if the serum potassium level is lower than 2.5 mmol/L. Mild hypokalemia is relatively uncommon and may go unnoticed for a long time, whereas severe hypokalemia (<2.5-3.0 mEq/L is usually asymptomatic and can be life-threatening.[1] Many have symptoms at levels higher than 2.5 due to the rapid decrease of serum K+ levels or other compounding factors, such as hypomagnesemia. Most common clinical manifestations are muscle weakness, cramps, cardiac arrhythmia, and rhabdomyolysis.

## CASE SERIES

### *Case 1: Supraventricular tachycardia due to hypokalemia*

A 60-year-old woman was admitted to the ICU for treatment for a lower respiratory tract infection with a complaint of chest discomfort and palpitation. She had a history of hypertension and chronic obstructive airway disease. On initial assessment, she was dyspneic with a blood pressure of 120/80 mmHg and an oxygen saturation of 93% on oxygen supplementation with nasal prongs and on diuretics and nebulization. The neurologic examination was normal without significant muscle weakness.

An electrocardiogram (ECG) showed changes of SVT. After synchronized cardioversion with 100J energy, the patient did not show any changes, and SVT persisted. The patient was then given intravenous metoprolol (10 mg) slowly over 10 minutes, and the patient showed marked clinical improvement. The ECG after stabilization showed sinus rhythm with features of hypokalemia. The patient was treated simultaneously with potassium correction via central venous access. The serum electrolyte had significant hypokalemia (K=2.4 mmol, normal=3.5-5.3 mmol/L) with borderline hypomagnesemia (Mg=1.7 mg/dL, normal=1.50-2.60 mg/dL). When the serum potassium level reached 4.56 mmol/L, the ECG was rechecked and showed the continued presence of ST depression and T inversion.[4,5,7]

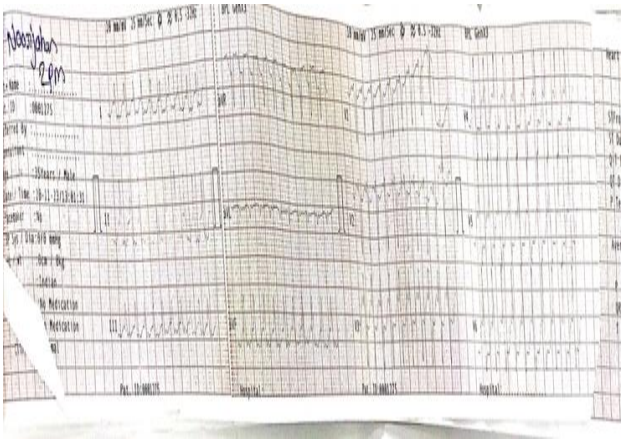

**Figure 1: ECG showing supraventricular tachycardia with heart rate of 194b pm.**

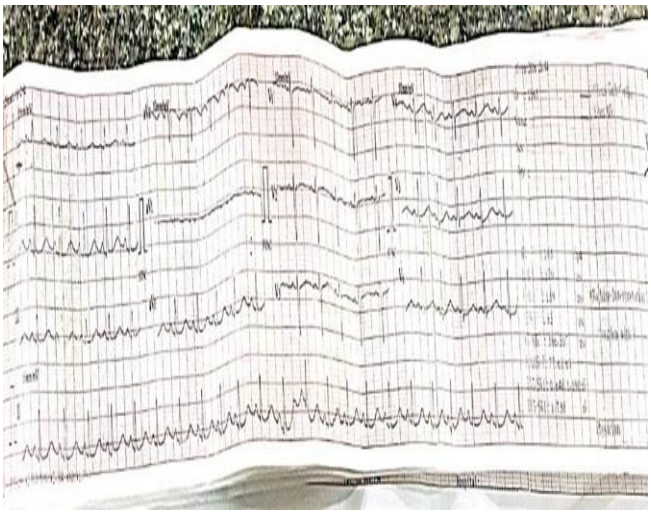

**Figure 2: Normal sinus rhythm with ST depression.**

***Case 2: Multifocal atrial tachycardia due to hypokalemia***

A 65-year-old man was in the ICU for treatment for a lower respiratory tract infection with complaints of breathlessness and coughing with expectoration. He was a known case of chronic obstructive airway disease. He had no history of chest pain or muscle weakness. He denied any history of previous heart disease, hypertension, diabetes, or kidney disease. He also had no history of taking regular medications, including inhalers. There was no family history of sudden cardiac death. On assessment, he was dyspneic with a blood pressure of 150/90 mmHg, a heart rate of 174 beats per minute (bpm)(figure.3), an oxygen saturation of 80 percent on face mask oxygen supplementation, and a temperature of 36.5°C. His glucose level was 112 mg/dL. A physical examination revealed tachycardia on auscultation. Abdominal and neurologic examinations were normal. There were no clinical features suggestive of hypothyroidism, hyperthyroidism, or Cushing's syndrome.[11] The initial ECG showed multifocal atrial tachycardia with a rate of 170 bpm. Vagal maneuvers were tried, but no response was elicited. Therefore, the patient was administered intravenous amiodarone 150 mg in normal saline 100 mL over 30 minutes. ECG rechecked after amiodarone infusion showed features of hypokalemia such as ST depression, a small T wave, prominent U waves, and a prolonged QT interval (Figure 4).[4,5] Later, the patient was started on diltiazem 30 mg TDS.

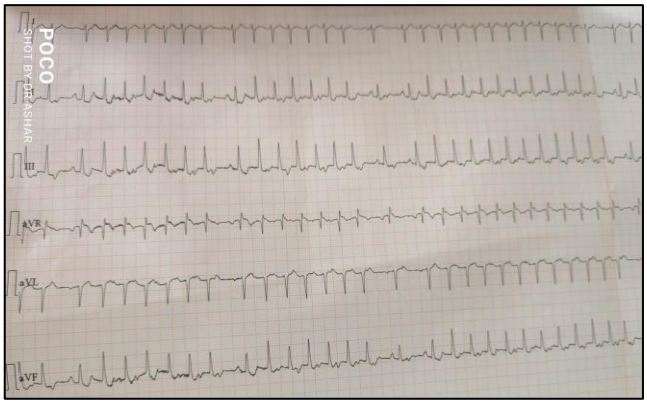


**Figure 3: ECG showing multifocal atrial tachycardia with heart rate of 174 bpm.**

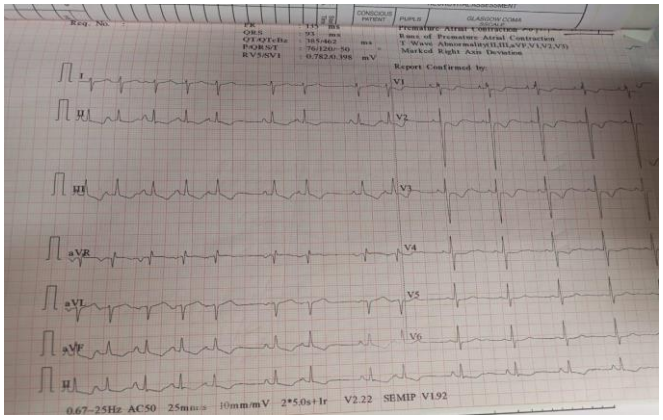

**Figure 4: ECG shows ST depression, a small T wave, prominent U waves.[4]**

***Case 3: Paroxysmal supraventricular tachycardia due to hypokalemia***

A 55-year-old man presented to the emergency department with palpitations since one day. He had no history of chest pain or breathlessness. There was a previous history of hypertension and diabetes on medications, including high doses of insulin and antihypertensives. On assessment, he had palpitations with a blood pressure of 130/90 mmHg, a heart rate of 196 beats per minute (bpm) (Figure 5), an oxygen saturation of 100% on room air, and a temperature of 36.5°C. A physical examination revealed normal first and second heart sounds with no added sound. Lungs were clear, and both abdominal and neurologic examinations were normal. The patient was administered intravenous adenosine (6 mg), followed by a normal saline flush, and the ECG showed no changes. A repeat injection of adenosine (6 mg) intravenously was given, and the ECG rechecked after a normal saline flush showed features of

hypokalemia such as ST depression, a small T wave, prominent U waves, and a prolonged QT interval with normal rhythm.[4,5,8] The patient's serum potassium level was significantly low (2.59 mmol/L), and borderline hypomagnesemia was also noted (Mg=1.5 mg/dL). After administering the first dose of intravenous potassium supplement (KCl 40 mmol), the ECG was rechecked, showing the appearance of a typical hypokalemic ECG pattern (Figure 6).[4,5,7] Further doses of intravenous potassium supplements were planned for administration to the patient. However, due to personal reasons, she wanted to be discharged against medical advice. He was given oral potassium and magnesium supplements and planned to follow-up to recheck the electrolyte results. Unfortunately, the patient also missed follow-up.

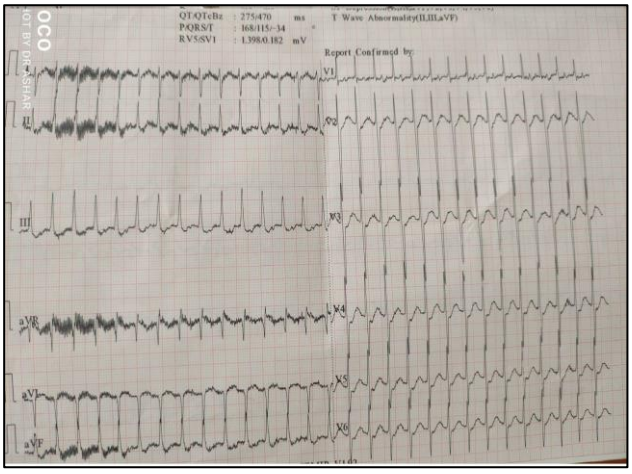

**Figure 5: ECG showing PSVT.**

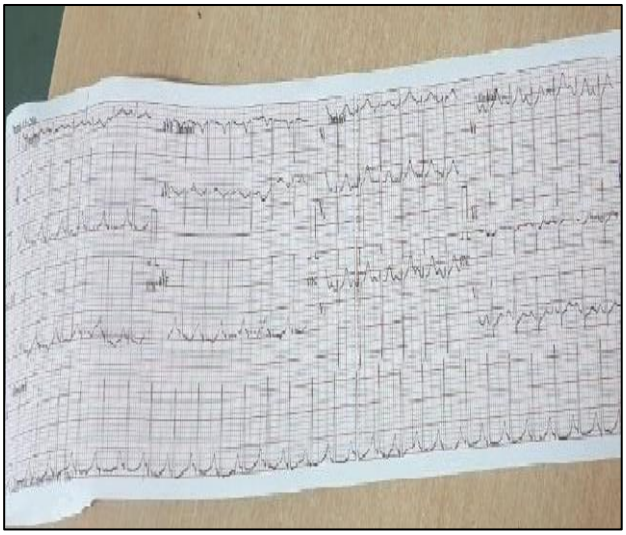

**Figure 6: Hypokalemic ECG pattern.**

## DISCUSSION

Typically, hypokalemia manifests as heart rhythm problems and muscle weakness. Three categories can be used to group the primary causes of hypokalemia: decreased dietary K+ intake, intracellular shift (caused by drugs or hormonal disorders), and increased loss (gastrointestinal or renal).[11] A variety of arrhythmias can occur in hypokalemic patients. Atrioventricular block, sinus bradycardia, paroxysmal atrial or junctional tachycardia, premature atrial complex, premature ventricular complex, and ventricular tachycardia or fibrillation are a few of these conditions. While not all individuals experience these distinctive ECGs, some might land up in cardiac arrest. Individuals who have hypokalemia may also have hypomagnesemia as a result of concomitant loss from diuretic medication, diarrhea, or renal potassium wasting if hypomagnesemia is the main abnormality. Patients of this type may not respond to potassium replacement therapy alone. Consequently, individuals with hypokalemia should have their serum magnesium levels measured, and if hypomagnesemia is already present, it should be treated.

The mechanism via which cardiac muscles contract is known as excitation-contraction coupling. The action potential (AP) originates in the sinoatrial node and travels throughout the heart via the Purkinje fibers, His bundle, and atrioventricular node.[9] The transcellular electrochemical gradient, which is mostly controlled by the opening and closure of different ion channels, determines the AP.[9] Arrhythmia can result from any disruptions in AP production and/or conduction. The concentration of potassium (K+) within cells is 30-40 times greater than that of potassium outside of cells. The sodium/potassium adenosine triphosphatase (ATPase) (Na+/K+ ATPase) electrophysiological activity of cell membranes tightly controls the serum K+ concentration within the range of 3.5-5.3 mmol/L. Na+/K+ ATPase is the primary Na+ efflux pathway in cardiac myocytes. Na+/K+ ATPase is a voltage- and energy-dependent cell membrane ion transporter that creates a net outward current by exchanging two extracellular K+ ions for three intracellular Na+ ions. The concentrations of extracellular K+, internal Na+, and membrane AP all support the action of Na+/K+ ATPase.[2,6]

Two different mechanisms-direct inhibition of K+ conductance and indirect suppression of Na+/K+ ATPase activity-are responsible for hypokalemia-induced arrhythmia.[10] The latter causes a buildup of Na+ inside the cell. Elevated intracellular Na+ causes the Na/Ca exchanger to cut down on the outgoing calcium (Ca+) current, which delays after-depolarization and increases intracellular Ca+ levels. Ca/calmodulin-dependent protein kinase II (CaMKII) is activated by cytosolic Ca+ through phosphorylation of the ryanodine receptor. Both the I-type Ca+ current (ICa-L) and the late Na+ current (INa) are stimulated by activated CaMKII.[10] It is this positive feedforward loop that raises intracellular Ca+ concentrations even more. Abnormalities related to pacemakers are linked to high Ca+. This ultimately leads to a modified repolarization mechanism and extended AP length, which in turn causes early and delayed after-depolarization and raises the possibility of different kinds of arrhythmia.

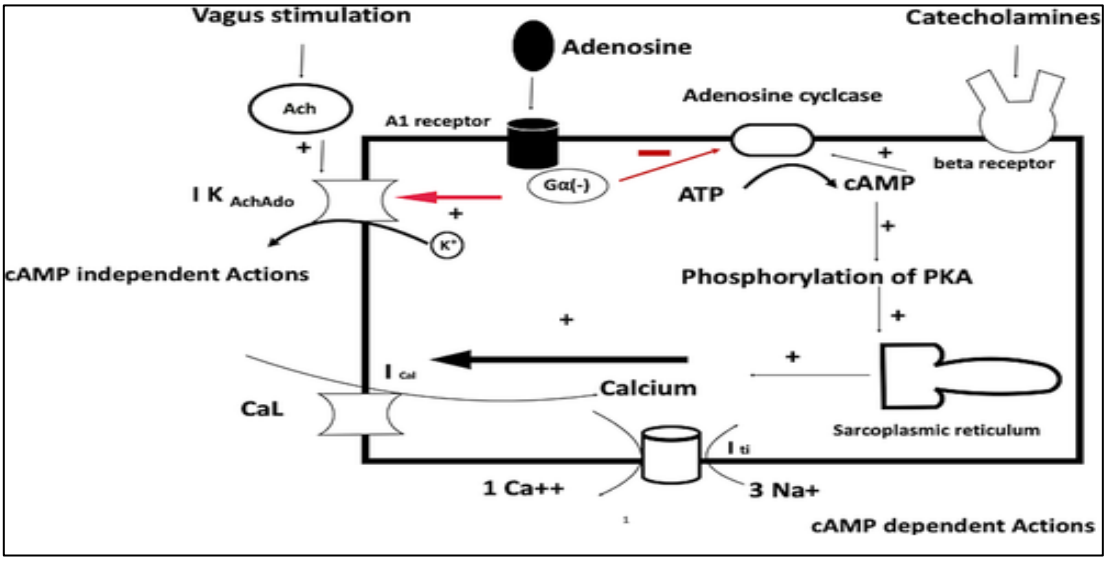


**Figure 7: Effect of adenosine on cardiac cells through A1 receptors can be either cAMP-dependent or independent. Activation of potassium channels (negative chronotropy and dromotropy) is cAMP-independent while inhibition of hyperpolarization-activated (funny $I_f$) current (phase 4 depolarization) and $I_{CaL}$ are cAMP-dependent (The figure is adapted from Lerman et al).[3]**

On the other hand, potassium reserve replenishment may not always be indicated by a normal serum potassium level following potassium replacement. It is possible that the patient is still experiencing intracellular potassium deficiency. Thus, it is advised to keep an eye on the serum potassium level for 24 hours, the ECG, and any indications of recurring hypokalemia in order to guarantee that a stable potassium level is reached.

When there is a lack of insulin and hyperosmolality, potassium is more likely to be released from cells, leading to either nonketotic hyperglycemia or diabetic ketoacidosis. Because of this, individuals may present with a severe potassium deficiency despite having normal or even higher serum potassium concentrations due to gastrointestinal and/or urine losses. Serum potassium will decrease toward the level suitable for the potassium shortage with the start of insulin therapy and fluid replacement. When the serum potassium content is 4.5 mEq/L or below, potassium supplementation is typically started.[12]

A small percentage of individuals with uncontrolled diabetes, such as 6% in one study, have more pronounced potassium loss and are hypokalemic when they first appear. These individuals need to be aggressively replaced with potassium (20 to 30 mEq/hour).[12] One way to do this is to add 40 to 60 mEq of potassium chloride to each liter of half-isotonic saline. Insulin therapy should be postponed until blood potassium is above 3.3 mEq/L to prevent hypokalemia-related problems such as respiratory muscle weakness and cardiac arrhythmias, as insulin will exacerbate the hypokalemia.[13]

The effect of adenosine on the SA node is shown in red. Adenosine leads to hyperpolarization (dotted line, red color) of the resting membrane potential; therefore, it decreases the phase 4 slope of diastolic depolarization. Excessive hyperpolarization can lead to sinus arrest (dotted line, brown color). It also inhibits hyperpolarization-activated (funny $I_f$) current (phase 4 depolarization) and $I_{CaL}$ (phase 1) as an indirect effect through cAMP inhibition in the presence of sympathetic stimulation.[8]

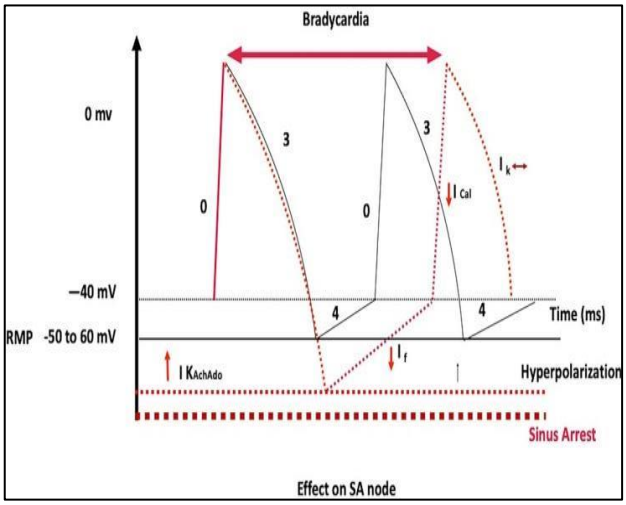


**Figure 8: The effect of adenosine on the SA node is shown in red. Adenosine leads to hyperpolarization (dotted line, red color) of the resting membrane potential; therefore, it decreases the phase 4 slope of diastolic depolarization. Excessive hyperpolarization can lead to sinus arrest (dotted line, brown color). It also inhibits hyperpolarization-activated (funny $I_f$) current (phase 4 depolarization) and $I_{CaL}$ (phase 1) as an indirect effect through cAMP inhibition in the presence of sympathetic stimulation.[3,8]**

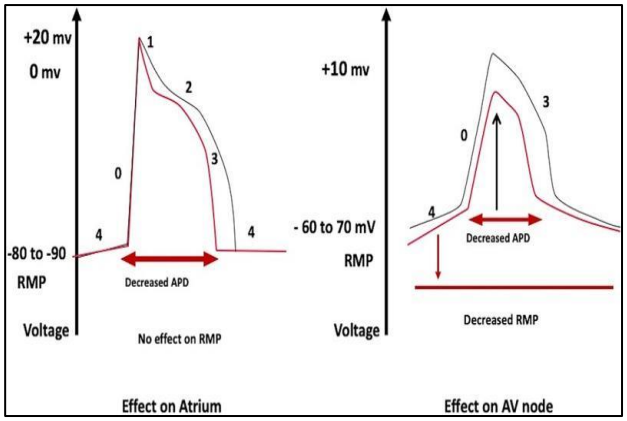


**Figure 9: Effect of adenosine on the atrium on the left and the atrioventricular (AV) node on the right. Adenosine decreases action potential duration (APD), hence the refractory period of the atrium without any effect on resting membrane potential (RMP), while decreasing the RMP (more negative) of the AV node without any effect on APD. (Red is the effect of adenosine, and black is control). (The figure is adapted from Lerman et al).[3,8]**

## CONCLUSION

Hypokalemia should be suspected in any patient presenting with arrhythmia. The early diagnosis of hypokalemia based on clinical presentation and changes in ECG is extremely important for timely intervention. Therefore, for the effective management of arrhythmia in acute care settings, clinicians should be familiar with different ECG manifestations of hypokalemia. Moreover, after potassium replacement, even with normal serum potassium levels, clinicians should be aware of intracellular potassium depletion, which may warrant monitoring for recurrent hypokalemia and further potassium supplementation.

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