CASE IN POINT

Following the Hyperkalemia Trail: A Case Report of ECG Changes and Treatment Responses

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Background: Hyperkalemia is an electrolyte disturbance with potentially severe consequences, including dysrhythmia and even asystole and death. Hyperkalemia can result from multiple factors, including impaired renal function, adverse drug reactions, adrenal disease, and heritable mutations. Although electrocardiogram (ECG) manifestations are well described, presentations can vary, and treatment response can be unpredictable. This case highlights dynamic ECG changes associated with severe hyperkalemia and the challenges associated with its management.

Case Presentation: An 81-year-old man with chronic kidney disease presented with muscle weakness and bradycardia. An ECG showed significant changes, including prolonged QRS duration and peaked T waves, corresponding to a serum potassium level of 9.8 mEq/L. The patient received calcium gluconate, albuterol, insulin, patiromer, and hemodialysis. However, fluctuating potassium levels required ongoing interventions.

Conclusions: This case underscores the importance of serial monitoring and dynamic treatment strategies in managing hyperkalemia, highlighting the critical role of ECG in guiding care.

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Hyperkalemia involves elevated serum potassium levels (> 5.0 mEq/L) and represents an important electrolyte disturbance due to its potentially severe consequences, including cardiac effects that can lead to dysrhythmia and even asystole and death.[1,2] In a US Medicare population, the prevalence of hyperkalemia has been estimated at 2.7% and is associated with substantial health care costs.[3] The prevalence is even more marked in patients with preexisting conditions such as chronic kidney disease (CKD) and heart failure.[4,5]

Hyperkalemia can result from multiple factors, including impaired renal function, adrenal disease, adverse drug reactions of angiotensin-converting enzyme inhibitors (ACEIs) and other medications, and heritable mutations.[6] Hyperkalemia poses a considerable clinical risk, associated with adverse outcomes such as myocardial infarction and increased mortality in patients with CKD.[5,7,8] Electrocardiographic (ECG) changes associated with hyperkalemia play a vital role in guiding clinical decisions and treatment strategies.[9] Understanding the pathophysiology, risk factors, and consequences of hyperkalemia, as well as the significance of ECG changes in its management, is essential for health care practitioners.

CASE PRESENTATION

An 81-year-old Hispanic man with a history of hypertension, hypothyroidism, gout, and CKD stage 3B presented to the emergency department with progressive weakness resulting in falls and culminating in an inability to ambulate independently. Additional symptoms included nausea, diarrhea, and myalgia. His vital signs were notable for a pulse of 41 beats/min. The physical examination was remarkable for significant weakness of the bilateral upper extremities, inability to bear his own weight, and bilateral lower extremity edema. His initial ECG upon arrival showed bradycardia with wide QRS, absent P waves, and peaked T waves (Figure 1a). These findings differed from his baseline ECG taken 1 year earlier, which showed sinus rhythm with premature atrial complexes and an old right bundle branch block (Figure 1b).

Medication review revealed that the patient was currently prescribed 100 mg allopurinol daily, 2.5 mg amlodipine daily, 10 mg atorvastatin at bedtime, 4 mg doxazosin daily, 112 mcg levothyroxine daily, 100 mg losartan daily, 25 mg metoprolol daily, and 0.4 mg tamsulosin daily. The patient had also been taking over-the-counter indomethacin for knee pain.

Based on the ECG results, he was treated with 0.083%/6 mL nebulized albuterol, 4.65 Mq/250 mL saline solution intravenous (IV) calcium gluconate, 10 units IV insulin with concomitant 50%/25 mL IV dextrose and 8.4 g of oral patiromer

TABLE 1. Laboratory Values Throughout Admission

Test	Day 1 17:28	Day 1 21:21	Day 2 05:47	Day 2 18:33	Day 3 05:21	Day 4 05:44	Day 5 05:27
Magnesium, mg/dL	1.9	—	—	—	—	1.7	—
Calcium, mg/dL	9.4	9.3	—	—	—	—	—
Creatinine, mg/dL	3.51	3.40	—	—	—	2.47	—
eGFR, mL/min/1.73 m^2	17	17	—	—	—	—	—
Sodium, mEq/L	138	141	140	—	138	137	—
Potassium, mEq/L	9.8	7.9	5.8	6.7	7.4	4.7	5.0
Chloride, mEq/L	115	119	109	—	113	104	—
CO_2, mEq/L	15.6	14.7	23.2	—	22.4	29.4	—
Glucose, mg/dL	104	—	—	—	—	92	—
Urea nitrogen, mg/dL	49.3	—	—	—	—	26.4	—

Abbreviations: CPK, creatine phosphokinase, eGFR, estimated glomerular filtration rate; HS, high sensitivity; SGOT, serum glutamic-oxaloacetic transaminase (aspartate aminotransferase); SGPT, serum glutamic pyruvic transaminase (alanine aminotransferase).

suspension. IV furosemide was held due to concern for renal function. The decision to proceed with hemodialysis was made. Repeat laboratory tests were performed, and an ECG obtained after treatment initiation but prior to hemodialysis demonstrated improvement of rate and T wave shortening (Figure 1c). The serum potassium level dropped from 9.8 mEq/L to 7.9 mEq/L (reference range, 3.5-5.0 mEq/L) (Table 1).

In addition to hemodialysis, sodium zirconium 10 g orally 3 times daily was added. Laboratory test results and an ECG was performed after dialysis continued to demonstrate improvement (Figure 1d). The patient's potassium level decreased to 5.8 mEq/L, with the ECG demonstrating stability of heart rate and further improvement of the PR interval, QRS complex, and T waves.

Despite the established treatment regimen, potassium levels again rose to 6.7 mEq/L, but there were no significant changes in the ECG, and thus no medication changes were made (Figure 1e). Subsequent monitoring demonstrated a further increase in potassium to 7.4 mEq/L, with an ECG demonstrating a return to the baseline of 1 year prior. The patient underwent hemodialysis again and was given oral furosemide 60 mg every 12 hours. The potassium concentration after dialysis decreased to 4.7 mEq/L and remained stable, not going above 5.0 mEq/L on subsequent monitoring. The patient had resolution of all symptoms and was discharged.

DISCUSSION

We have described in detail the presentation of each pathology and mechanisms of each treatment, starting with the patient's initial condition that brought him to the emergency room—muscle weakness. Skeletal muscle weakness is a common manifestation of hyperkalemia, occurring in 20% to 40% of cases, and is more prevalent in severe elevations of potassium. Rarely, the weakness can progress to flaccid paralysis of the patient's extremities and, in extreme cases, the diaphragm.

Muscle weakness progression occurs in a manner that resembles Guillain-Barré syndrome, starting in the lower extremities and ascending toward the upper extremities.[10] This is known as secondary hyperkalemic periodic paralysis. Hyperkalemia lowers the transmembrane gradient in neurons, leading to neuronal depolarization independent of the degree of hyperkalemia. If the degree of hyperkalemia is large enough, this depolarization inactivates voltage-gated sodium channels, making neurons refractory to excitation. Electromyographical studies have shown reduction in the compounded muscle action potential.[11] The transient nature of this paralysis is reflected by rapid correction of

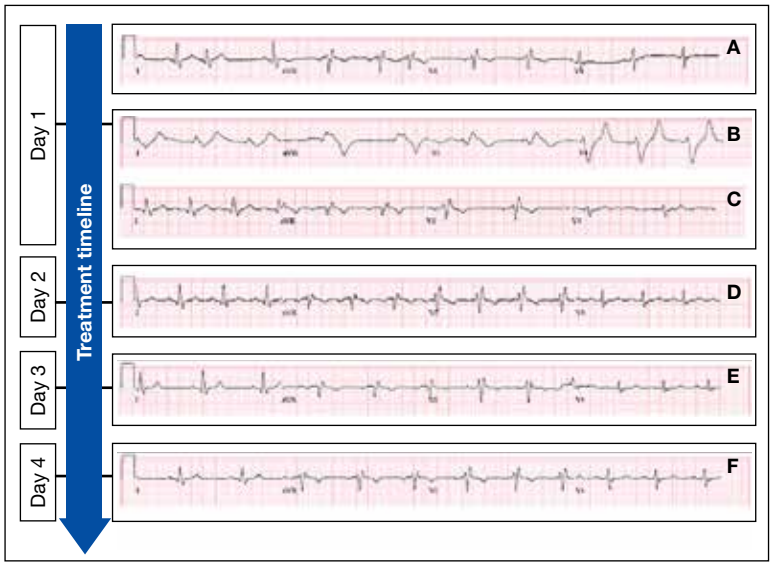

FIGURE 1. Comparison of Electrocardiograms Over Time

A, Baseline; B, Potassium level, 9.8 mEq/L, treated with nebulized albuterol, calcium gluconate, insulin, dextrose, and patiromer; C, Potassium level, 7.9 mEq/L, although there was improvement in electrocardiogram, the patient was scheduled for hemodialysis and continued with sodium zirconium 10 g orally 3 times daily, in the interim; D, Potassium level, 5.8 mEq/L after first hemodialysis, with resolution of patient's symptoms; E, Potassium level, 6.7 mEq/L. No medical management was provided; F, Potassium level, 7.0 mEq/L, no electrocardiogram changes noted, most likely because intracellular stores were depleted compared with arrival. The patient was treated with repeat hemodialysis and Lasix 60 mg.

weakness and paralysis when the electrolyte disorder is corrected.

The patient in this case also presented with bradycardia. The ECG manifestations of hyperkalemia can include atrial asystole, intraventricular conduction disturbances, peaked T waves, and widened QRS complexes. However, some patients with renal insufficiency may not exhibit ECG changes despite significantly elevated serum potassium levels.[12]

The severity of hyperkalemia is crucial in determining the associated ECG changes, with levels > 6.0 mEq/L presenting with abnormalities.[13] ECG findings alone may not always accurately reflect the severity of hyperkalemia, as up to 60% of patients with potassium levels > 6.0 mEq/L may not show ECG changes.[14] Additionally, extreme hyperkalemia can lead to inconsistent ECG findings, making it challenging to rely solely on ECG for diagnosis and monitoring.[8] The level of potassium that causes these effects varies widely through patient populations.

The main mechanism by which hyperkalemia affects the heart's conduction system is through voltage differences across the conduction fibers and eventual steady-state inactivation of sodium channels. This combination of mechanisms shortens the action potential duration, allowing more cardiomyocytes to undergo synchronized depolarization. This amalgamation of cardiomyocytes repolarizing can be reflected on ECGs as peaked T waves. As the action potential decreases, there is a period during which cardiomyocytes are prone to tachyarrhythmias and ventricular fibrillation.

A reduced action potential may lead to increased rates of depolarization and thus conduction, which in some scenarios may increase heart rate. As the levels of potassium rise, intracellular accumulation impedes the entry of sodium by decreasing the cation gradient across the cell membrane. This effectively slows the sinus nodes and prolongs the QRS by slowing the overall propagation of action potentials. By this mechanism, conduction delays, blocks, or asystole are manifested. The patient in this case showed conduction delays, peaked T waves, and disappearance of P waves when he first arrived.

Hyperkalemia Treatment

Hyperkalemia develops most commonly due to acute or chronic kidney diseases, as was the case with this patient. The patient's hyperkalemia was also augmented by the use of nonsteroidal anti-inflammatory drugs (NSAIDs), which can directly affect renal function. A properly functioning kidney is responsible for excretion of up to 90% of ingested potassium, while the remainder is excreted through the gastrointestinal (GI) tract. Definitive treatment of hyperkalemia is mitigated primarily through these 2 organ systems. The treatment also includes transitory mechanisms of potassium reduction. The goal of each method is to preserve the action potential of cardiomyocytes and myocytes. This patient presented with acute symptomatic hyperkalemia and received various medications to acutely, transitorily, and definitively treat it.

Initial therapy included calcium gluconate, which functions to stabilize the myocardial cell membrane. Hyperkalemia decreases

the resting membrane action potential of excitable cells and predisposes them to early depolarization and thus dysrhythmias. Calcium decreases the threshold potential across cells and offsets the overall gradient back to near normal levels.[15] Calcium can be delivered through calcium gluconate or calcium chloride. Calcium chloride is not preferred because extravasation can cause pain, blistering and tissue ischemia. Central venous access is required, potentially delaying prompt treatment. Calcium acts rapidly after administration—within 1 to 3 minutes—but only lasts 30 to 60 minutes.[16] Administration of calcium gluconate can be repeated as often as necessary, but patients must be monitored for adverse effects of calcium such as nausea, abdominal pain, polydipsia, polyuria, muscle weakness, and paresthesia. Care must be taken when patients are taking digoxin, because calcium may potentiate toxicity.[17] Although calcium provides immediate benefits it does little to correct the underlying cause; other medications are required to remove potassium from the body.

Two medication classes have been proven to shift potassium intracellularly. The first are β-2 agonists, such as albuterol/levalbuterol, and the second is insulin. Both work through sodium-potassium-ATPase in a direct manner. β-2 agonists stimulate sodium-potassium-ATPase to move more potassium intracellularly, but these effects have been seen only with high doses of albuterol, typically 4× the standard dose of 0.5 mg in nebulized solutions to achieve decreases in potassium of 0.3 to 0.6 mEq/L, although some trials have reported decreases of 0.62 to 0.98 mEq/L.[15,18] These potassium-lowering effects of β-2 agonist are modest, but can be seen 20 to 30 minutes after administration and persist up to 1 to 2 hours. β-2 agonists are also readily affected by β blockers, which may reduce or negate the desired effect in hyperkalemia. For these reasons, a β-2 agonist should not be given as monotherapy and should be provided as an adjuvant to more independent therapies such as insulin. Insulin binds to receptors on muscle cells and increases the quantity of sodium-potassium-ATPase and glucose transporters. With this increase in influx pumps, surrounding tissues with higher resting membrane potentials can absorb the potassium load, thereby protecting cardiomyocytes.

Potassium Removal

Three methods are currently available to remove potassium from the body: GI excretion, renal excretion, and direct removal from the bloodstream. Under normal physiologic conditions, the kidneys account for about 90% of the body's ability to remove potassium. Loop diuretics facilitate the removal of potassium by increasing urine production and have an additional potassium-wasting effect. Although the onset of action of loop diuretics is typically 30 to 60 minutes after oral administration, their effect can last for several hours. In this patient, furosemide was introduced later in the treatment plan to manage recurring hyperkalemia by enhancing renal potassium excretion.

Potassium binders such as patiromer act in the GI tract, effectively reducing serum potassium levels although with a slower onset of action than furosemide, generally taking hours to days to exert its effect. Both medications illustrate a tailored approach to managing potassium levels, adapted to the evolving needs and renal function of the patient. The last method is using hemodialysis—by far the most rapid method to remove potassium, but also the most invasive. The different methods of treating hyperkalemia are summarized in Table 2. This patient required multiple days of hemodialysis to completely correct the electrolyte disorder. Upon discharge, the patient continued oral furosemide 40 mg daily and eventually discontinued hemodialysis due to stable renal function.

Often, after correcting an inciting event, potassium stores in the body eventually stabilize and do not require additional follow-up. Patients prone to hyperkalemia should be thoroughly educated on medications to avoid (NSAIDs, ACEIs/ARBs, trimethoprim), an adequate low potassium diet, and symptoms that may warrant medical attention.[19]

CONCLUSIONS

This case illustrates the importance of recognizing the spectrum of manifestations of

TABLE 2. Medications Used to Remove Potassium From Bloodstream

Method/medication	Mechanism of action	Onset of action	Duration of action	Elimination
Calcium gluconate/ chloride	Stabilizes cardiac cell membranes by antagonizing the effects of hyperkalemia on the heart	Immediate (within minutes)	30-60 min	None
Insulin with glucose	Drives potassium into cells by increasing activity of the Na+/K+ ATPase pump	15-30 min	4-6 h	Transitory
Nebulized β-2 agonist	Stimulates cellular uptake of potassium via the Na+/K+ ATPase pump	30 min	2-4 h	Transitory
Sodium bicarbonate	Alkalinizes the blood, promoting potassium shift into cells, especially in patients with acidosis	30-60 min	2-6 h	Transitory
Loop diuretics	Increases renal excretion of potassium by inhibiting sodium reabsorption in the kidneys	1-2 h	4-6 h	Definitive
Sodium polystyrene sulfonate	Cation exchange resin that binds potassium in the gastrointestinal tract for excretion	Variable, hours to days	Variable, 6-24 h	Definitive
Patiromer	Nonabsorbed polymer that binds potassium in gastrointestinal tract for excretion	4-7 h	24 h	Definitive
Sodium zirconium cyclosilicate	Selective potassium binder that exchanges sodium and hydrogen ions for potassium in the gastrointestinal tract	Starts within 1 h	Not systematically absorbed and excreted fecally	Definitive
Hemodialysis	Directly removes potassium from the blood through filtration	Immediate (within minutes)	Variable	Definitive

hyperkalemia, which ranged from muscle weakness to cardiac dysrhythmias. Management strategies for the patient included stabilization of cardiac membranes, potassium shifting, and potassium removal, each tailored to the patient's individual clinical findings.

The case further illustrates the critical role of continuous monitoring and dynamic adjustment of therapeutic strategies in response to evolving clinical and laboratory findings. The initial and subsequent ECGs, alongside laboratory tests, were instrumental in guiding the adjustments needed in the treatment regimen, ensuring both the efficacy and safety of the interventions. This proactive approach can mitigate the risk of recurrent hyperkalemia and its complications.

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Ethics and consent
Verbal informed consent was provided by the patient in accordance with Veterans Affairs Caribbean Healthcare System protocol.

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