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Moderate Hyperkalemia Presenting As Sinus Arrest With Junctional Escape Rhythm From A Hospital With Resources Limited Setting: A Case Report And Narrative Review

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Abstract

Hyperkalemia is a potentially life-threatening electrolyte disturbance that can cause severe cardiac conduction abnormalities, even at moderate serum potassium levels, particularly in elderly patients with underlying cardiovascular disease. Management of acute hyperkalemia remains challenging, especially in resource-limited healthcare settings where advanced therapies such as urgent dialysis may not be readily available. We report a 77-year-old woman who presented with generalized weakness and recurrent syncope. Electrocardiography revealed sinus arrest with a junctional escape rhythm, and laboratory evaluation showed moderate hyperkalemia (6.84 mmol/L) accompanied by acute kidney injury, anemia, and mild metabolic acidosis. The patient had a history of anterior ST-elevation myocardial infarction and was receiving potassium-retaining medications. She was admitted to the intensive care unit and treated with protocol-based glucose–insulin therapy, intravenous calcium gluconate, adjunctive β-adrenergic agonists, and supportive care, without immediate access to dialysis. Serial ECGs demonstrated recovery of sinus rhythm, and serum potassium levels gradually normalized. This case illustrates that moderate hyperkalemia can precipitate severe bradyarrhythmias such as sinus arrest. Early recognition of ECG changes and prompt, protocol-driven management can result in favorable outcomes, even in resource-limited settings without immediate dialysis availability.

Keywords: Hyperkalemia; Sinus arrest; Junctional escape rhythm; Bradyarrhythmia; Resource-limited setting; Electrocardiography

INTRODUCTION

Hyperkalemia, defined as an increase in serum potassium (K^+) concentration to greater than 5.0 or greater than 5.5 mEq/L (mmol/L), is an electrolyte disorder that may lead to life-threatening consequences. The risk of hyperkalemia is increased in patients with chronic kidney disease, diabetes, heart failure, and in those receiving renin–angiotensin–aldosterone system inhibitors (Sarnowski et al., 2022). Although many patients are asymptomatic, hyperkalemia may manifest as muscle weakness, with early symptoms including paresthesia and muscle fasciculations in the upper and lower extremities, which can progress to paralysis, cardiac conduction abnormalities, and potentially fatal arrhythmias (Lindner et al., 2020; Chiu et al., 2023). In the general outpatient population, hyperkalemia is rare, with a prevalence ranging from 1.3% to 3.3%, whereas its prevalence ranges between 3.5% and 11% in hospitalized patients or in pooled outpatient and inpatient population (Tetti et al., 2024).

Elevated serum potassium levels in the blood can be detected through a surface electrocardiogram (ECG) (Harmon et al., 2024). Electrocardiographic manifestations of hyperkalemia progress with severity, beginning in mild cases (K^+ 5.5–6.5 mEq/L) with tall peaked T waves and fascicular blocks, advancing in moderate hyperkalemia (K^+ 6.5–7.5 mEq/L) to first-degree atrioventricular block, decreased or absent P waves, sinus arrest, and ST-segment changes, and culminating in severe hyperkalemia (K^+ >7.5 mEq/L) with intraventricular conduction delay, atypical bundle branch block, ventricular arrhythmias including ventricular tachycardia, ventricular fibrillation, idioventricular rhythm, "sine-wave" patterns, and asystole, sometimes mimicking Brugada syndrome, with variable cardiac effects that most prominently affect atrial myocardial cells before other conduction tissues (Campese & Adenuga, 2011).

Guidelines/consensus statements for hyperkalemia management have been developed (Palmer et al., 2021). Hyperkalemia management lacks standardized definitions, treatment thresholds and consistent guidelines and laboratory practices. Acute hyperkalemia requires immediate treatment to stabilize the patient and lower K^+ levels using agents such as calcium gluconate, insulin with glucose, and potassium-excreting therapies, while chronic management involves potassium binders or kidney replacement therapy in severe cases (AlSahow et al., 2024). However, in resource-limited hospitals, restricted access to advanced interventions can pose challenges and increase the risk of life-threatening complications. Therefore, prompt recognition and protocol-driven treatment are essential in such settings (Rossignol et al., 2016).

Here, we present a case of an elderly patient who developed sinus arrest with a junctional escape rhythm secondary to moderate hyperkalemia, successfully managed without immediate dialysis availability. This report aims to highlight the clinical impact of moderate hyperkalemia on cardiac conduction and emphasize the importance of effective management in resource-limited environments.

RESULTS AND DISCUSSION

A 77-year-old female patient presented to the emergency department with a 2-day history of generalized weakness and fatigue. According to her family, she had several brief episodes of apparent loss of consciousness at home. The symptoms were accompanied by nausea and vomiting. She denied shortness of breath, chest pain, or palpitations. During observation in the emergency department, the patient experienced two episodes of syncope. The patient had a history of anterior STEMI with late onset, left ventricular ejection fraction (LVEF) of 53%, mild mitral and tricuspid regurgitation, and anemia in one year earlier. Her prior medications included spironolactone 25 mg/day, isosorbide dinitrate 4 mg three times daily, atorvastatin 20 mg/day, amlodipine 10 mg/day, bisoprolol 5 mg/day, candesartan 16 mg/day, clopidogrel 75 mg/day, etabion tablets, and lansoprazole 30 mg/day.

On presentation, her Glasgow Coma Scale (GCS) score was E4M6V5. Her vital signs were as follows: blood pressure, 100/60 mmHg; pulse rate, 50–60 beats per minute; respiratory rate, 20 breaths per minute; body temperature, 37.8 °C; and oxygen saturation, 98% on room air. Physical examination revealed jugular venous pressure (JVP) approximately 2 cm above the sternal angle, normal heart sounds and clear lung fields, with no audible S3 gallop or murmurs, and no signs of congestion. The conjunctiva appeared pale, and the lower extremities were noted to be cold to the touch.

The patient's ECG showed sinus arrest with junctional escape rhythm (**Figure 1**). Routine laboratory investigations (**Table 1**) revealed moderate anemia with hemoglobin of 8.7 g/dL with other parameters within normal limits. There was an elevation in liver enzymes, with SGOT 188.1 U/L and SGPT 138.6 U/L, as well as a mild increase in kidney function markers, with urea 60.9 mg/dL and creatinine 1.6 mg/dL. Blood sugar was within the normal range (124 mg/dL). Electrolyte examination showed hyperkalemia (6.84 mmol/L), while other parameters: sodium 145.67 mmol/L and chloride 117.18 mmol/L. Arterial blood gas analysis indicated mild metabolic acidosis with partial respiratory compensation and mild hypoxemia (**Table 2**). Chest X-ray examination revealed early signs of pulmonary edema characterized by the stag's antler sign, with bilateral perihilar and right pericardial infiltrates suggestive of bronchopneumonia, as well as findings of cardiomegaly, left ventricular hypertrophy (LVH), and aortic sclerosis (**Figure 2)**.

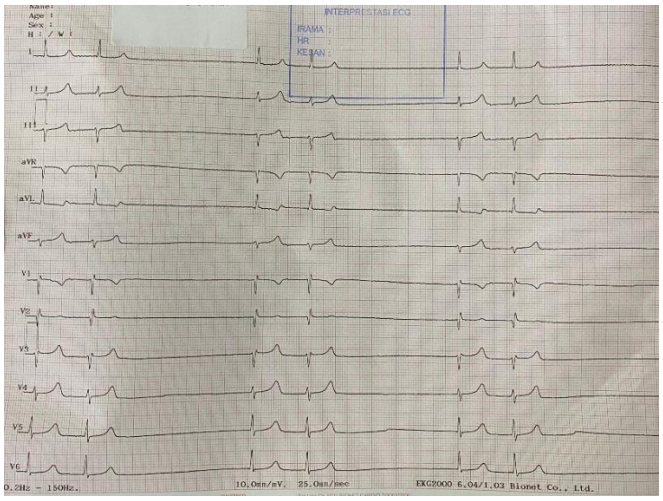

Figure 1. 12-Lead electrocardiogram on admission showed sinus arrest with junctional escape rhythm.

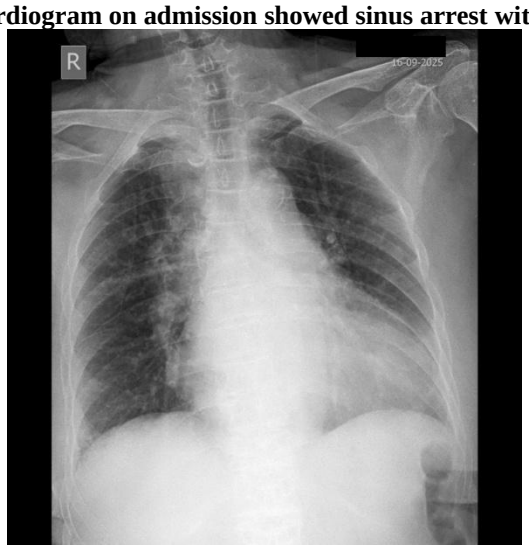

Figure 2. Chest X-ray showing early pulmonary edema, bronchopneumonia, cardiomegaly, left ventricular hypertrophy (LVH), and aortic sclerosis.

Table 1. Laboratory findings on arrival at the emergency room (ER) 16/9/2025

Parameters	**Results**
Hb	8.7 g/dL
Hematocryte	27.2 %
Leukocyte	9.5 /mm^3
Granulocytes	64.4%
Lymphocytes	26.8A%
MID	8.8%
Ureum	60.9 mg/dL
Creatinine	1.6 mg/dL
SGOT	188.1U/L
SGPT	138.6 U/L
Blood glucose	124 mg/dL

Table 2. Blood Gas Analysis findings on arrival at the emergency room (ER) 16/9/2025

Parameter	Result
Temperature	36 C
sO2 (est)	96%
pH	7.37
pC02 (T)	32.2 mmHg
pO2 (T)	78 mmHg
HCO3-Act	18.6 mmol/L
CtCO2	20 mmol/L
BE (ecf)	-6.8 mmol/L
BE (B)	-6.1 mmol/L
HCO3-Std	19.4 mmol/L
pO2(A-a)	77 mmHg
RI(T)	1.08
pO_2/FiO_2	280 mmHg

The patient was diagnosed with post-syncope secondary to sinus arrest with a junctional escape rhythm due to moderate hyperkalemia. Comorbidities included anemia, hepatic insufficiency, acute kidney injury, bronchopneumonia (CURB-65 score of 3; PSI score 87), and a history of late-presentation anterior STEMI with preserved systolic function (EF 53%) and mild mitral and tricuspid regurgitation. The patient was admitted to the intensive care unit (ICU) for continuous cardiac monitoring and prompt management.

Upon admission to the ICU, the patient continued to demonstrate sinus arrest with a junctional escape rhythm, requiring hemodynamic support. An intravenous dopamine infusion was initiated at 5 µg/kg/min and subsequently titrated based on clinical response to maintain a heart rate above 50 beats per minute. Due to persistent bradycardia during the first 24 hours of care, the dopamine infusion was gradually increased to 8 µg/kg/min. On the second day of ICU hospitalization, electrocardiographic monitoring showed resolution of sinus arrest, with a junctional rhythm replaced by sinus bradycardia (**Figure 3**). As a result, the dopamine infusion was progressively tapered off while maintaining close rhythm surveillance. Empiric intravenous ceftriaxone 2 g once daily was administered to treat suspected bronchopneumonia. Additional supportive medications included intravenous paracetamol 500 mg every 8 hours, intravenous lansoprazole 30 mg every 12 hours, intravenous ondansetron 4 mg every 12 hours. Aspirin 80 mg daily and atorvastatin 40 mg daily were also continued during hospitalization.

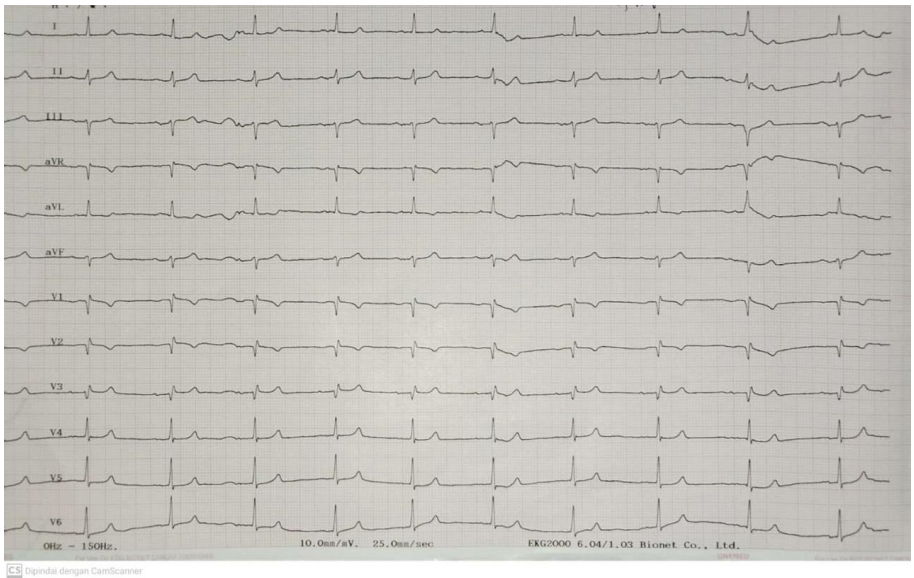

Figure 3. Day-2 ECG showing sinus bradycardia (HR ~54 bpm), T-wave depression in V1–V2, and tall T-waves, indicating recovery from prior sinus arrest with junctional escape rhythm.

Hyperkalemia was aggressively managed in the ICU following local hospital protocols with repeated cycles of Glucose–Insulin Correction therapy (GICT) consisting of 40% dextrose 50 mL plus 10 units of regular insulin per cycle, along with intravenous calcium gluconate 10 mL of 10% solution for cardiac membrane stabilization. Capillary blood glucose levels were monitored before and after each cycle to prevent hypoglycemia. By day three, adjunctive therapy was added, including nebulized salbutamol every 8 hours and intravenous furosemide 20 mg daily to support further potassium reduction. Serial laboratory tests showed a gradual decline in serum potassium from 6.84 mmol/L on admission to 5.32 mmol/L by hospital day four.

Table 3. Serial Electrolyte Levels, Glucose Shifts, and Therapeutic Interventions During ICU Care

ICU Day & Time	Electrolytes (mmol/L)	Therapy	Glucose Monitoring (mg/dL)
Day 1 (19.30)	K^+ 6.84 Na^+ 145.67 Cl^- 117.18	3 cycles G/I therapy + 10 ml of 10% calcium gluconate	Cycle 1: 193 → 290 Cycle 2: 185 → 265 Cycle 3: 125 → 274
Day 2 (05.00)	K^+ 6.85 Na^+ 146.32 Cl^- 116.70	3 cycles G/I therapy + 10 ml of 10% calcium gluconate	Cycle 1: 111 → 210 Cycle 2: 159 → 207 Cycle 3: 167 → 256
Day 2 (17.00)	K^+ 6.37 Na^+ 145.67 Cl^- 115.49	2 cycles G/I therapy	Cycle 1: 112 → 128 Cycle 2: 119 → 254
Day 2 (23.00)	K^+ 6.07 Na^+ 146.64 Cl^- 116.23	3 cycles G/I therapy	Cycle 1: 111 → 195 Cycle 2: 133 → 200 Cycle 3: 108 → 193
Day 3 (11.00)	K^+ 6.38 Na^+ 145.68 Cl^- 115.58	3 cycles G/I therapy + 10 ml of 10% calcium gluconate + furosemide 20 mg IV daily + nebulized salbutamol q8h	Cycle 1: 127 → 438 Cycle 2: 241 → 436 Cycle 3: 127 → 301
Day 3 (20.00)	K^+ 5.85 Na^+ 145.38 Cl^- 113.06	2 cycles G/I therapy + 10 ml of 10% calcium gluconate	Cycle 1: 150 → 201 Cycle 2: 225 → 204
Day 4 (05.00)	K^+ 5.32 Na^+ 145.07 Cl^- 112.33	1 cycles G/I therapy	Cycle 1: 95 → 171

On the fourth day of ICU care, the patient demonstrated marked clinical improvement, with stabilization of serum potassium levels and resolution of syncopal episodes. Electrocardiographic evaluation showed restoration of a stable sinus rhythm with sinus bradycardia and no recurrence of sinus arrest (**Figure 4**). She was subsequently transferred to the general ward on hospital day five and was discharged in stable condition on day six, following a total of four days in the ICU and two days in the ward. Her discharge medications included lansoprazole 30 mg once daily, aspirin 80 mg once daily, atorvastatin 40 mg once daily, paracetamol 500 mg three times daily, and furosemide 40 mg once daily. A cardiology follow-up appointment was scheduled within one week after discharge.

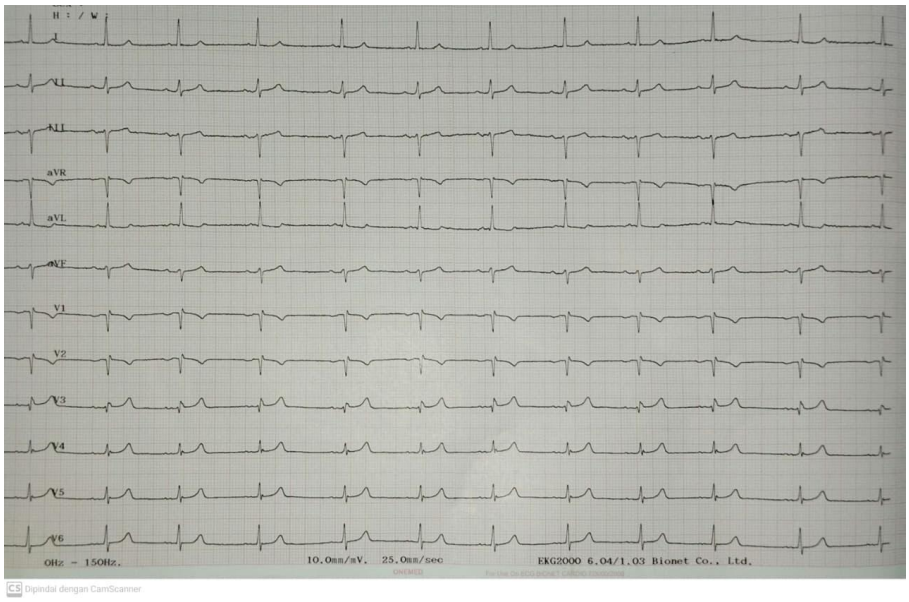

Figure 4. Day-4 ECG showing sinus bradycardia (HR 56 bpm)

Discussion

This case highlights the critical need for prompt identification of hyperkalemia-related ECG findings, as delayed recognition in acute settings may lead to severe and potentially life-threatening consequences. Potassium levels within the normal range of 3.5–5.4 mmol/L are vital for normal cardiac function, whereas hyperkalemia is often asymptomatic but may progress to cardiac arrhythmias and fatal outcomes when severe (Chiu et al., 2023). In this case, the patient demonstrated sinus arrest with a junctional escape rhythm despite a serum potassium level of 6.84 mmol/L on admission, categorized as moderate hyperkalemia. The presence of structural heart disease such as prior myocardial infarction likely contributed to increased vulnerability to conduction failure. This aligns with previous observations that comorbidities can lower the threshold at which hyperkalemia produces clinically significant arrhythmias.

Hyperkalemia is characterized by an increased potassium level, although there is no universally accepted definition. It is most often defined as a potassium concentration ≥5.5 mmol/L, but this threshold may vary across laboratories and between plasma and serum measurements (Linder et al., 2020). Hyperkalemia generally arises from increased potassium intake, reduced renal excretion, or transcellular shifts of potassium from the intracellular to the extracellular space, however, excessive intake alone rarely causes hyperkalemia due to the high excretory capacity of healthy kidneys. Major risk factors include impaired renal function reflected by a reduced estimated glomerular filtration rate, higher baseline potassium levels, and comorbid conditions such as diabetes mellitus, heart failure, and coronary artery disease. In the emergency department setting, additional important risk factors include rhabdomyolysis, particularly following major soft tissue trauma, as well as electrical burns and combat-related injuries. Elevated potassium levels have also been associated with anemia, strenuous physical activity, and the use of various medications, notably potassium-sparing diuretics, mineralocorticoid receptor antagonists (e.g., spironolactone and eplerenone), angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, direct renin inhibitors, β-blockers, nonsteroidal anti-inflammatory drugs, heparin, and penicillin G (Lindner et al., 2020; Sevamontree et al., 2024; Massicotte-Azamiouch et al., 2023). In this case, hyperkalemia was most likely precipitated by drug-induced potassium retention due to the combined use of spironolactone and an angiotensin receptor blocker in the setting of advanced age and reduced renal function, with additional contribution from mild metabolic acidosis and underlying structural heart disease

Although many patients remain asymptomatic, hyperkalemia can present with muscle weakness, with paresthesias and muscle fasciculations in the extremities as early manifestations; in severe cases,

it may progress to paralysis, cardiac conduction disturbances, and potentially fatal arrhythmias (Ortiz-Cortés C et al., 2025). Hyperkalemia-induced cardiotoxicity results from increased resting membrane potential, reduced depolarization, and shortening of the depolarization duration, which collectively decrease sodium channel availability and slow the phase 0 upstroke of the action potential (Vmax), leading to impaired impulse conduction manifested as PR interval prolongation and QRS widening. Concurrently, enhanced potassium conductance, particularly through IKr channels during phases 2 and 3 of the action potential, accelerates potassium efflux from myocytes and shortens repolarization, producing characteristic ECG changes such as peaked T waves and ST–T segment depression. As serum potassium levels continue to rise, conduction through the sinoatrial (SA) and atrioventricular (AV) nodes becomes impaired, predisposing to escape rhythms, especially junctional rhythms at potassium concentrations around 6.5 mmol/L. With more severe hyperkalemia, the atrial myocardium, which is more sensitive to elevated potassium than the SA node, ventricular myocardium, and His bundle, becomes electrically unexcitable, resulting in sinoatrial block and loss of P waves. At potassium levels of approximately 8–9 mmol/L, the SA node may directly stimulate the ventricles without atrial depolarization, producing a sino-ventricular rhythm; although sinoatrial block typically occurs at potassium levels above 8 mmol/L, it has also been reported in cases of mild hyperkalemia (around 5.5 mmol/L) (Littman, 2023; Suwari et al., 2021; Quintero et al., 2024).

High-quality studies on acute hyperkalemia management are lacking, consequently, no international guideline specifically focused on the management of hyperkalemia exists to date (Lindner et al., 2020). Treatment of hyperkalemia aims to stabilize excitable cell membranes with intravenous calcium, promote intracellular potassium shifting using insulin–glucose or β-adrenergic agonists such as albuterol, and enhance potassium removal through diuretics, exchange resins, or dialysis (Littman, 2023).

Appropriate treatment of hyperkalemia is essential to prevent fatal arrhythmias. Intravenous calcium administration stabilizes the cardiac cell membrane and improves cardiac manifestations, and should be initiated as first-line therapy in the presence of hyperkalemia-associated cardiotoxicity. The European Resuscitation Council recommends administering 10 mL of 10% calcium chloride over 2–5 minutes in hyperkalemic patients with ECG changes, with an onset of action within 1–3 minutes. The dose may be repeated after 5–10 minutes if there is no clinical response, and repeated dosing may be required if cardiac abnormalities initially resolve but subsequently recur (Lemoine et al., 2021). Intravenous calcium gluconate administration should be avoided in patients receiving digitalis, as several cases of sudden cardiac death have been reported (Suwari et al., 2021).

Potassium correction therapy aims to manage hyperkalemia by promoting an intracellular shift of potassium ions through enhanced Na^+/K^+-ATPase activity, which actively transports potassium from the extracellular space into the cell in exchange for sodium, utilizing ATP against the concentration gradient (Sebastian et al., 2024). Because calcium salts do not lower serum potassium levels, additional therapies are required to shift potassium from the extracellular to the intracellular space, most commonly insulin and β-adrenergic agonists. Intravenous short-acting insulin at a dose of 10 units has been shown to effectively reduce potassium levels with efficacy comparable to 20 units but with a lower risk of hypoglycemia, therefore intravenous glucose 25–50 g should be administered concurrently and blood glucose levels should be closely monitored. β-Adrenergic agonists such as salbutamol may be used as an alternative or adjunct to insulin, with both intravenous and nebulized routes demonstrating effectiveness, and nebulized salbutamol at a dose of 10 mg producing a significant reduction in serum potassium with a peak effect approximately 120 minutes after administration. Sodium bicarbonate may also contribute to potassium lowering by activating the Na^+/K^+-ATPase and correcting metabolic acidosis, although evidence regarding its efficacy is conflicting, and its use is generally recommended only in patients with metabolic acidemia who can tolerate the sodium load (Lindner et al., 2020).

Potassium can be removed from the body only through potassium-binding agents, loop diuretics, and dialysis. Loop diuretics are commonly used in acute hyperkalemia, particularly in patients with

volume overload, although evidence for their potassium-lowering efficacy remains limited. Data on potassium-binding agents in the acute setting are also scarce; however, newer agents such as patiromer and sodium zirconium cyclosilicate have shown promising potassium-lowering effects, with sodium zirconium cyclosilicate demonstrating a relatively rapid onset of action. Dialysis is the most effective modality for potassium removal and should be considered in cases of hyperkalemia that are refractory to first-line therapy or when ECG abnormalities persist despite calcium administration, especially in the presence of acute kidney injury. Nevertheless, due to practical and time constraints, dialysis is generally regarded as a second-line intervention, while the primary priority in the emergency department remains stabilization of myocardial excitability and rapid intracellular shifting of potassium using insulin and β-adrenergic agonists (Chiu et al., 2023; Lemoine et al., 2021; Chaitman et al., 2016).

In this case, the patient received therapy based on our institutional protocol for hyperkalemia, which consisted of repeated cycles of glucose–insulin correction therapy (GICT) using 50 mL of 40% dextrose combined with 10 units of regular insulin per cycle, along with intravenous calcium gluconate (10 mL of 10% solution) for cardiac membrane stabilization. Capillary blood glucose levels were closely monitored before and after each GICT cycle to minimize the risk of hypoglycemia. Given the persistence of hyperkalemia during the early phase of ICU care, adjunctive potassium-lowering strategies were subsequently introduced, including nebulized salbutamol to enhance intracellular potassium shift and intravenous furosemide to promote renal potassium excretion. This multimodal approach resulted in a gradual and sustained reduction in serum potassium levels, accompanied by resolution of hyperkalemia-associated electrocardiographic abnormalities and clinical improvement.

This case highlights the challenges of managing acute hyperkalemia in a resource-limited hospital setting. Advanced extracorporeal potassium elimination strategies, particularly urgent hemodialysis, were not immediately available, despite guideline recommendations to consider dialysis in patients with refractory hyperkalemia, persistent ECG abnormalities after calcium administration, or concomitant acute kidney injury. In such settings, time constraints and limited access to renal replacement therapy necessitate reliance on first-line measures, including membrane stabilization with calcium salts and intracellular potassium shifting using insulin–glucose therapy and β-adrenergic agonists. As emphasized in current consensus statements, prevention of myocardial hyperexcitability and rapid transcellular potassium redistribution remain the primary priorities in the emergency department, while dialysis serves as a second-line option when feasible. The favorable clinical outcome in this patient highlights the importance of early ECG recognition, protocol-driven therapy, and close monitoring to mitigate life-threatening complications when definitive potassium removal modalities are constrained by limited hospital resources.

CONCLUSION

This case illustrates that moderate hyperkalemia can precipitate severe bradyarrhythmias, including sinus arrest with junctional escape rhythm, particularly in elderly patients with underlying cardiac disease and impaired potassium regulation. Early recognition of electrocardiographic changes and rapid initiation of potassium-lowering therapy are crucial to prevent life-threatening complications. Successful management in a resource-limited setting underscores the importance of protocol-driven care, continuous ECG monitoring, and timely laboratory assessment, even when advanced therapies such as dialysis are not immediately available. Clinicians should remain vigilant, as the severity of hyperkalemia-related arrhythmias does not always correlate with the serum potassium level.

REFERENCES

AlSahow A, Bulbanat B, Alhelal B, et al. Management of hyperkalemia: Expert consensus from Kuwait–a Modified Delphi Approach. Int J Nephrol Renovasc Dis 2024;227–240.

Campese VM, Adenuga G. Electrophysiological and clinical consequences of hyperkalemia. Kidney Int Suppl (2011) 2016;6(1):16–19.

Chaitman M, Dixit D, Bridgeman MB. Potassium-Binding Agents for the Clinical Management of Hyperkalemia. P T 2016;41(1):43–50.

Chiu M, Garg AX, Moist L, Jain AK. A new perspective to longstanding challenges with outpatient hyperkalemia: a narrative review. Can J Kidney Health Dis 2023;10.

Harmon DM, Liu K, Dugan J, et al. Validation of Noninvasive Detection of Hyperkalemia by Artificial Intelligence–Enhanced Electrocardiography in High Acuity Settings. Clinical Journal of the American Society of Nephrology 2024;19(8):952–958.

Lemoine L, Bastard Q Le, Batard E, Montassier E. An evidence-based narrative review of the emergency department management of acute hyperkalemia. J Emerg Med 2021;60(5):599–606.

Lindner G, Burdmann EA, Clase CM, et al. Acute hyperkalemia in the emergency department: a summary from a Kidney Disease: Improving Global Outcomes conference. European Journal of Emergency Medicine 2020;27(5):329–337.

Littmann L. Common ECG interpretation software mistakes Part II: Computer errors that hide diagnostic clues. J Electrocardiol 2023;81:277–280.

Massicotte-Azarniouch D, Canney M, Sood MM, Hundemer GL. Managing hyperkalemia in the modern era: a case-based approach. Kidney Int Rep 2023;8(7):1290–1300.

Ortiz-Cortés C, Navarro-González JF. Current insights and challenges in the management of hyperkalemia. Rev Esp Cardiol 2025;

Palmer BF, Carrero JJ, Clegg DJ, et al. Clinical management of hyperkalemia. Mayo Clin Proc 2021;96(3):744–762.

Quintero JA, Medina CA, Penagos F, et al. Electrocardiographic Abnormalities in Patients with Hyperkalemia: A Retrospective Study in an Emergency Department in Colombia. Open Access Emergency Medicine 2024;133–144.

Rossignol P, Legrand M, Kosiborod M, et al. Emergency management of severe hyperkalemia: guideline for best practice and opportunities for the future. Pharmacol Res 2016;113:585–591.

Sarnowski A, Gama RM, Dawson A, Mason H, Banerjee D. Hyperkalemia in chronic kidney disease: links, risks and management. Int J Nephrol Renovasc Dis 2022;215–228.

Sebastian M, Erbano BRH, Wardhana YK. Sine-wave pattern in a case of severe hyperkalemia and end-stage renal failure from a hospital in a rural area: A case report and narrative review. Intisari Sains Medis 2024;15(2):590–594.

Sevamontree C, Jintajirapan S, Phakdeekitcharoen P, Phakdeekitcharoen B. The prevalence and risk factors of hyperkalemia in the outpatient setting. Int J Nephrol 2024;(1).

Suwari CIP, Puradipa B, Wulandari NLES. Junctional Bradikardia pada Pasien Hiperkalemia. Jurnal Kesehatan Andalas 2021;10(2):125–130.

Tetti M, Burrello J, Hureaux M, et al. Prevalence of Hyperkalemia and Familial Hyperkalemic Hypertension in 5100 Patients Referred to a Tertiary Hypertension Unit. Hypertension 2024;81(11):2275–2285.