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CASE REPORT

Complete Atrioventricular Block Due to Severe Hyperkalemia in a Hemodialysis Patient: Successful Management with Temporary Transvenous Pacing

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Abstract: Severe hyperkalemia is a potentially fatal electrolyte disturbance that can produce a broad spectrum of cardiac conduction abnormalities. Although electrocardiographic changes classically follow a predictable progression, advanced atrioventricular block is an uncommon and often overlooked manifestation. We report the case of a 70-year-old woman with end-stage renal disease on maintenance hemodialysis who presented with altered mental status, profound bradycardia, and hypotension. Initial electrocardiography demonstrated complete atrioventricular block with a ventricular escape rhythm of 15–20 beats per minute. Laboratory evaluation confirmed severe hyperkalemia with a serum potassium level of 8.5 mmol/L. Despite prompt initiation of standard medical therapy, including intravenous calcium gluconate, insulin with glucose, and nebulized salbutamol, the patient remained hemodynamically unstable with no improvement in heart rate. Emergency temporary transvenous pacing was therefore performed, resulting in immediate hemodynamic stabilization. Definitive treatment with urgent hemodialysis led to normalization of serum potassium levels and complete recovery of normal sinus rhythm within 24 hours, allowing safe removal of the temporary pacemaker. This case highlights a rare and life-threatening presentation of hyperkalemia-induced complete atrioventricular block and underscores the limitations of medical therapy alone in advanced conduction disturbances. Early recognition and timely implementation of temporary transvenous pacing can be life-saving in hemodynamically unstable patients while definitive correction of hyperkalemia is achieved.

Keywords: Hyperkalemia, complete atrioventricular block, end-stage renal disease, hemodialysis, bradyarrhythmia, temporary transvenous pacing

Introduction

Hyperkalemia is a frequent and potentially fatal electrolyte disturbance encountered in acute and chronic medical settings, particularly among patients with impaired renal potassium excretion.[1] The myocardium is especially sensitive to elevations in extracellular potassium, and even modest increases can alter cardiac conduction and excitability.[2] In patients with advanced chronic kidney disease, impaired renal clearance, dietary indiscretion, missed dialysis sessions, or intercurrent illness may precipitate abrupt rises in serum potassium, resulting in rapid clinical deterioration.[3]

Electrocardiography is traditionally used as a bedside tool to assess the cardiac effects of hyperkalemia. Classical teaching describes a progressive sequence of ECG changes, beginning with peaked T waves and advancing through PR prolongation, QRS widening, sine-wave morphology, and ultimately ventricular fibrillation or asystole.[4] However, real-world presentations often deviate from this pattern, and the correlation between serum potassium concentration and ECG findings is inconsistent.[5] Severe hyperkalemia may occasionally present without typical ECG changes, while conversely, profound conduction abnormalities may occur unexpectedly.[6]

High-grade atrioventricular conduction disturbances are an uncommon manifestation of hyperkalemia.[7] Complete atrioventricular block, in particular, is rarely reported and may obscure the metabolic etiology, leading clinicians to

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initially consider intrinsic conduction disease, ischemia, or drug toxicity.[8] In dialysis-dependent patients, this diagnostic uncertainty can delay appropriate treatment and increase the risk of circulatory collapse.

Management of life-threatening hyperkalemia involves immediate myocardial membrane stabilization, redistribution of potassium into cells, and definitive potassium removal.[9] While these measures are often effective in reversing early electrophysiological abnormalities, they may not promptly restore adequate heart rate or cardiac output in patients with advanced conduction block.[10] In such situations, temporary cardiac pacing may be required as a supportive bridge until serum potassium levels can be corrected.[11]

We report a case of severe hyperkalemia in a patient undergoing maintenance hemodialysis who presented with complete atrioventricular block and a slow ventricular escape rhythm, resulting in profound hemodynamic instability. This case highlights an atypical electrocardiographic manifestation of hyperkalemia and emphasizes the critical role of temporary transvenous pacing when standard medical therapy alone fails to stabilize the patient.[12]

This case is notable for the presentation of severe hyperkalemia (K^+ 8.5 mmol/L) with complete atrioventricular block in the absence of classical electrocardiographic progression, failure of standard medical therapy to restore conduction, and the requirement for temporary transvenous pacing prior to definitive hemodialysis. These features highlight an atypical and life-threatening presentation requiring early escalation of care.

Case Presentation

A 70-year-old woman with a history of end-stage renal disease on maintenance hemodialysis twice weekly presented to the emergency department with acute alteration in mental status. Her medical history was significant for long-standing hypertension and chronic kidney disease. There was no known history of ischemic heart disease, prior conduction abnormalities, or use of atrioventricular nodal–blocking medications. According to the available history, there had been no recent changes in her medication regimen, and no preceding chest pain or syncope was reported.

The patient had been receiving regular hemodialysis sessions twice weekly, with her last session occurring approximately three days prior to presentation. There was no clearly documented history of missed dialysis sessions. Information regarding dialysate potassium concentration was not available. She was not taking any known potassium-raising medications, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, or potassium supplements.

On arrival, the patient was profoundly bradycardic, with a heart rate of 15–20 beats per minute, and hypotensive, with a blood pressure of 80/40 mmHg. She appeared drowsy and poorly responsive, consistent with cerebral hypoperfusion. Oxygen saturation was maintained on supplemental oxygen. Physical examination revealed cool extremities with delayed capillary refill. Cardiac auscultation demonstrated markedly slow heart sounds without murmurs, while lung examination was unremarkable. There were no clinical signs suggestive of acute volume overload or infection.

A 12-lead electrocardiogram obtained on presentation demonstrated complete atrioventricular block with a ventricular escape rhythm (Figure 1). The ventricular rate was approximately 15–20 beats per minute. Atrial activity was difficult to discern, and P waves were not consistently visible, making precise assessment of atrial rate and PR interval unreliable. The QRS complexes were widened (approximately 140–160 ms), consistent with a ventricular escape rhythm. Notably, classical electrocardiographic features of hyperkalemia, such as peaked T waves, were absent, indicating an atypical presentation.

Urgent laboratory investigations confirmed severe hyperkalemia with a serum potassium level of 8.5 mmol/L. Additional findings included elevated serum creatinine (5.6 mg/dL) and blood urea nitrogen (65 mg/dL), consistent with renal dysfunction. Arterial blood gas analysis revealed metabolic acidosis with a pH of 7.26 and bicarbonate level of 16 mmol/L. Serum calcium was reduced (7.3 mg/dL), while magnesium was within normal limits (1.8 mg/dL). Blood glucose was 132 mg/dL. A mildly elevated cardiac troponin level (0.12 ng/mL) was noted in the absence of clinical or electrocardiographic evidence of acute coronary syndrome. A summary of laboratory findings is provided in Table 1.

Standard emergency treatment for hyperkalemia was initiated promptly. The patient received intravenous calcium gluconate (10 mL of 10% solution) for myocardial stabilization, followed by intravenous regular insulin (10 units) with 25 g of glucose to facilitate intracellular potassium shift. Nebulized salbutamol was also administered. Intravenous

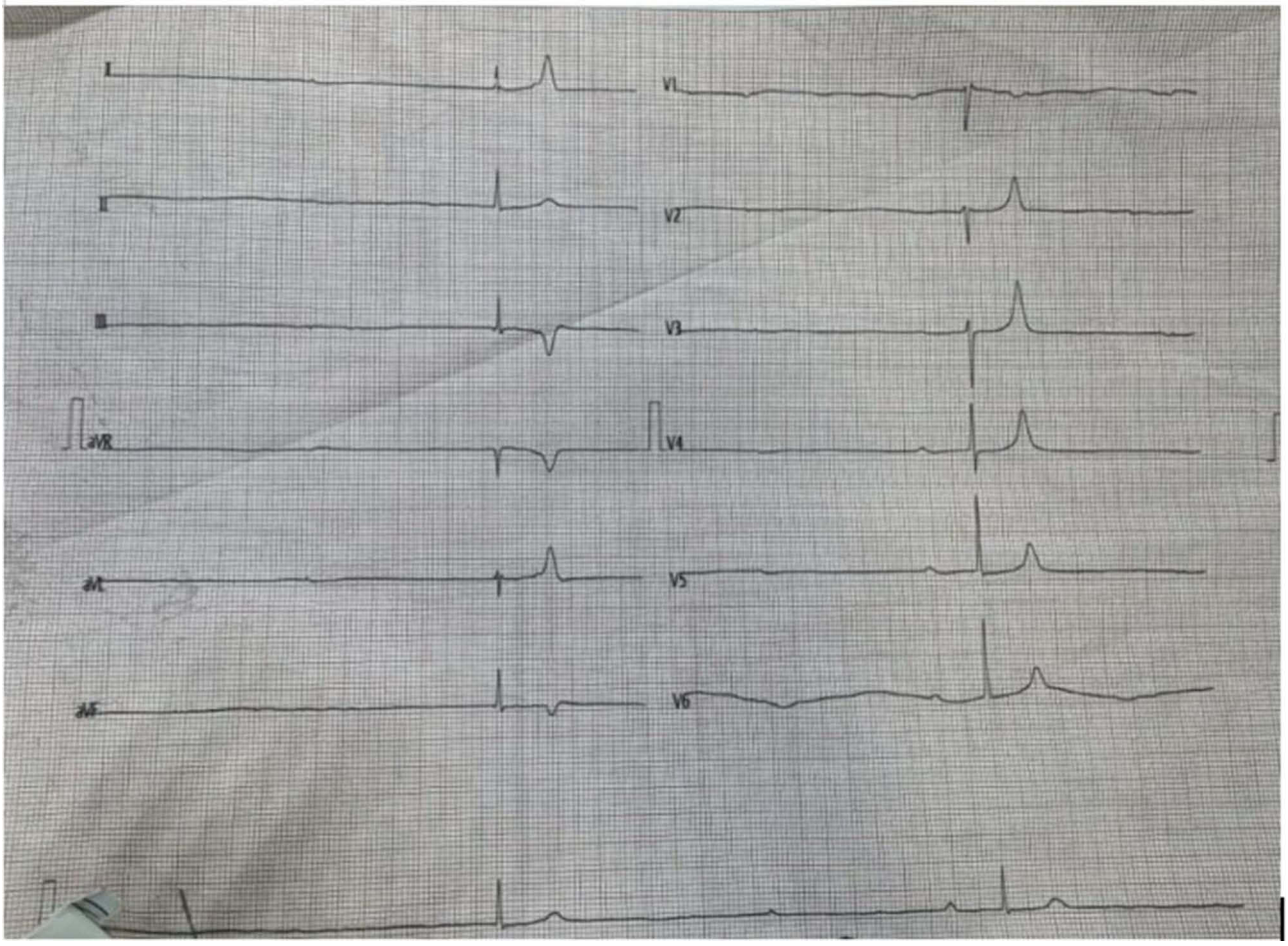

Figure 1 12-lead electrocardiogram demonstrating complete atrioventricular block with a ventricular escape rhythm and marked bradycardia at presentation.

atropine (1 mg) was given in an attempt to improve bradycardia; however, no significant improvement in heart rate or blood pressure was observed.

Given persistent severe bradycardia and hemodynamic instability, urgent temporary transvenous pacing was performed. The patient was transferred to the cardiac catheterization laboratory, where a temporary pacemaker was successfully inserted, resulting in immediate restoration of a stable paced rhythm at approximately 102 beats per minute (Figure 2). This intervention led to rapid improvement in blood pressure and level of consciousness.

Table 1 Laboratory Findings on Admission

Parameter	Value	Normal Range
Potassium	8.5 mmol/L	3.5–5.0 mmol/L
Creatinine	5.6 mg/dL	0.6–1.2 mg/dL
Blood Urea Nitrogen	65 mg/dL	7–20 mg/dL
pH	7.26	7.35–7.45
Bicarbonate	16 mmol/L	22–28 mmol/L
Calcium	7.3 mg/dL	8.5–10.5 mg/dL
Magnesium	1.8 mg/dL	1.7–2.2 mg/dL
Glucose	132 mg/dL	70–140 mg/dL
Troponin	0.12 ng/mL	<0.04 ng/mL

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Figure 2 Cardiac monitor tracing demonstrating effective ventricular pacing following insertion of a temporary trans venous pacemaker, with a paced rhythm of approximately 102 beats per minute.

The patient was subsequently admitted to the intensive care unit, where urgent hemodialysis was performed. Following dialysis, serum potassium levels normalized to 4.0 mmol/L. Within 24 hours, repeat electrocardiography demonstrated restoration of normal sinus rhythm with intact atrioventricular conduction (Figure 3). The temporary pacemaker was removed without complication, and the patient remained clinically stable thereafter.

Discussion

This case illustrates an uncommon but clinically significant manifestation of severe hyperkalemia presenting as complete atrioventricular block with a profoundly slow ventricular escape rhythm. The distinguishing features of this case include the presence of extreme hyperkalemia (K^+ 8.5 mmol/L), absence of classical electrocardiographic progression, failure of standard medical therapy to restore atrioventricular conduction, and the requirement for temporary transvenous pacing prior to definitive hemodialysis. Although hyperkalemia-related conduction abnormalities are well recognized, this combination of findings remains rare and poses important diagnostic and therapeutic challenges.[7,8]

The electrophysiological effects of hyperkalemia are primarily mediated by a reduction in the transmembrane potassium gradient, resulting in partial depolarization of the resting membrane potential and inactivation of fast sodium channels. This leads to impaired impulse generation and slowed conduction throughout the myocardium.[13] While sinus node suppression, PR interval prolongation, and QRS widening are commonly described, progression to complete atrioventricular block is relatively uncommon and likely reflects increased susceptibility of the atrioventricular node and His–Purkinje system to elevated extracellular potassium levels.[14]

A notable feature of this case is the absence of classical electrocardiographic progression typically associated with hyperkalemia. Traditional teaching describes a predictable sequence of ECG changes, beginning with peaked T waves and

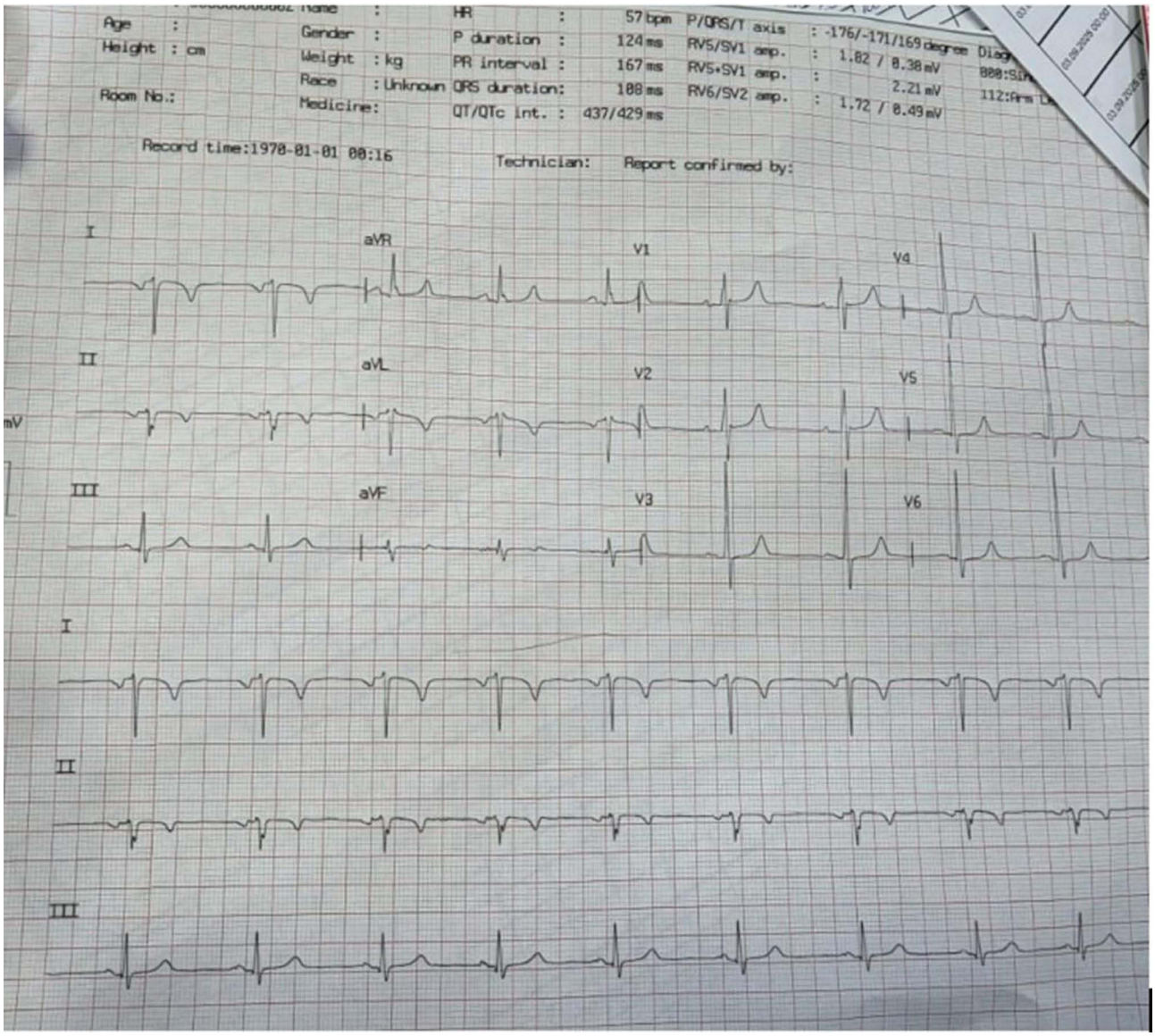

Figure 3 12-lead electrocardiogram demonstrating restoration of normal sinus rhythm with intact atrioventricular conduction following correction of hyperkalemia.

progressing to widening of the QRS complex and eventual sine-wave morphology.[4] However, increasing evidence suggests that this progression is inconsistent and unreliable in clinical practice.[5,15] In this patient, the presence of complete atrioventricular block without preceding classical features highlights the limitation of relying solely on ECG patterns to assess the severity of hyperkalemia, particularly in high-risk populations such as patients with end-stage renal disease.

Another important aspect is the lack of response to standard medical therapy. Despite prompt administration of intravenous calcium gluconate, insulin with glucose, and beta-agonist therapy, there was no improvement in heart rate or hemodynamic status. This observation underscores that while medical therapy is essential for myocardial stabilization and intracellular potassium redistribution, it may be insufficient to reverse advanced conduction disturbances once established.[9,16]

Temporary transvenous pacing played a critical role in the management of this patient. The presence of profound bradycardia and hypotension indicated severely compromised cardiac output, necessitating immediate intervention. In this context, temporary pacing should be considered a life-saving supportive measure rather than a failure of medical therapy. It provides hemodynamic stabilization while definitive potassium removal is achieved through hemodialysis. The rapid restoration of normal sinus rhythm following correction of hyperkalemia further supports the reversible metabolic nature of

the conduction disturbance and highlights the importance of addressing the underlying cause before considering permanent pacemaker implantation.[17,18]

From a diagnostic perspective, it is essential to consider alternative causes of complete atrioventricular block. These include acute coronary syndrome, drug-induced conduction disturbances, degenerative conduction disease, endocrine disorders such as hypothyroidism, and electrolyte abnormalities, particularly hyperkalemia. Thyroid dysfunction, especially overt hypothyroidism, is a recognized but uncommon cause of high-grade atrioventricular block and may present with reversible conduction abnormalities if promptly identified and treated, as demonstrated in previously reported cases.[19] In contrast, hyperkalemia is a well-established metabolic cause of conduction disturbances, although progression to complete atrioventricular block remains relatively rare.[7] In the present case, the absence of clinical features suggestive of thyroid disease, together with the rapid resolution of conduction abnormalities following correction of hyperkalemia, strongly supports a metabolic etiology. Acute coronary syndrome was also considered, particularly in light of the mildly elevated troponin level; however, the absence of ischemic symptoms, lack of dynamic electrocardiographic changes, and complete recovery after normalization of serum potassium made this diagnosis unlikely. Similarly, drug-induced conduction disturbances were excluded due to the absence of atrioventricular nodal–blocking medications, and degenerative conduction system disease was considered less likely given the complete reversibility of the atrioventricular block.[20]

This case also carries important clinical implications. First, clinicians should maintain a high index of suspicion for hyperkalemia in patients presenting with bradyarrhythmias, particularly those with underlying renal disease, even in the absence of classical ECG findings.[6] Second, reliance on electrocardiographic patterns alone may be misleading, and early laboratory confirmation is essential. Third, in patients with severe hyperkalemia and hemodynamic instability, temporary transvenous pacing may be required as a bridge to definitive therapy, especially when there is delayed access to hemodialysis.[11,21] Finally, the complete reversibility of conduction abnormalities following correction of hyperkalemia underscores the importance of identifying metabolic causes before considering permanent pacing interventions.

Conclusion

Severe hyperkalemia is a life-threatening electrolyte disturbance that may rarely present as complete atrioventricular block, particularly in patients with end-stage renal disease. This case highlights an atypical presentation characterized by the absence of classical electrocardiographic progression and lack of response to standard medical therapy. In the setting of hemodynamic instability, temporary transvenous pacing serves as a critical life-saving intervention, maintaining adequate cardiac output while definitive correction of hyperkalemia is achieved through urgent hemodialysis.

The complete resolution of conduction abnormalities following normalization of serum potassium underscores the reversible nature of this condition and emphasizes the importance of identifying metabolic causes before considering permanent pacing. Early recognition, prompt escalation of care, and a multidisciplinary approach are essential to improving outcomes in such high-risk patients.

Generative Artificial Intelligence

Generative artificial intelligence tools (ChatGPT, OpenAI) were used for language editing and stylistic refinement of the manuscript. The authors take full responsibility for the content, accuracy, and integrity of the work.

Ethics Approval

Based on the regulations of the review board of Mogadishu Somali Türkiye Training and Research Hospital, institutional review board approval is not required for case reports.

Consent for Publication

The patient provided written informed consent for the publication of this case report and any accompanying images.

Patient Consent

Written informed consent was given by the patient for this case report, along with any data and images that may be included, to be published. The patient is aware that their identity will be kept private and that the data will only be utilized for educational purposes.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; agreed on the journal; and are accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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