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A REVERSIBLE CASE OF COMPLETE HEART BLOCK DUE TO HYPERKALEMIA FOLLOWING GASTROENTERITIS AND PRE-RENAL AKI: A RARE PRESENTATION WITH SUCCESSFUL REVERSAL

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Abstract

Background: Hyperkalaemia is a frequent electrolyte disturbance encountered in emergency and critical care settings, often associated with high morbidity and mortality due to its cardiac effects. While classical electrocardiographic changes progress in a predictable sequence from peaked T waves to sine-wave morphology, the occurrence of complete heart block (CHB) is rarely reported.

Case Presentation: We report the case of a middle-aged male who presented with vomiting, diarrhea, dehydration, and acute onset dyspnea with presyncope. He was found to have hyperkalaemia in the setting of pre-renal acute kidney injury, with electrocardiography revealing CHB and a ventricular escape rhythm. The patient was managed with intravenous calcium gluconate, insulin-dextrose infusion, β-agonist nebulization, oral potassium binders, antiemetics, intravenous hydration, and supportive therapy. Owing to hemodynamic instability, a temporary pacemaker was placed via the transfemoral approach. Within 36 hours, his serum potassium normalized, and sinus rhythm was restored, allowing safe removal of the pacemaker. At discharge and two-week follow-up, ECG and electrolytes were normal, with no conduction abnormalities.

Conclusion: This case highlights the importance of recognizing hyperkalaemia as a potentially reversible cause of CHB, even at modest potassium elevations, and underscores the role of timely electrolyte correction and temporary pacing in management.

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Keywords: Hyperkalaemia; Complete heart block; Acute kidney injury; Electrolyte disturbance; Temporary pacing.

INTRODUCTION

Hyperkalaemia, defined as a serum potassium concentration above 5.0 mmol/L, is one of the most clinically significant electrolyte abnormalities in medical practice. Its prevalence ranges from 1% to 10% in hospitalized patients, rising sharply in those with renal impairment or on renin-angiotensin-aldosterone system inhibitors [1,2]. Hyperkalaemia is associated with adverse outcomes, including increased risk of cardiac arrhythmias and sudden death [3].

The effects of hyperkalaemia on the heart are well established, with electrocardiographic (ECG) changes usually progressing in a dose-dependent sequence: tall peaked T waves, prolongation of the PR interval, QRS widening, and ultimately, sine-wave morphology preceding ventricular fibrillation or asystole [4,5].

Complete atrioventricular block (CHB), however, is an uncommon manifestation. In most cases, hyperkalaemia affects conduction tissue uniformly, leading to generalized slowing of conduction rather than discrete nodal block. CHB tends to occur sporadically, and reports in the literature remain limited [6–9]. Moreover, when it does occur, it is usually associated with severe hyperkalaemia or advanced renal dysfunction.

Here, we describe the case of a middle-aged male who developed transient CHB at a relatively modest potassium level, in the setting of acute pre-renal kidney injury caused by gastrointestinal fluid loss. The case is clinically important as it illustrates a rare but reversible presentation of hyperkalaemia, emphasizes the need for early recognition, and highlights the importance of temporary pacing as a bridge to recovery.

Case Presentation

A 50-year-old male with a history of hypertension presented to the emergency department with acute onset of giddiness and shortness of breath (grade IV dyspnea) in the early hours of the morning. He had a preceding history of multiple episodes of watery diarrhea over two days, associated with four to five episodes of vomiting that were non-bilious and non-blood-stained. There was no history of chest pain, palpitations, syncope, or prior cardiovascular disease. He was not taking any medications known to influence potassium homeostasis such as ACE inhibitors, ARBs, potassium-sparing diuretics, or digoxin.

On examination, the patient was dehydrated and mildly tachypneic. His blood pressure was 110/70 mmHg, heart rate 36 beats per minute, oxygen saturation 98% on room air, and temperature was afebrile. Systemic examination was otherwise unremarkable.

An electrocardiogram performed in the emergency department revealed bradycardia with atrioventricular dissociation consistent with complete heart block (Figure 2). A prior ECG obtained during a routine health check-up had shown normal sinus rhythm (Figure 1).

A bedside echocardiogram demonstrated normal left ventricular systolic function with no regional wall motion abnormalities, normal chamber dimensions, and no structural or valvular abnormalities (Figure 3). A chest radiograph performed on admission showed no acute abnormalities.

Laboratory investigations revealed: serum potassium 6.4 mmol/L, serum creatinine 1.4 mg/dL (baseline 0.7 mg/dL), serum urea 56 mg/dL, and arterial blood gas showing mild metabolic acidosis. These findings were consistent with acute kidney injury, probably pre-renal in etiology due to volume depletion from gastrointestinal losses. Other laboratory values included sodium 134 mmol/L, calcium 8.7 mg/dL, magnesium 1.8 mg/dL, phosphorus 3.2 mg/dL, and normal liver function and complete blood counts. C-reactive protein was elevated at 42 mg/L. Thyroid function tests were within normal limits. Cardiac biomarkers, including troponin I and CK-MB, were negative, helping exclude acute coronary syndrome and myocarditis.

The patient was promptly managed with intravenous calcium gluconate, insulin in dextrose infusion, salbutamol nebulization, and oral potassium-binding resins. He also received adequate intravenous hydration, anti-emetics, and other supportive medications. In view of hemodynamic instability and persistent bradycardia, a temporary transvenous pacemaker was inserted via the femoral vein, and post-procedure chest radiography and ECG confirmed appropriate lead placement (Figure 4,5).

Over the subsequent 36 hours, with aggressive correction of hyperkalaemia and rehydration, serum potassium levels normalized. The patient's rhythm reverted to normal sinus rhythm, allowing safe removal of the temporary pacemaker.

He was monitored in the intensive care unit and later shifted to the general ward. After a period of observation, he was discharged in stable condition. At two-week follow-up, repeat ECG showed normal sinus rhythm with no conduction abnormalities (Figure 6), and serum electrolytes were within normal limits.

DISCUSSION

Pathophysiological Basis of Hyperkalaemia-Induced Conduction Defects

Potassium plays a pivotal role in cardiac electrophysiology, maintaining resting membrane potential and influencing excitability. Modest increases in extracellular potassium decrease the transmembrane gradient, causing partial depolarization of resting membrane potential and reducing the rate of phase 0 depolarization. This slows conduction through atrial myocardium, the atrioventricular (AV) node, and the His-Purkinje system[10,11].

The classical progression of ECG abnormalities in hyperkalaemia follows a predictable sequence: peaked T waves, PR prolongation, QRS widening, and eventual sine-wave morphology[4,12]. CHB remains a rare event, likely because hyperkalaemia typically produces diffuse conduction slowing rather than focal block. However, variations in tissue sensitivity across different conduction pathways may predispose to atrioventricular dissociation and ventricular escape rhythms. This explains why CHB may occur abruptly and sometimes at potassium levels that are not profoundly elevated, as in this patient.

Epidemiology and Risk Factors

Hyperkalaemia occurs in up to 10% of hospitalized patients and is most frequent in those with chronic kidney disease, heart failure, diabetes mellitus, and those receiving RAAS inhibitors or potassium-sparing diuretics [1–3]. Cardiac conduction disturbances typically occur at potassium concentrations above 6.5–7.0 mmol/L, but susceptibility varies considerably. Risk factors for severe arrhythmias include rapid potassium rise, baseline conduction disease, underlying structural heart disease, and concomitant metabolic acidosis [7,13].

The rarity of CHB as a complication is notable. In a pooled review of hyperkalaemia cases, advanced AV block was found in less than 5% [14]. Most reported cases are in elderly individuals, those with advanced renal dysfunction, or in the presence of interacting drugs. The present case is distinctive in that CHB developed at a relatively modest potassium elevation in a patient without prior conduction abnormalities or chronic kidney disease.

Review of Reported Cases in Literature

Fisch et al. were among the first to systematically describe ECG changes in hyperkalaemia, noting that conduction disturbances can appear at variable potassium thresholds [12]. Since then, sporadic case reports have documented hyperkalaemia-induced CHB, many of which reversed after potassium normalization [15–18]. Schnaubelt et al. (2020) described a similar case of persistent CHB due to hyperkalaemia in chronic kidney disease, resolving with correction [19]. Baratloo et al. reported a patient with advanced renal failure and severe hyperkalaemia (K+ 7.6 mmol/L) presenting with CHB, which reversed after dialysis [20].

More recent reports confirm that arrhythmias may develop unpredictably, not strictly correlating with potassium levels, but influenced by acute changes, conduction reserve, and coexistent metabolic disturbances [21,22]. This highlights the need for vigilance even when potassium elevation appears only moderate.

Clinical Correlation with the Present Case

The present case reinforces several key diagnostic and therapeutic principles. The patient had no pre-existing conduction abnormalities, with a baseline ECG confirming normal sinus rhythm. His CHB developed in the setting of acute pre-renal kidney injury and resolved rapidly after potassium correction, supporting a causal relationship.

The role of temporary pacing was critical, serving as a bridge until metabolic correction was achieved. Importantly, permanent pacemaker implantation was avoided. At follow-up, the patient demonstrated no residual conduction disease, underlining the reversibility of this phenomenon.

Diagnostic Challenges

The differential diagnosis of CHB is broad, encompassing ischemic heart disease, myocarditis, idiopathic degeneration of the conduction system, drug toxicity (e.g., beta-blockers, calcium channel blockers, digoxin), and electrolyte abnormalities [23]. In acute presentations, rapid evaluation is essential to distinguish reversible causes.

In this case, normal echocardiography, negative cardiac biomarkers, and absence of offending drugs excluded structural and ischemic causes. The temporal association with hyperkalaemia, dehydration, and AKI provided

a unifying explanation. This underscores the importance of urgent electrolyte evaluation in all patients presenting with CHB, especially when no prior cardiac disease is documented.

Therapeutic Implications

Management of hyperkalaemia rests on three pillars: membrane stabilization, intracellular potassium shift, and potassium elimination. Intravenous calcium salts (calcium gluconate or chloride) provide immediate cardioprotection. Insulin-dextrose infusions and β-agonists shift potassium intracellularly, while binders and renal clearance eliminate it. Dialysis remains the definitive option when conservative measures fail [24].

In the present case, medical management sufficed as renal function improved with rehydration. Potassium binders were useful adjuncts, and temporary pacing provided hemodynamic stability. This case illustrates the principle that temporary pacing should be considered a bridge rather than definitive therapy in metabolic conduction block.

Lessons for Clinical Practice

- Hyperkalaemia can produce life-threatening conduction abnormalities, including CHB, even at modest levels.
- Electrolyte testing should be mandatory in all patients with new-onset advanced conduction defects.
- Temporary pacing is often required to bridge patients with unstable bradyarrhythmias.
- Permanent pacemaker implantation should be deferred until reversible metabolic causes are excluded.

CONCLUSION

This case illustrates that hyperkalaemia-induced CHB, though rare, can occur abruptly and even at moderate potassium elevations. Early recognition, protocol-driven management of hyperkalaemia, and temporary pacing as a bridge to recovery are essential for preventing morbidity and mortality. Clinicians must maintain a high index of suspicion for electrolyte disturbances in all cases of new-onset CHB.

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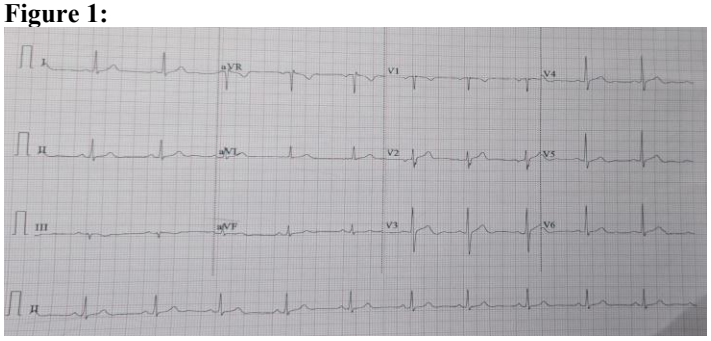
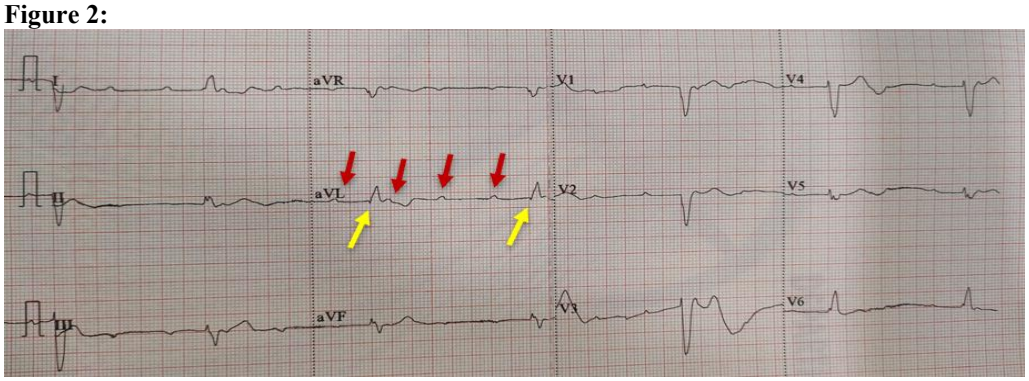
Figure 1:



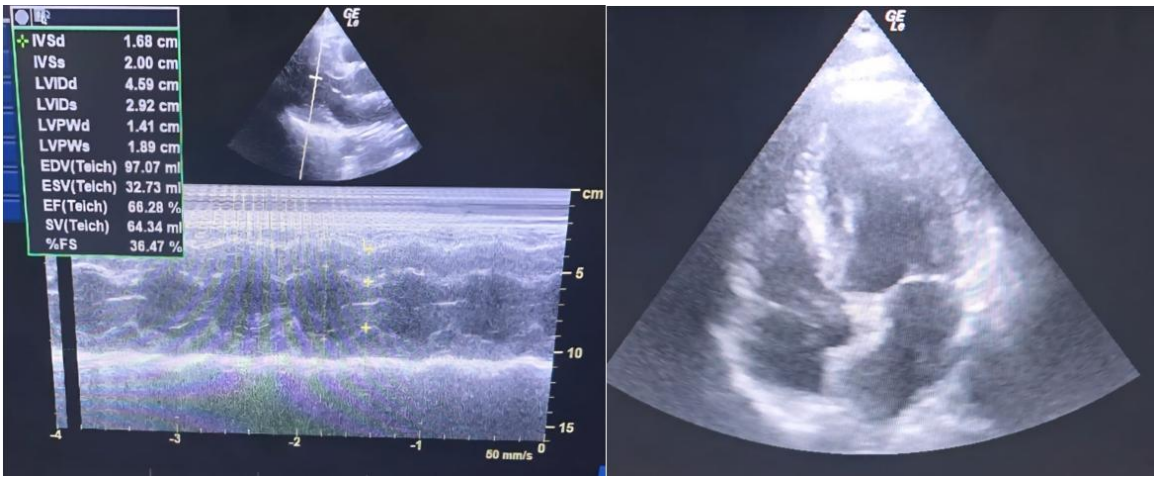
Baseline ECG obtained during a routine healthcare checkup 1 year prior showing normal sinus rhythm.

Figure 2:



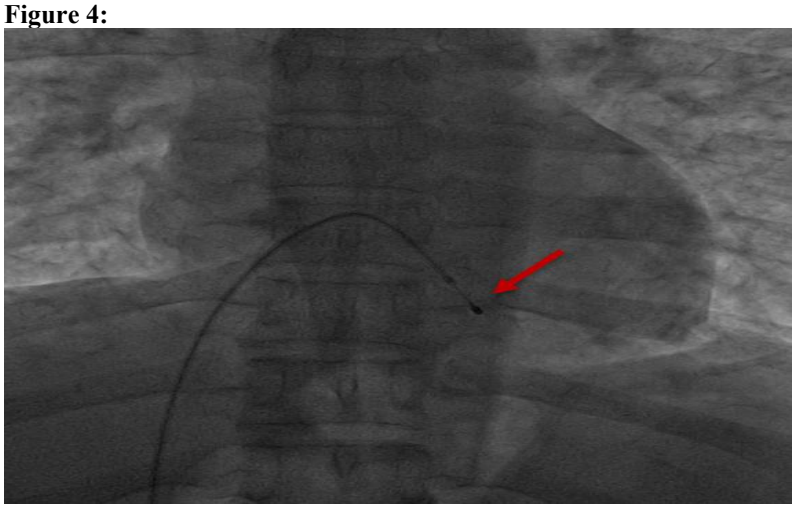
ECG at presentation demonstrating AV dissociation (Red arrows indicate p waves and yellow arrows QRS complexes) - complete atrioventricular block.

Figure 3:



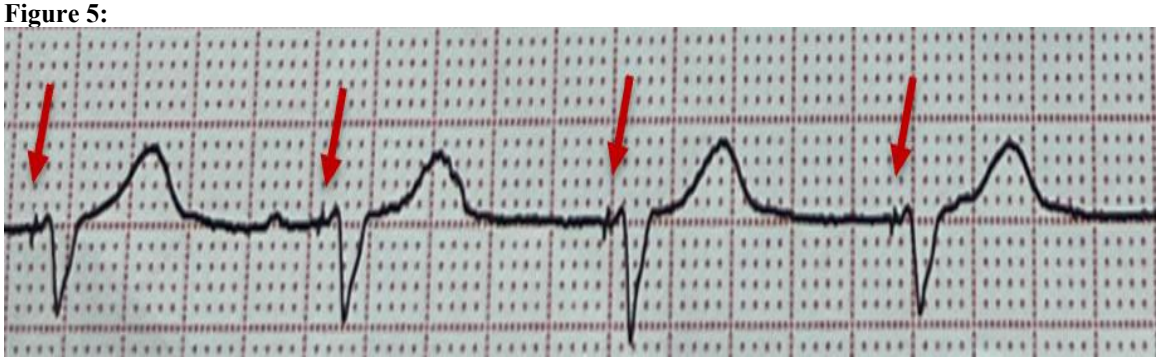
Echocardiographic images – parasternal long-axis view showing normal left ventricular systolic function and apical four-chamber view demonstrating normal chamber dimensions and valves.

Figure 4:



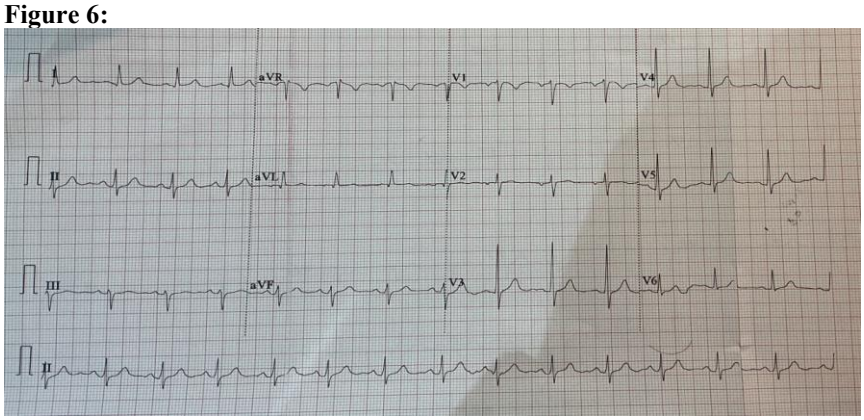
Chest X-ray following procedure showing radiomarked pacing electrode (Red arrow) secured in right ventricle via transfemoral approach.

Figure 5:



ECG taken post temporary pacemaker insertion showing Pacing spikes (Red arrow)

Figure 6:



ECG at discharge and two-week follow-up showing normal sinus rhythm with no conduction abnormalities.