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Bolus doses of insulin versus a continuous insulin infusion for treating hyperkalaemia in adults: A case series

Nicole King[1,2], Neil Martinson[2,3], Mmakwata Lesudi[1] and Ebrahim Variava[1,2]

Abstract

Hyperkalaemia is a medical emergency that may manifest with a spectrum of cardiac conduction abnormalities and, if not promptly managed, can progress to cardiac arrest. While standard management typically involves stat doses of insulin to lower serum potassium, the optimal dosing regimen remains controversial. Patients whose serum potassium exceeds the upper normal limit by more than 1 mmol/L often require repeated conventional management to shift potassium intracellularly – usually every 2 to 4 h. Although dialysis is indicated in patients with concomitant renal dysfunction, its availability in resource-constrained settings is limited. We present three cases of symptomatic hyperkalaemia successfully managed with continuous insulin infusions, thereby obviating the need for dialysis. These cases underscore the potential role of continuous insulin infusions as an alternative strategy for correcting hyperkalaemia in settings where dialysis is not immediately available.

Keywords

insulin infusion, hyperkalaemia, heart block

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Introduction

Hyperkalaemia predominantly occurs in the context of renal dysfunction. Additional risk factors include cellular redistribution, beta blockers (impair cellular uptake), hypoaldosteronism, the use of renin-angiotensin-aldosterone system (RAAS) inhibitors (ACE inhibitors and spironolactone), and albeit rarely, excessive dietary intake.[1] Over the past three decades, the global all-age prevalence of chronic kidney disease has increased by 29.3%, with a disproportionate disease burden in developing countries.[2] Poverty and lower socioeconomic status are independently associated with an increased risk of CKD, and when combined with residence in remote areas with limited healthcare access, these factors contribute to a particularly vulnerable population with severely restricted access to dialysis.[3] Reports on continuous insulin and dextrose infusions extending beyond 1 h for the correction of hyperkalaemia are scarce, with a single case report of a patient who refused dialysis, failed conventional insulin measures, but had a junctional bradycardia which responded to continuous insulin infusion.[4]

Literature review

Potassium is the primary intracellular cation, with approximately 98% of total body potassium residing within cells. The Na-K-ATPase pump is critical for maintaining the intracellular–extracellular potassium gradient, which in turn is a major determinant of the resting membrane potential of cells. Both hypokalaemia and hyperkalaemia can disrupt this

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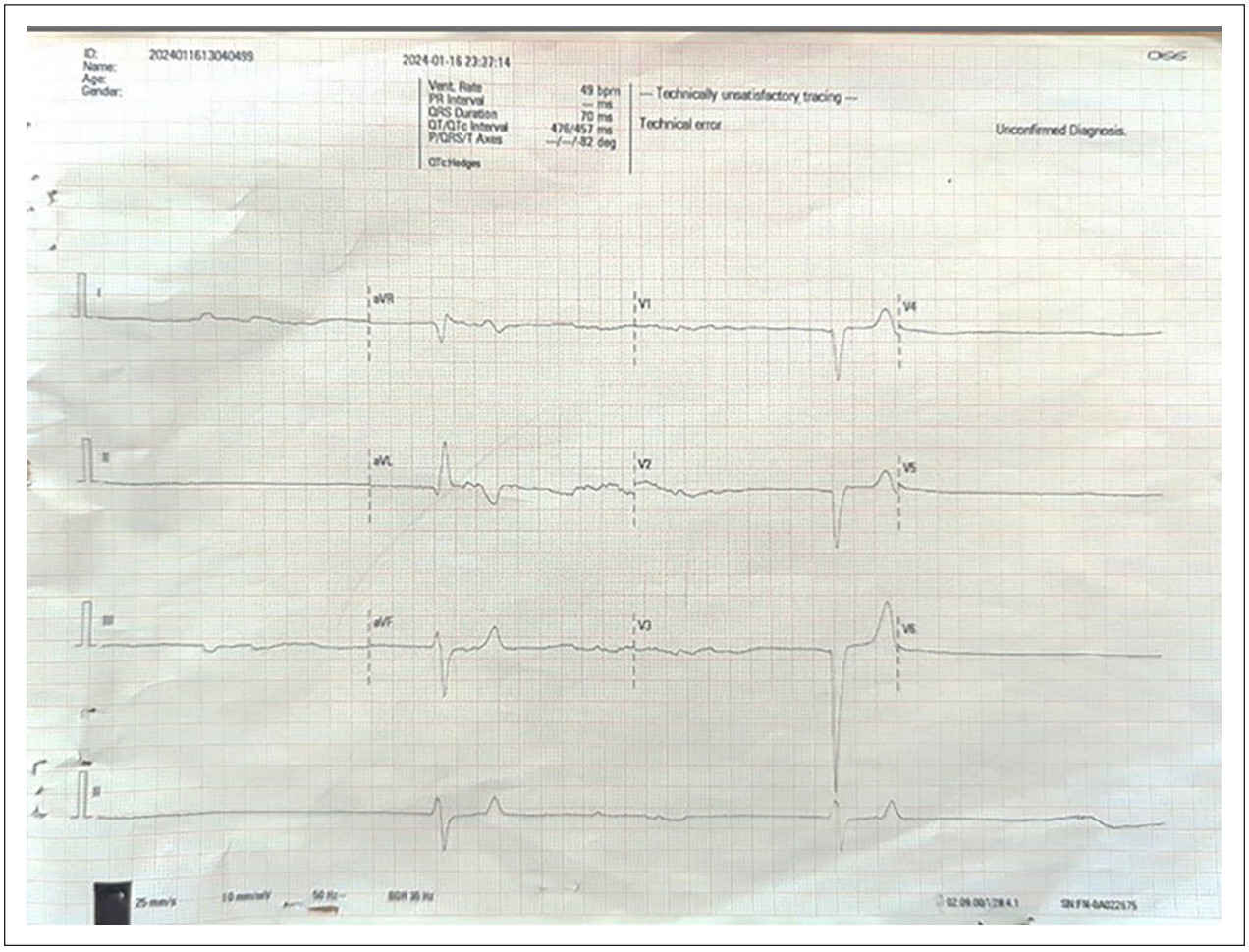


Figure 1. Patient 1: ECG on presentation showing complete heart block. Serum potassium 8.5 mmol/L.

gradient, leading to muscle paralysis and a variety of cardiac arrhythmias that may precipitate cardiac arrest.[5]

Clinically, patients with hyperkalaemic emergencies present with symptoms including muscle weakness, paralysis, bradycardia, sinus arrest, slow idioventricular rhythms, ventricular tachycardia, ventricular fibrillation and asystole. The recommended therapeutic approach includes intravenous calcium to stabilise cardiac cell membranes, intravenous insulin to promote cellular uptake of potassium, diuretic therapy in those with normal renal function to excrete potassium and to correct reversible causes by withholding non-steroidal anti-inflammatory drugs (NSAIDS) and RAAS inhibitors.[6,7]

In patients with impaired renal function, haemodialysis is indicated. However, many facilities, especially those in resource-limited settings, lack immediate dialysis access which may cause life-threatening delays in initiating appropriate therapy during transfer of critically ill patients.[8]

A bolus of 10 units of insulin administered concurrently with 50 mL of 50% dextrose rapidly induces a hypokalaemic effect; however, this is transient. The high insulin levels necessary for potassium lowering are not sustained after a single bolus – the half-life of an insulin bolus in plasma is 7 to 8 min[9] – and hypoglycaemia (reported in 8%–75% of patients in two studies)[10] may ensue an hour or more later despite the initial dextrose bolus. The half-life of a dextrose bolus greatly varies: the normal degree of variability of intravenous fluids spans at least a 10-fold variation.[11]

Case presentations

Case 1

A 51-year-old female with a history of hypertension, chronic kidney disease (CKD) and a subaortic membrane leading to left ventricular obstruction was awaiting surgery when she presented to the emergency department following an episode of dizziness and syncope. On arrival, she was critically ill, with a severe bradycardia (15 bpm) and hypotensive with a blood pressure of 76/43 mmHg. She had a depressed level of consciousness (Glasgow Coma Scale 11/15: E3V3M5). An electrocardiogram (Figure 1) demonstrated a complete (third-degree) atrioventricular block, prompting the immediate application of transcutaneous pacing pads due to her haemodynamic instability.

Table 1. Patient 1: Potassium concentration, ventricular rate, glucose trend per interventional measure over time.

Time:	Presentation (Hour 0)	Hour 2	Hour 5	Hour 9	Hour 10	Hour 16	Hour 22
Potassium (mmol/L)	8.5	8.6	6.4	7.8	7.2	5.0	3.8
Ventricular Rate (bpm)	15	37	33	31	31	46	67
Type of Intervention	NA	10 IU Actrapid 10 ml 10% Calcium carbonate 50 ml 50% Dextrose	10 IU Actrapid 10 ml 10% Calcium carbonate 50 ml 50% Dextrose	10 IU Actrapid 10 ml 10% Calcium carbonate 50 ml 50% Dextrose	Start of Actrapid Infusion (0.1 IU/kg/h)	6 h into Actrapid infusion (0.1 IU/kg/h)	12 h into Actrapid infusion (0.1 IU/kg/h)
Glucose (mmol/L)	13.8	20.7	7.3	4.2	3.6	5.4	4.3

Arterial blood gas analysis revealed a serum potassium of 8.5 mmol/L. Despite an initial potassium-shifting intervention consisting of 10 IU of Actrapid, 50 ml 50% dextrose and 10 ml 10% calcium carbonate, her potassium remained elevated at 8.6 mmol/L. Subsequent attempts using the same agents and dosages as prior produced transient reductions in serum potassium – nadir 6.4 mmol/L – without resolution of complete heart block Table 1). With acute haemodialysis, a decision was made to commence a continuous infusion of Actrapid at 0.1 IU/kg/h, accompanied by a 50% dextrose infusion starting at 1 ml/kg/hr to maintain normoglycaemia (Table 2). Within 12 h, the patient's serum potassium normalized to a value of 3.8 mmol/L (Graft 1), and her dependency on transcutaneous pacing diminished. By the following morning, she had reverted to sinus rhythm with a heart rate exceeding 60 bpm (Figure 2). Her glucose was monitored hourly for the duration of the infusion.

Case 2

A 74-year-old female was referred from a district hospital to our tertiary centre for cardiology evaluation after being diagnosed with presumed complete heart block. Her past medical history included hypertension complicated by hypertensive heart disease, for which she was receiving a combination of five antihypertensive medications (amlodipine, enalapril, spironolactone, furosemide and carvedilol). She also had peptic ulcer disease and osteoarthritis, managed with proton pump inhibitors and analgesics. Prior to referral, several potassium-shifting interventions were attempted – namely, stat doses of 10 IU Actrapid insulin, 50 mL of 50% dextrose, 10 mL of 10% calcium gluconate and nebulised salbutamol. Notably, the referring clinicians were initially unaware of her impaired renal function due to laboratory delays precipitated by a ransomware attack[12]; subsequent laboratory results revealed a serum creatinine of 377 µmol/L, urea of 46 mmol/L and an eGFR of 10.

On arrival at our facility, her vital signs included a bradycardia, 39 bpm and a blood pressure of 95/59 mmHg, with a Glasgow Coma Scale of 13/15 (E4V4M5). Her ECG (Figures 3 and 4) showed a junctional rhythm. Arterial blood gas analysis documented a serum potassium of 8.1 mmol/L. Shortly after arrival boluses of insulin (10 IU stat) and glucose (50 ml 50% dextrose) were given (Table 3), the patient developed a hypoglycaemic episode (glucose 2.1 mmol/L) complicated by seizures, necessitating intubation and mechanical ventilation. Despite prior interventions, hyperkalaemia persisted. Once a normoglycaemic state was re-established (blood glucose 6.6 mmol/L), a continuous insulin infusion was initiated at 0.1 IU/kg/h, accompanied by a titrated dextrose infusion started at 1 ml/kg/h of 50% dextrose to maintain euglycaemia. Her glucose was monitored hourly for the duration of the infusion and the following 4 h. Thereafter monitoring was reduced to 4-hourly checks. This regimen successfully normalised her serum potassium (to 4.1 mmol/L) and resolved the complete heart block within 8 h, thereby obviating the need for emergency dialysis (Graft 2).

Case 3

A 65-year-old female, with a recent history of a gluteal abscess and hypoglycaemia secondary to glimepiride use, developed symptoms of heart block 8 days after admission. Her clinical deterioration was characterised by hypotension and an elevated serum lactate. A blood gas at the time showed a potassium of 8.3 mmol/L. An electrocardiogram performed during this period confirmed the presence of a third-degree heart block (Figure 5). Due to the unavailability of an intensive care unit bed, she was transferred to an admission ward for closer monitoring.

In response to her clinical decline, a continuous infusion of Actrapid was initiated at a rate of 0.1 IU/kg/h, together with a 50% dextrose infusion at 1 ml/kg/h to maintain normoglycaemia. During the treatment course, the patient experienced one symptomatic hypoglycaemic event, with a point-of-care glucose reading of 0.7 mmol/L. This episode was promptly managed with a 50 ml 50% dextrose bolus, (Figure 6). Her potassium normalised after 8 h of the insulin infusion, and she returned to sinus rhythm after 12 h since

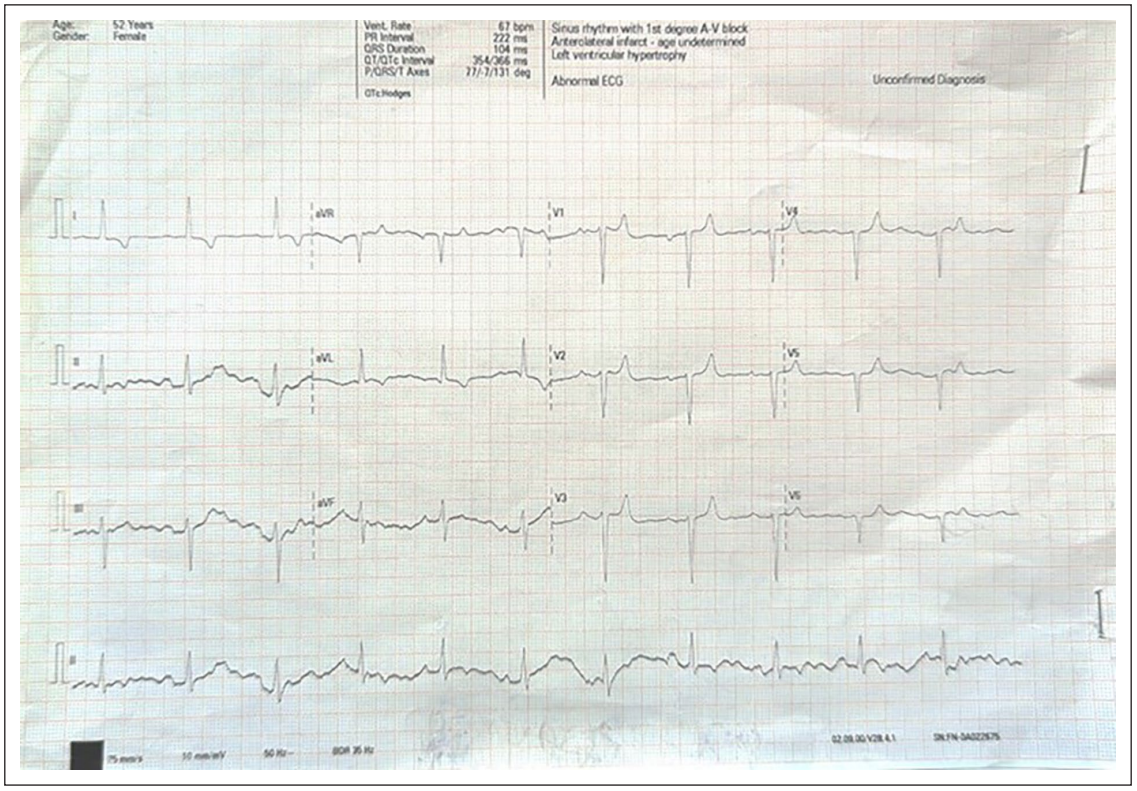


Figure 2. Patient 1: ECG 12 h into insulin infusion. Complete heart block resolved. Serum potassium 3.8 mmol/L.

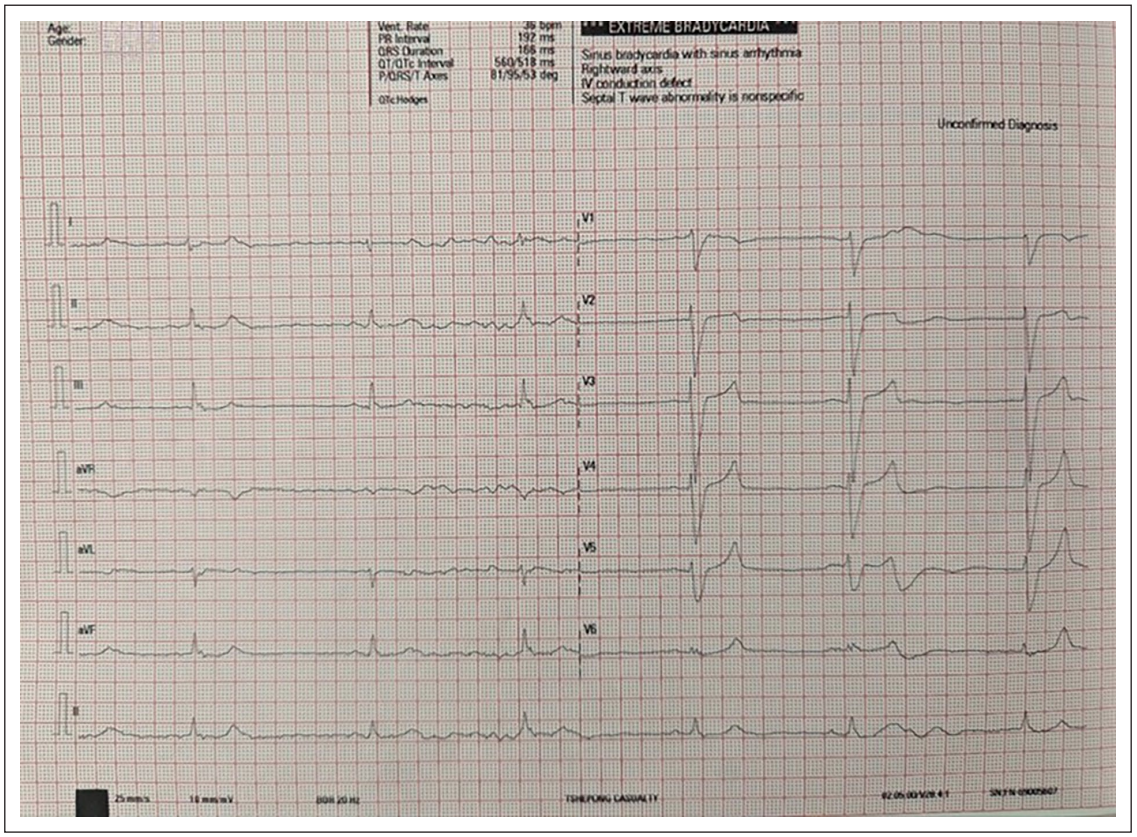


Figure 3. Patient 2: ECG on presentation showing junctional rhythm. Serum potassium of 8.1 mmol/L .

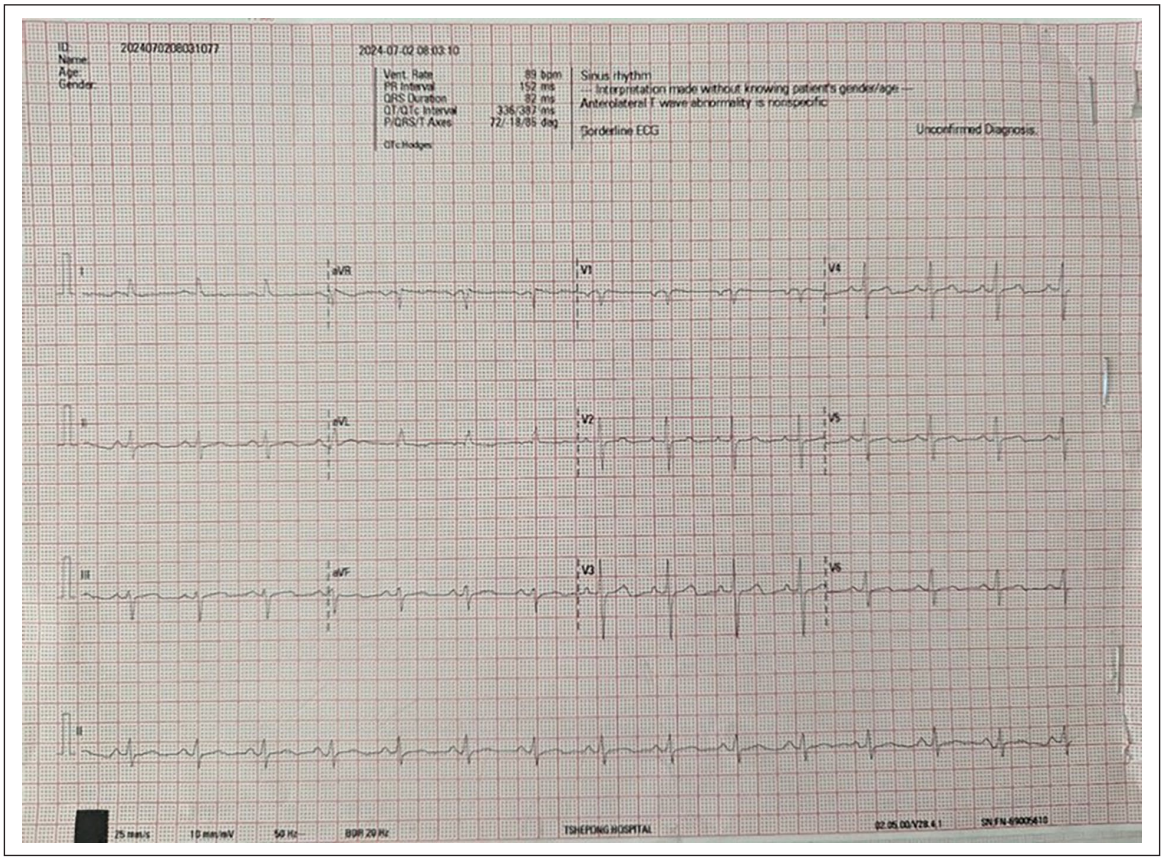


Figure 4. Patient 2: ECG showing resolution of abnormal rhythm 8 h into Actrapid infusion. Serum potassium 4.1 mmol/L.

Table 2. Patient 2: Potassium concentration, ventricular rate, glucose trend per interventional measure over time.

Time:	Presentation (Hour 0)	Hour 2	Hour 4	Hour 5	Hour 13
Potassium (mmol/L)	8.1	8.1	6.4	6.4	4.1
Ventricular Rate (bpm)	36	37	52	59	72
Type of Intervention	10 IU Actrapid 10 ml 10% Calcium carbonate 50 ml 50% Dextrose	10 IU Actrapid 10 ml 10% Calcium carbonate 50 ml 50% Dextrose	None	Start of Actrapid Infusion (0.1 IU/kg/h)	8 h into Actrapid Infusion (0.1 IU/kg/h)
Glucose (mmol/L)	3.7	3.6	2.1	6.6	5.2

beginning the insulin infusion (Graft 3). The patient subsequently made a full recovery.

Discussion

Conventional approaches to managing severe hyperkalaemia – most notably, stat insulin boluses combined with dextrose and calcium gluconate – have long been the mainstay of therapy. However, these methods carry significant risks, as evidenced by our second patient's hypoglycaemic seizures. Rapid bolus administration often results in only transient reductions in serum potassium and is associated with a high incidence of hypoglycaemia, particularly in patients with renal dysfunction where insulin clearance is impaired.[13]

These cases illustrate the potential efficacy of continuous insulin infusion – with concomitant high-dose dextrose titration – in the management of severe need to mention doses of glucose 10% glucose with added 100 ml 50% hyperkalaemia complicated by conduction abnormalities. This approach may serve as a valuable alternative in settings where emergency haemodialysis is not immediately available.

Our use of higher dose insulin with high-dose glucose was adapted from protocols used in betablocker and calcium channel blocker overdose management. The concept of continuous insulin infusion is not entirely novel; it has been successfully applied in the management of calcium channel blocker overdose, where high-dose insulin therapy is used to stabilise haemodynamics and mitigate metabolic effects.[14]

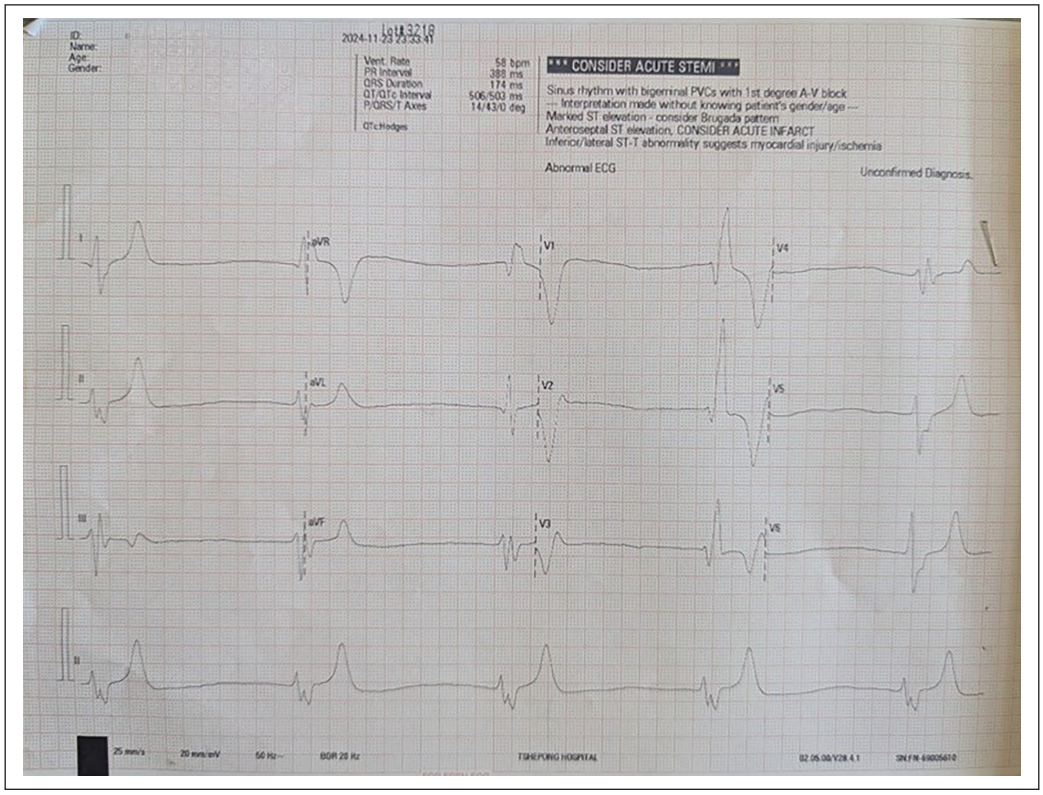


Figure 5. Patient 3: ECG on deterioration showing complete heart block. Serum potassium 8.3 mmol/L.

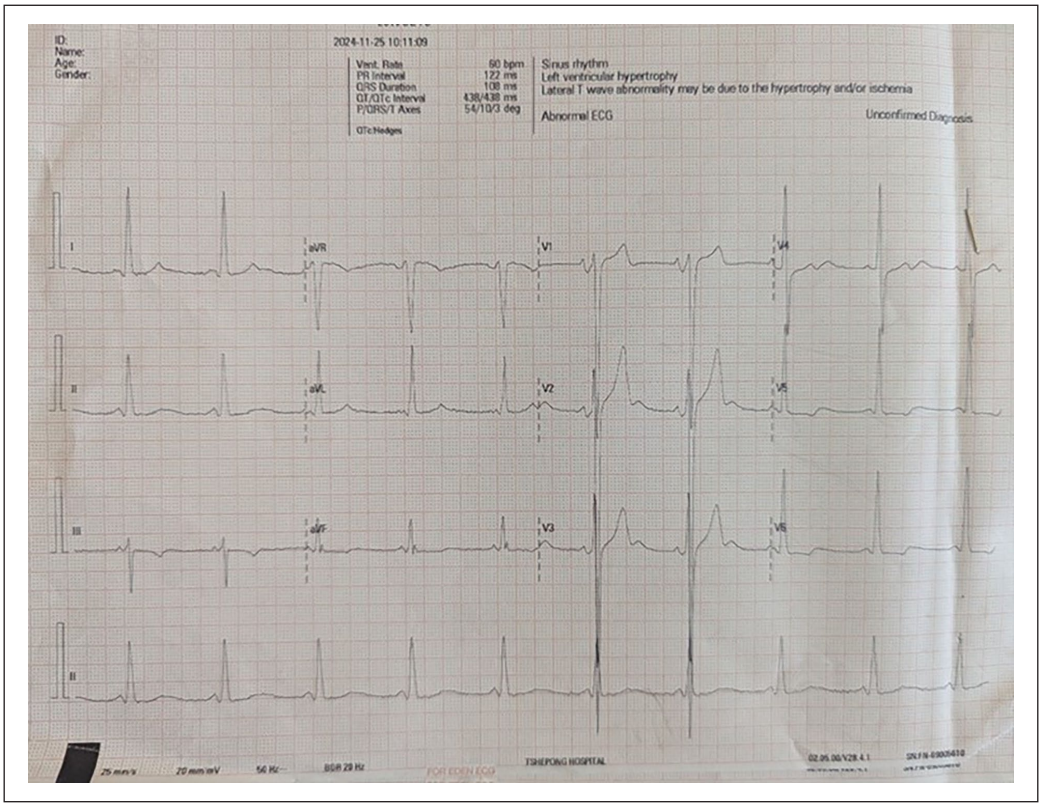
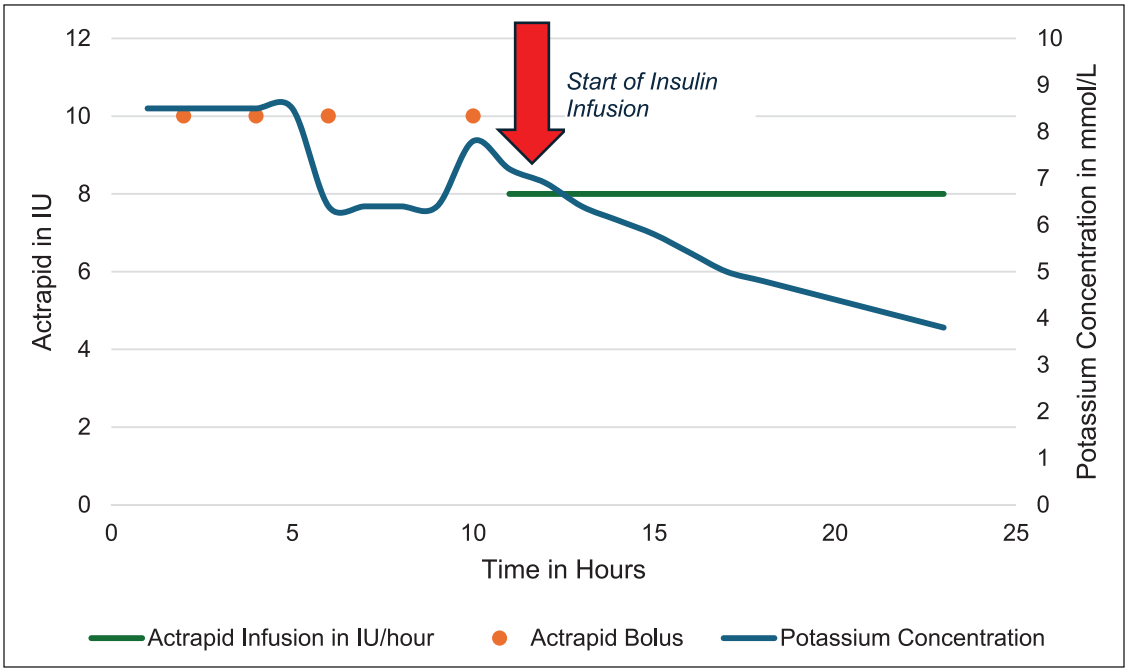


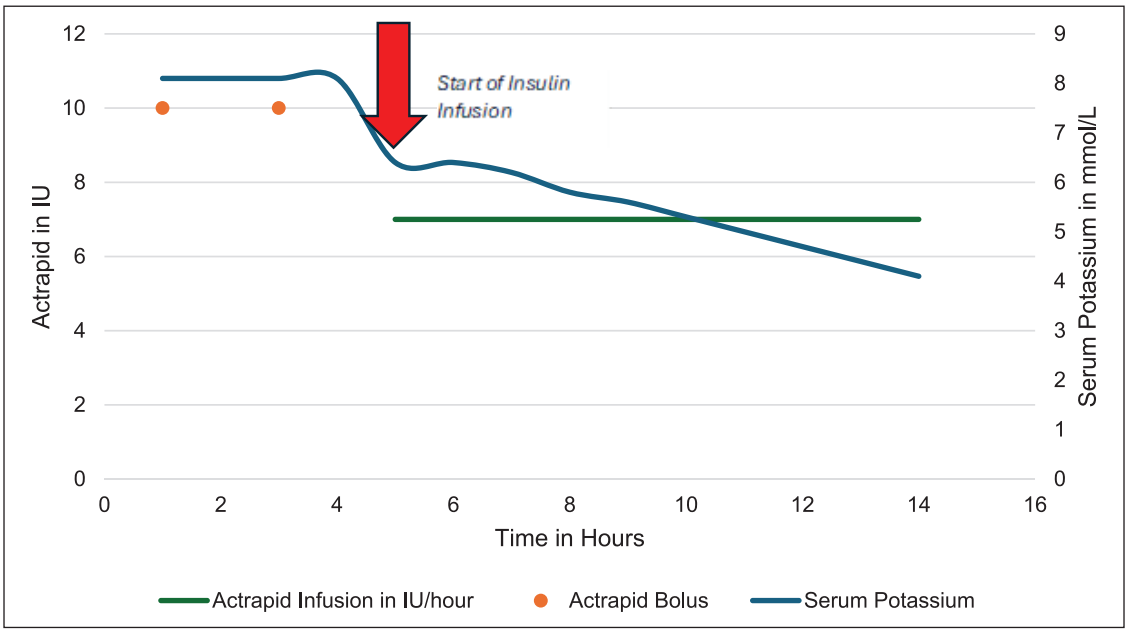
Figure 6. Patient 3: ECG 12 hours into Actrapid infusion showing resolution of complete heart block. Serum potassium 5.3 mmol/L.

Table 3. Patient 3: Potassium concentration, ventricular rate, glucose trend per interventional measure over time.

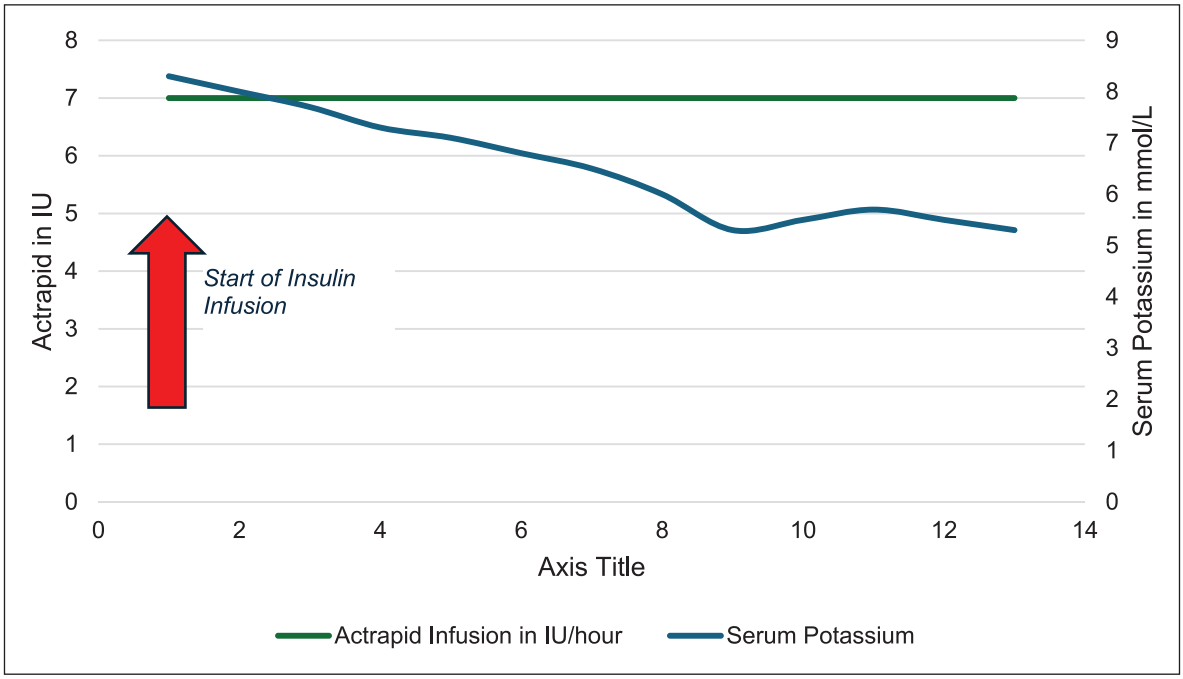
Hours since start of Actrapid infusion	0	3	8	10	12
Potassium (mmol/L)	8.3	7.3	5.3	5.7	5.3
Ventricular Rate (bpm)	27	22	39	41	60
Glucose (mmol/L)	9.0	4.7	11.2	4.3	11.2



Graft 1. Patient 1: Potassium concentration (mmol/L) and Actrapid infusion over time.



Graft 2. Patient 2: Potassium concentration (mmol/L) and Actrapid infusion over time.



Graft 3. Potassium trend case 3.

The continuous insulin infusion strategy employed in our cases offers a slower, more controlled reduction in serum potassium. By initiating an Actrapid infusion at 0.1 IU/kg/h alongside a carefully titrated dextrose infusion, we were able to maintain a steady state of insulin activity. This approach facilitates consistent cellular uptake of potassium, thereby avoiding the abrupt metabolic shifts associated with bolus dosing.[15]

Two of our patients received their insulin infusions in an intensive care unit (ICU) setting, where comprehensive monitoring of cardiac rhythm, serum glucose, and potassium levels was feasible. This allowed for rapid identification and correction of any deviations, contributing to the favourable outcomes observed – namely, the resolution of conduction abnormalities and normalization of serum potassium levels. In the third case, managed on the ward due to ICU bed unavailability, only a single symptomatic hypoglycaemic event was recorded; this episode was promptly addressed, and the patient made a full recovery. Although continuous infusion is not entirely without risk, the more controlled administration appears to reduce the frequency and severity of adverse events compared to conventional bolus strategies.

All three of our patients made a full recovery during their hospital stay and were discharged home without receiving any dialysis.

Conclusion

In summary, while conventional stat insulin bolus administration remains widely used, our case series suggests that continuous insulin infusion – with vigilant dextrose titration – can provide a safer and more controlled alternative for managing severe hyperkalaemia complicated by conduction disturbances. This method minimizes the risk of abrupt hypoglycaemic events and may reduce the need for urgent dialysis, particularly in settings with limited resources. Further prospective studies are warranted to define optimal dosing protocols and to assess the long-term outcomes associated with this therapeutic strategy.

Considering the high prevalence of chronic kidney disease, and that hyperkalaemia is a frequent, life-threatening complication in this population, alternative treatment modalities such as continuous insulin infusion become especially relevant in resource-constrained settings where emergent haemodialysis may not be immediately available.

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Ethical considerations

Our institution does not require ethical approval for reporting individual cases or case series.

Consent to participate

Written informed consent has been obtained from each of the patients included in this case series. A copy of their signed informed consent can be found in their patient record.

Consent for publication

Each of the patients included in the case series has signed informed consent for its publication. A copy of their consent can be found in the patient record.

Informed Consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Author contributions

Conceptualisation: Prof Ebrahim Variava; First draft, editing: Dr Nicole King; Data curation: Dr Mmakwata Lesudi; Review and editing: Prof Ebrahim Variava and Prof Neil Martinson.

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Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Data availability statement

The data supporting the findings of this case series are available within the article and its references.

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