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MANAGEMENT OF RECURRENT SEVERE HYPOKALEMIA IN CORONARY ARTERY BYPASS SURGERY

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ABSTRACT

Background: Intracellular and serum potassium levels have important effects on homeostasis cardiovascular system. Patients undergoing cardiac surgery may develop perioperative hypokalemia. Electrocardiographic (ECG) manifestations of hypokalemia include flattening of the T wave followed by T wave inversion, ST wave depression, prominent U wave, prolongation of the QTc interval, torsades de pointes, to asystole. **Case Presentation:** In this case report, a patient with coronary heart disease who underwent coronary artery bypass surgery, with moderate to severe hypokalemia, before surgery to postoperative care in the ICU. Management of hypokalemia in this patient using potassium chloride 10-50meq/hour (total 250 meq). **Conclusion:** Establishing a diagnosis, looking for underlying disease related to hypokalemia, and a history of preoperative medication should also be considered. Optimal conditions preoperatively can reduce morbidity and mortality. Appropriate management of severe hypokalemia should be known to all medical personnel. Periodic monitoring of ECG and potassium levels is necessary.

INTRODUCTION

Intracellular and blood potassium levels play a crucial role in maintaining cardiovascular system homeostasis.[1] Proper regulation of potassium levels is essential, as many cellular functions are highly sensitive to changes in extracellular fluid potassium concentration.[2] Patients undergoing cardiac surgery may experience perioperative hypokalemia. This condition is associated with the patient's preoperative status and therapies, intraoperative drug use and procedures, as well as postoperative care. The electrocardiographic (ECG) manifestations of hypokalemia include flattened T waves followed by T wave inversion, ST segment depression, prominent U waves, prolonged QTc interval, torsades de pointes, and even asystole.[3]

This case report describes a patient with coronary artery disease who underwent coronary artery bypass grafting (CABG) and developed persistent severe hypokalemia from the preoperative period through postoperative day 0. Despite aggressive correction with high-dose potassium chloride (up to 50 mEq/hour; total 250 mEq), recurrent hypokalemia was associated with serious arrhythmias and postoperative complications. The case highlights the challenges of managing extreme potassium requirements and underscores the need for vigilant perioperative monitoring beyond standard protocols.

CASE PRESENTATION

A 58-year-old male patient, weighing 62 kg, was diagnosed with coronary artery disease and scheduled to undergo CABG at Harapan Kita National Cardiovascular Center. His medical history included hypertension and a minor stroke without residual symptoms. His ongoing medications consisted of furosemide 40 mg once daily, nifedipine extended-release 30 mg once daily, spironolactone 80 mg once daily, ramipril 10 mg once daily, simvastatin 20 mg once daily, isosorbide mononitrate sustained-release 5 mg twice daily, and bisoprolol 10 mg once daily, and a potassium slow-release (KSR) tablet once daily. Laboratory results showed a hemoglobin level of 13.1 g/dL, creatinine 1.26 mg/dL, and a critically low

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potassium level of 2.5 mEq/L. Other laboratory parameters were within normal limits. A chest X-ray revealed no abnormalities.

Coronary angiography revealed significant coronary artery disease. The left main (LM) coronary artery showed 60% stenosis from the mid to distal segments. The left anterior descending (LAD) artery had 90% stenosis from the ostial to proximal segments, 90% stenosis in the mid segment after the first diagonal branch (D1), and 50% stenosis in the distal segment. The left circumflex (LCx) artery had 60% stenosis at the ostium, with multiple stenoses throughout the proximal to distal segments causing 70–90% narrowing. The right coronary artery (RCA) appeared aneurysmal with 50% stenosis in the mid segment, and 80% stenosis in both the posterior descending artery (PDA) and the posterolateral branch (PLB). These findings were consistent with three-vessel disease (CAD 3VD) and left main (LM) involvement.

Echocardiography demonstrated left ventricular (LV) systolic dysfunction with an ejection fraction (EF) of 42%, LV hypertrophy, mild mitral regurgitation (MR), and mild tricuspid regurgitation (TR), with a high probability of pulmonary hypertension. Right ventricular (RV) contractility was preserved. A CT scan of the head showed signs of infarction in the anterior horn of the right lateral ventricle and the left internal capsule.

On physical examination, the patient was alert (compos mentis) with stable vital signs: blood pressure 110/50 mmHg, heart rate 70 beats per minute, respiratory rate 15 breaths per minute, body temperature 36°C, and oxygen saturation 98%. Heart sounds were regular with no murmurs. Breath sounds were vesicular without rhonchi or wheezing. Abdominal and extremity examinations were unremarkable.

In the operating room, standard monitoring was established. A peripheral intravenous line was inserted in the dorsum of the right hand, and an arterial line was placed in the right radial artery. ECG monitoring showed sinus rhythm, with blood pressure at 136/70 mmHg, heart rate 66 bpm, respiratory rate 18 bpm, and oxygen saturation 99%. Anesthesia was induced using midazolam 5 mg, propofol 50 mg, sufentanil 25 mcg, and vecuronium 10 mg. The patient was intubated with an 8.0 mm endotracheal tube placed at 22 cm from the lip. A 7F central venous catheter was placed in the left subclavian vein, and an 8F side port catheter was inserted in the right internal jugular vein.

Anesthesia was maintained with sevoflurane (1–2%), vecuronium (2 mg/hour), and sufentanil (10 mcg/hour). After administration of 18,000 IU heparin, the activated clotting time (ACT) reached 482 seconds. Arterial cannulation was performed at the aorta, and venous cannulation was performed at the right atrium. CPB was initiated. Serial arterial blood gas (ABG) analyses were conducted throughout the surgery (see Tables 1, 2, and 3).

The first correction for hypokalemia (2.5 mmol/L) was carried out using potassium chloride 25 mEq administered at a rate of 10 mEq/hour. The priming fluid for the CPB machine included crystalloids, mannitol, albumin, and sodium bicarbonate. High-potassium cardioplegia solution was used during the ischemic period, which lasted between 12 to 28 minutes.

A second correction was required when the potassium level dropped further to 2.0 mmol/L, using 40 mEq of potassium chloride. Following aortic cross-clamp removal, the patient developed ventricular tachycardia/ventricular fibrillation (VT/VF) and was treated with three direct current (DC) shocks of 5 joules each, after which the heart rhythm returned to sinus rhythm.

During weaning from CPB, the patient experienced hemodynamic instability, prompting a decision to return to bypass. ABG analysis at 14:19 showed anemia with a hemoglobin level of 7.7 g/dL, leading to the transfusion of one unit of packed red blood cells and a third correction of hypokalemia (2.4 mmol/L) with 35 mEq of potassium chloride.

A second attempt at weaning from CPB was successful, with hemodynamic stability achieved. The total CPB duration was 299 minutes, and the total aortic cross-clamp time was 271 minutes. Conventional ultrafiltration (CUF) removed 7400 ml of fluid, and urine output during CPB was 400 ml. The patient's body temperature was gradually reduced to 28.8°C during CPB and rewarmed to 36°C prior to weaning from bypass.

Surgical intervention involved five grafts: LIMA to LAD, saphenous vein grafts (SVG) to LCx, obtuse marginal (OM), diagonal, and distal RCA. Endarterectomy was performed on the vein grafts.

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Transit time flow measurements for all grafts showed satisfactory results.

At the end of the procedure, 200 mg of protamine and 1 gram of calcium gluconate were administered slowly via intravenous infusion over 10 minutes. Activated clotting time (ACT) was measured at 137 seconds.

The patient experienced hemodynamic instability marked by ventricular arrhythmias including ventricular tachycardia/ventricular fibrillation (VT/VF) and recurrent atrial fibrillation (AF). A temporary pacemaker (TPM) and an intra-aortic balloon pump (IABP) were inserted to support cardiac function.

A fourth episode of severe hypokalemia (1.87 mmol/L) was corrected with 50 mEq of potassium chloride administered at a rate of 20 mEq/hour. The patient also presented with anemia and received transfusion of three units of packed red blood cells. Hyperglycemia was managed with an insulin infusion at 10 units/hour.

The patient was transferred to the intensive care unit (ICU) with a hemodynamic profile as follows: blood pressure 100/57 mmHg, Mean Arterial Pressure (MAP) 62 mmHg, heart rate 108 bpm (paced rhythm), and central venous pressure (CVP) 10 mmHg. Vasoactive support included adrenaline at 0.1 mcg/kg/min, noradrenaline at 0.1 mcg/kg/min, and nitroglycerin (NTG) at 0.5 mcg/kg/min. The IABP was set to maximum augmentation with a 1:1 frequency.

Upon arrival in the ICU, the patient was still under the effects of anesthesia, intubated, and receiving mechanical ventilation in volume-controlled (VC) mode with FiO2 50%, respiratory rate 15 breaths/min, and PEEP 5 cmH2O. Inotropic support continued with adrenaline 0.1 mcg/kg/min, noradrenaline 0.2 mcg/kg/min, and NTG 0.5 mcg/kg/min. The IABP remained in use. Postoperative analgesia was provided with morphine at 20 mcg/kg/hour. Hyperglycemia management continued with insulin infusion at 10 units/hour.

Laboratory evaluation in the ICU (see Table 3) revealed severe hypokalemia (1.9 mmol/L). A fifth correction was initiated at a rate of 50 mEq/hour. Two hours after the correction, arterial blood gas analysis showed that the potassium level remained low at 2.1 mmol/L. Rapid potassium supplementation was continued, with a total of 100 mEq of potassium chloride administered.

On the first day of ICU care, the patient developed hyperkalemia, with potassium levels ranging from 5.3 to 6.4 mmol/L, which was managed with appropriate hyperkalemia treatment (See Figures 1 and 2). Potassium levels on subsequent days remained within normal limits (3.4–4.1 mmol/L) until the patient was transferred to the intermediate care unit. No arrhythmias were observed during the ICU stay.

The patient required ventilatory support from the time of ICU admission until day 10 of ICU care. A tracheostomy was performed on day 8 to facilitate prolonged mechanical ventilation.

Antibiotic therapy, nutritional support, and other supportive treatments were administered based on the

patient's clinical needs. Gradual improvement in clinical condition and laboratory parameters was observed throughout the ICU stay.

After 13 days in the ICU, the patient was transferred to the intermediate care unit in stable condition, fully conscious, with a patent airway via tracheostomy, and requiring low-dose inotropic support with noradrenaline at 0.04 mcg/kg/min.

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Table 1. Arterial blood gas (ABG) results before and during cardiopulmonary bypass (CPB)

Time	*Pre CPB* 08.12	*On CPB* 10.38	*On CPB* 11.54	*On CPB* 14.19
pH	7,48	7,49	7,43	7,34
PCO2 (mmHg)	42,7	38,3	34	50,8
PO2 (mmHg)	196	272	255	281
HCO3 mmol/L	32,6	31,5	25	29,5
BE (Blood) (mmol/L)	8,8	6,6	-0,5	3,1
BE (ECF) (mmol/L)	8,9	6,6	-1,2	2,7
SpO2 (%)	99,9	99,9	99	99,9
Hb (g/dL)	13,4	10,1	9	7,7
Hct (%)	40	30	27	23
Na (mmol/L)	141	140	137	141
K (mmol/L)	2,5	2	2,8	2,4
Cl (mmol/L)	98,2	99,9	102	105,2
Mg Ion (mmol/L)	0,57	0,96	1,08	>1,5
Ca Ion (mmol/L)	1,14	1,2	1,23	1,31
Blood sugar (mg/dL)	126	150	181	146
Lactate (mmol/L)	3,8	4,2	3,9	3,7

Table 2. ABG results after weaning from the CPB machine

Time	15.43	16.33	17.19
pH	7,34	7,29	7,18
PCO2 (mmHg)	40,7	41,8	55,5
PO2 (mmHg)	204,7	149,5	99,3
HCO3 mmol/L	21,8	20,7	21,3
BE(Blood) (mmol/L)	-3,1	-4,7	-6,2
BE(ECF) (mmol/L)	-4,3	-5,9	-7,2
Saturasi (%)	99,9	99,3	95,7
Hb (g/dL)	8,5	8,1	9,1
Hct (%)	26	24	27
Na (mmol/L)	144,3	145,9	148,9
K (mmol/L)	1,87	2,48	2,18
Cl (mmol/L)	108,6	109,6	110,7
Mg Ion (mmol/L)	>1,5	1,48	1,31
Ca Ion (mmol/L)	1,27	1,26	1,31
Blood Sugar (mg/dL)	267	277	271
Lactate (mmol/L)	9,5	11,4	11,6

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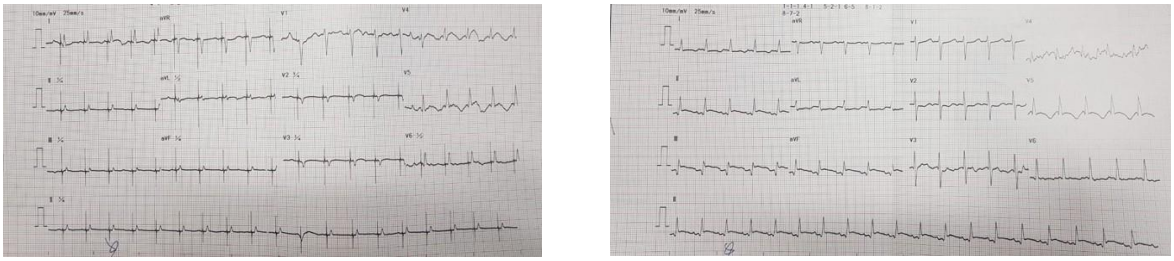
Table 3. Laboratory results during ICU care on day 0, 1, and 2

Time	ICU Day 0		ICU Day 1					ICU Day 2
	19.38	**21.54**	**04.30**	**10.57**	**15.20**	**18.12**	**23.15**	**04.51**
pH	7,31	7,22	7,36	7,36		7,4	7,42	7,42
PCO2 (mmHg)	40,5	46,6	39	44,9		43,6	42,1	41,1
PO2 (mmHg)	191,5	174,9	185,7	146,5		151,1	149,6	149,2
HCO3 mmol/L	20,9	21,5	22,4	25,9		27,4	27,7	27
BE(Blood)(mmol/L)	-5	-4,4	-2,1	0,7		2,8	3,5	2,9
BE (ECF) (mmol/L)	-6,1	-5,5	-3,2	0,2		2,3	3	2,2
Saturasi (%)	99,9	99,7	99,9	99,5		99,8	99,9	99,9
Hb (g/dL)	9,5		8,8			9,8	9,5	10
Hct (%)	28,4		25,7			29	28	30
Leukocyte	23.750		20.390					
Thrombocyte	146.000		140.000					
Na (mmol/L)	150	152	157	157	156	153	152	152
K (mmol/L)	1,9	2,1	6	6,2	6,4	5,8	5,3	4,5
Cl (mmol/L)	112	114	119	115	115	115	112	112
Mg Ion (mmol/L)	1,17	1,09	1,48	0,68	0,69	0,65	0,57	0,55
Ca Ion (mmol/L)	1,3	1,26	1,23	1,21		1,2	1,18	1,19
Blood sugar (mg/dL)	368	331	163	157		162	211	141
Lactate (mmol/L)	10,7	8,4	6,3	3,2		2,6	2,7	2,4
Ureum (mg/dL)			48,1					
BUN (mg/dL)			22					
Procalcitonin (ng/dL)								129,6
Creatinin (mg/dL)			1,98					



(a) (b)

Figure 1. ECG on ICU day 0. ECG recording was performed upon the patient's arrival in the ICU. (a) ECG recording with a temporary pacemaker. (b) ECG recording after the temporary pacemaker was turned off.

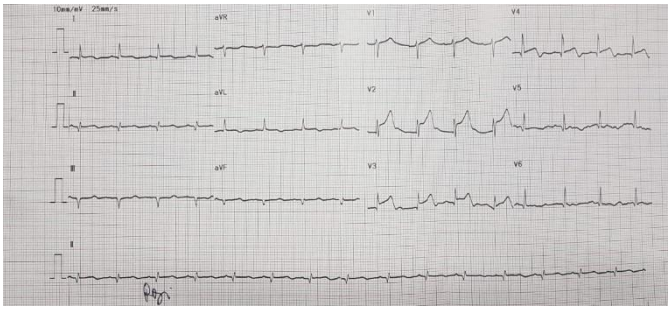


Figure 2. ECG on ICU day 1. ECG recording was performed when the patient was experiencing hyperkalemia (6.0 mmol/L).

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DISCUSSION

In this case report, a patient with coronary artery disease undergoing coronary artery bypass grafting (CABG) experienced moderate to severe hypokalemia, starting preoperatively and continuing throughout the postoperative care in the ICU. Hypokalemia is one of the most common electrolyte disturbances seen in clinical practice. It is classified as mild when serum potassium is 3.0–3.5 mmol/L, moderate when 2.5–3.0 mmol/L, and severe when potassium levels fall below 2.5 mmol/L.[2,3]

Hypokalemia results from an imbalance between potassium input and output. It may be caused by decreased potassium intake, excessive loss through urine or the gastrointestinal tract.[4] Excessive urinary potassium excretion may be due to diuretic use, endocrine disorders such as primary hyperaldosteronism, renal dysfunction, or genetic syndromes affecting renal function. Gastrointestinal losses are usually due to prolonged diarrhea or vomiting, chronic laxative abuse, bowel obstruction, or infection. Intracellular potassium shift can be triggered by insulin administration, sympathetic nervous system stimulation, thyrotoxicosis, or periodic paralysis. Excessive mineralocorticoid effects and penicillin use are also common causes of hypokalemia.[3]

In this case, the patient experienced moderate hypokalemia preoperatively. From the clinical history, there were no complaints of muscle weakness or cardiac arrhythmias. There was no history of nausea or vomiting, but the patient had been receiving diuretic therapy preoperatively with furosemide 40 mg once daily.

Catecholamines play a key role in potassium homeostasis by altering intracellular potassium distribution through both alpha- and beta-receptors. Beta-2 receptors promote cellular uptake of potassium by activating the Na+/K+-ATPase pump.

Beta-adrenergic activity also enhances insulin secretion from the pancreas by direct stimulation and increases glycolysis, which raises blood glucose levels.[4]

Insulin plays a vital role in promoting potassium uptake into cells, helping to prevent hyperkalemia before renal excretion mechanisms come into play.[5,6] It acts on specific cell surface receptors, promoting the translocation of GLUT4 (Glucose Transporter Type-4) to the cell membrane, thereby enhancing glucose uptake in insulin-responsive tissues such as skeletal muscle, adipose tissue, and cardiomyocytes. During this process, potassium uptake is also increased due to enhanced Na+/K+-ATPase activity.[6]

A review article noted a relationship between hypothermia and hypokalemia.[7] Recent case reported that in case of head injury or brain hypoxia, hypothermia can cause hypokalemia through potassium shifts from the extracellular to intracellular or extravascular spaces.[8]

Hypomagnesemia may worsen hypokalemia. Intracellular magnesium normally inhibits potassium secretion through renal outer medullary potassium (ROMK) channels in the distal nephron.[5] Rashmi Soori et al. reported a case of recurrent hypokalemia during mitral valve replacement surgery, with hemodynamic instability during CPB weaning. Laboratory tests revealed hypomagnesemia. In addition to potassium correction, magnesium supplementation was also administered.[9]

Symptoms of hypokalemia include nausea, vomiting, loss of appetite, constipation, generalized weakness, muscle cramps, and palpitations. In severe cases, paralytic ileus, arrhythmias, respiratory muscle paralysis, and even respiratory arrest may occur. The cardiac action potential consists of five phases: phase 0 (rapid depolarization), phase 1 (initial repolarization), phase 2 (plateau), phase 3 (repolarization), and phase 4 (resting membrane potential) (see Figure 3).[10]

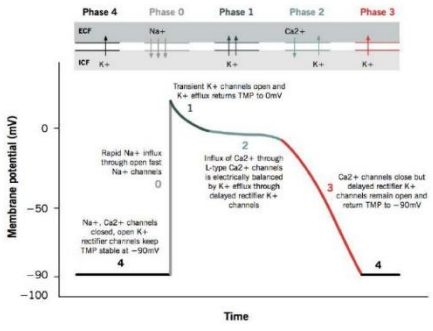

Figure 3. Cardiac muscle action potential

Afterdepolarizations can occur during the repolarization phase of the cardiac action potential. These are classified as early afterdepolarizations (EAD), which occur during phases 2 and 3, or delayed afterdepolarizations (DAD), which occur after

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completion of repolarization in phase 4 (See Figure 4.). Hypokalemia is closely associated with the development of EADs, which can trigger tachyarrhythmias.[11]

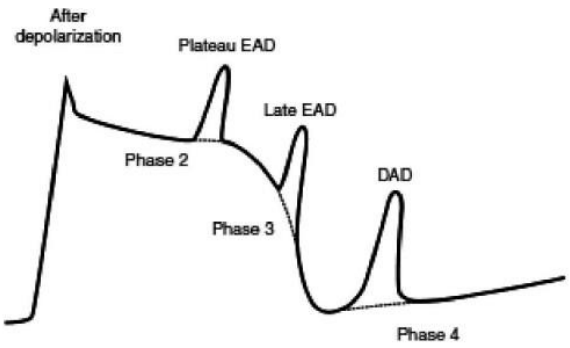

Figure 4. Classification of afterdepolarizations

On electrocardiography (ECG), hypokalemia is typically characterized by flattened T waves followed by T wave inversion, ST segment depression, prominent U waves, QTc interval prolongation, torsades de pointes, and even asystole.[4] Case of hypokalemia where a sudden ECG change was observed—sinus rhythm degenerated into polymorphic wide QRS tachycardia during monitoring.[10] A prominent U wave was seen before transitioning abruptly into torsades de pointes.[11]

Hyperkalemia is another electrolyte imbalance that commonly occurs during CPB. This rise in serum potassium is usually transient and returns to normal once the administration of high-potassium cardioplegia solution is stopped. Management techniques for elevated potassium during CPB include the use of diuretics, glucose-containing fluids, insulin, and zero-balance hemofiltration.[12]

For potassium levels below 4.5 mmol/L, hypokalemia is often treated with intravenous potassium chloride in boluses of 10–20 mmol. Rapid bolus administration of potassium during CPB may cause transient vasodilation. It is essential that potassium correction is completed before weaning from CPB, ideally based on laboratory results taken at a core temperature of at least 35°C.[12]

There are four main goals in the treatment of hypokalemia:

a. Reducing potassium loss
b. Replenishing potassium stores
c. Evaluating for potential toxicity
d. Identifying and treating the underlying cause[13]

Potassium supplementation, either orally or intravenously, should be tailored according to the severity of the hypokalemia. The use of potassium supplements in asymptomatic or borderline cases of hypokalemia remains controversial. However, treatment is recommended when serum potassium levels fall below 3.0 mmol/L.[13]

In high-risk patients—such as those with heart failure, arrhythmias, myocardial infarction, ischemic heart disease, or those receiving digoxin therapy—potassium levels should be maintained within a range of 4.0–4.5 mmol/L. For patients taking diuretics, oral potassium tablets may be prescribed at 40–100 mmol/day, divided into 2–3 doses. This may be combined with potassium-sparing diuretics. Some sources recommend an initial dose of 60–80 mmol/day, which can be increased to 100–150 mmol/day if potassium losses persist.[13]

If the serum potassium level is below 2.5 mmol/L, intravenous potassium replacement is necessary, with continuous ECG monitoring and frequent serum potassium checks. Intravenous administration should preferably be through a large vein to reduce the risk of peripheral vein irritation and pain, with an infusion rate of 10–20 mmol/hour. In life-threatening cases, doses of 40–100 mmol/hour or higher may be used under close monitoring.[14]

Correction of hypomagnesemia should also be considered in cases of recurrent hypokalemia, as magnesium deficiency can exacerbate potassium loss.[13,14]

Compared to standard guidelines, which recommend cautious potassium replacement with maximum infusion rates typically around 10–20 mEq/hour to prevent complications such as hyperkalemia and vein irritation, this case highlights the challenges of managing refractory or recurrent severe hypokalemia during complex cardiac surgery like CABG. While guidelines emphasize gradual correction, continuous cardiac monitoring, and addressing underlying causes, there is limited evidence to guide treatment in cases requiring very high cumulative potassium doses intra- and postoperatively, as observed in this patient.[15,16] This underscores the need for individualized dosing strategies and vigilant monitoring beyond conventional protocols when managing severe hypokalemia in such high-risk surgical settings.

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CONCLUSION

Cardiac surgery patients presenting with hypokalemia require a multidisciplinary approach. Accurate diagnosis, evaluation of underlying conditions contributing to hypokalemia, and review of preoperative medications are essential. Optimizing the patient's condition before surgery can significantly reduce morbidity and mortality. Proper management of severe hypokalemia must be well understood by all healthcare providers involved. Continuous ECG monitoring and regular assessment of serum potassium levels are critical components of care.

In addition to established recommendations, this case report highlights the complexity of managing recurrent, severe hypokalemia in cardiac surgery patients, demonstrating the challenges of balancing aggressive potassium replacement with the risk of subsequent hyperkalemia and arrhythmias. It underscores the critical importance of individualized dosing, frequent electrolyte monitoring, and multidisciplinary coordination in perioperative care to improve patient safety and outcomes beyond standard guideline protocols.

ETHICAL APPROVAL

There is no ethical approval.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization, D.R.P. and H.P.; methodology, D.R.P. and I.M.A.P.; validation, D.R.P., H.P., and I.M.A.P.; formal analysis, D.R.P.; investigation, D.R.P.; resources, H.P.; data curation, D.R.P.; writing—original draft preparation, D.R.P.; writing—review and editing, H.P. and I.M.A.P.; visualization, I.M.A.P.; supervision, I.M.A.P. and H.P.; project administration, D.R.P.

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