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LAPORAN KASUS

Severe Hypokalemia in the Intensive Care Unit: Case Series on Potassium Correction Strategies and Clinical Outcomes

Daniswara, Erlangga Prasamya
Department of Anesthesiology and Intensive Therapy Faculty of Medicine, Public Health, and Nursing Universitas Gadjah Mada/Dr. Sardjito General Hospital, Yogyakarta, Indonesia

Abstract

Hypokalemia is one of the electrolyte disorders that often occurs in intensive care units (ICUs), defined as a serum potassium concentration below 3.5 mmol/L. Its severity is classified as mild (3.0–3.4 mmol/L), moderate (2.5–3.0 mmol/L), and severe (<2.5 mmol/L). Hypokalemia occurs when the body loses too much potassium due to several factors such as vomiting, excessive diarrhea, kidney disease, hormonal disorders, or taking diuretic drugs. Symptoms of hypokalemia generally appear when serum potassium is less than 3.0 mmol/L, ranging from mild weakness to life-threatening cardiac arrhythmias. In critically ill patients, untreated severe hypokalemia can lead to cardiac arrhythmias, respiratory arrest, and renal dysfunction, with a higher risk of complications and mortality in patients with hypotension, diabetes, or chronic kidney disease. This case series involved six ICU patients with severe hypokalemia (K^+ ≤1.8 mmol/L) who underwent rapid potassium correction at a rate of 10–40 mEq/hour adjusted to the patient's clinical severity. In patients with ventricular arrhythmias, initial correction of 2 mEq/minute was followed by 10 mEq over 5–10 minutes. Most patients showed clinical improvement, while worse outcomes were observed in patients with hyperthyroidism and after return of spontaneous circulation (ROSC). This case series highlights the importance of individualized potassium replacement strategies, immediate intervention, and careful monitoring to prevent life-threatening complications and improve outcomes in patients with severe hypokalemia in the ICU.

Keywords: Hypokalemia; intensive care unit; life threatening; potassium correction; prognosis sever

Hipokalemia Berat di Unit Perawatan Intensif: Seri Kasus Strategi Koreksi Kalium dan Luaran Klinis

Abstrak

Hipokalemia adalah salah satu gangguan elektrolit yang sering terjadi di unit perawatan intensif (ICU) dengan konsentrasi kalium serum di bawah 3,5 mmol/L. Tingkat keparahan diklasifikasikan menjadi: ringan (3,0–3,4 mmol/L), sedang (2,5–3,0 mmol/L), dan berat (<2,5 mmol/L). Hipokalemia terjadi ketika tubuh kehilangan banyak kalium akibat berbagai macam faktor seperti muntah, diare berlebihan, penyakit ginjal, gangguan hormonal, atau penggunaan obat diuretik. Gejala hipokalemia umumnya muncul ketika kalium serum < 3,0 mmol/L, mulai dari kelemahan ringan hingga aritmia jantung yang mengancam nyawa. Pada pasien kritis, hipokalemia berat yang tidak diobati menyebabkan aritmia jantung, henti napas, dan disfungsi ginjal, dengan risiko komplikasi. Mortalitas yang lebih tinggi terjadi pada pasien dengan hipotensi, diabetes, atau penyakit ginjal kronis. Seri kasus ini melibatkan enam pasien ICU dengan hipokalemia berat (K^+ ≤1,8 mmol/L) yang menjalani koreksi kalium cepat dengan laju 10–40 mEq/jam, disesuaikan dengan keparahan klinis pasien. Pada pasien dengan aritmia ventrikular, koreksi awal 2 mEq/menit diikuti 10 mEq selama 5–10 menit. Sebagian besar pasien menunjukkan perbaikan klinis, sementara hasil yang lebih buruk terjadi pada pasien dengan hipertiroidisme dan setelah kembalinya sirkulasi spontan (ROSC). Seri kasus ini menggambarkan pentingnya strategi penggantian kalium, intervensi segera dan pemantauan yang cermat untuk mencegah komplikasi yang mengancam nyawa serta meningkatkan hasil pada pasien dengan hipokalemia berat di ICU.

Kata kunci: Hipokalemia; koreksi kalium; prognosis berat; mengancam jiwa; unit perawatan intensif

Korespondensi: Daniswara, dr., SpAn-TI., MMR., Department of Anesthesiology and Intensive Therapy Faculty of Medicine, Public Health, and Nursing Universitas Gadjah Mada, Indonesia/Dr. Sardjito General Hospital, Yogyakarta, Indonesia, E-mail: dr.daniswara@gmail.com

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Introduction

Hypokalemia is one of the most common electrolyte disturbances in the intensive care unit (ICU), with serum potassium concentrations below 3.5 mmol/L.[1] The severity of hypokalemia is classified into three levels: mild (3.0–3.4 mmol/L), moderate (2.5–3.0 mmol/L), and severe (<2.5 mmol/L).[1,2] Causes of hypokalemia include inadequate potassium intake, excessive intracellular potassium absorption, and excessive potassium loss through the kidneys or gastrointestinal tract.[2] Risk factors include nutritional deficiencies, diuretic use, hormonal disorders such as hyperaldosteronism, kidney disease, and chronic diarrhea or vomiting.[2]

Clinical symptoms usually appear when serum potassium levels are below 3.0 mmol/L. Symptoms of hypokalemia vary from mild to severe, including life-threatening cardiac arrhythmias, and generally improve after potassium correction.[3] Additional symptoms include muscle paralysis or weakness (hypokalemic paralysis), gastrointestinal paralysis, and respiratory arrest.[4]

In the ICU, untreated severe hypokalemia can lead to cardiac arrhythmias, respiratory arrest, and renal dysfunction.[3] The risk of complications and mortality increases in patients with hypotension, uncontrolled blood sugar in diabetes, and chronic kidney disease.[1–3] The purpose of this case series is to describe and evaluate potassium correction strategies in patients with severe hypokalemia and their impact on clinical outcomes.

Cases

Case 1

A 33-year-old, 37–38 week pregnant female known case of preeclampsia presented in the hospital with complaints of severe weakness of all four limbs accompanied by atrial fibrillation with a rapid ventricular response- AFRVR, episodes of VT- ventricular tachycardia, and frequent PVCs-premature ventricular contractions. She was found to have initial laboratory findings documenting hypokalemia with a serum potassium level of 1.6 mmol/L. She was given 100 mEq of potassium chloride over one hour. No electrocardiographic changes suggestive of tall T waves noted in hyperkalemia were seen during the correction. Her post-correction improvement revealed an increase in the serum potassium level to 2.4 mmol/L with remarkable clinical improvement evidenced by improvement in the Glasgow Coma Scale- GCS 4-5-6 score and motor function betterment and ECG now showing only sinus tachycardia.

Case 2

A 67 year old female, 48 kg, has presented with severe hypokalemia post-ROSC. She was admitted in the condition of paraplegia requiring norepinephrine infusion at 50 ng/kg/min to maintain BP at 98/60 mmHg. Other vitals were HR -98 bpm, RR- 28 breaths per minute, SpO2- 98% on O2 by non-rebreathing mask at 10 L/min. Electrocardiography has shown markedly prolonged QT interval and laboratory reports have critically revealed a potassium level of 1.4 mmol/L. Femoral central line is inserted and then immediately after that potassium supplementation is started at a rate of forty mEq per hour for two hours under continuous ECG monitoring. No elevated potassium electrocardiographic changes such as tall T waves are noticed during replacement. Following correction, the patient's serum potassium increased to 1.9 mmol/L, accompanied by improvement in her level of consciousness (GCS 4-5-6) and partial recovery of lower limb movement, although the QT interval remained prolonged. The patient remained hemodynamically stable after treatment.

Case 3

A male patient aged 18 years and weighing 89 kilograms presented with profound muscle weakness in all four limbs secondary to severe hypokalemia. He was fully conscious and well-oriented, with a GCS of 4-5-6 but demonstrated marked weakness of the limbs. His vital signs were within normal limits at a blood pressure reading of 127/76 mmHg, pulse rate of 82 beats per minute, respiratory rate at 20 cycles

Table 1 Patients' Characteristics

No.	Age (years)	Initial Clinical Condition	Initial Potassium Level (mmol/L)	Potassium Correction Strategy	Monitoring	Post-Correction Outcome	Final Outcome
1	33	Gravid 37/38 weeks, preeclampsia, tetraparesis, AFRVR, run VT, PVC	1.6	100 mEq in 1 hour	No sign of hyperkalemia (Tall T)	Potassium 2.4, improved GCS and mobility, sinus tachycardia on ECG	Stable
2	67	Post-ROSC, low intake, somnolent, prolonged QT, norepine-phrine 50ng/ kgBW/min	1.4	40 mEq/ hour for 2 hours	No sign of hyperkalemia (Tall T)	Potassium 1.9, improved GCS, still prolonged QT	Stable
3	18	Tetraparesis, GCS 456, NSR on ECG	1.8	10 mEq/ hour for 10 hours	No sign of hyperkalemia (Tall T)	Potassium 2.6, improved motor strength	Stable
4	45	Anorexia, AGE, weakness, NSR on ECG	1.7	10 mEq/ hour for 10 hours	No sign of hyperkalemia (Tall T)	Potassium 2.4, reduced symptoms	Stable
5	27	Hyper-thyroidism, respiratory distress, hypotension, GCS 111	1.7	10 mEq/ hour for 10 hours	No sign of hyperkalemia (Tall T)	Died after 6 hours	Deceased
6	17	Hyper-thyroidism, post-ROSC, apnea, GCS 111	1.4	10 mEq/ hour for 10 hours	No sign of hyperkalemia (Tall T)	Died after 4 hours	Deceased

per minute, and saturation at 97% on 3 liters by nasal cannula. An initial electrocardiogram showed NSR without any arrhythmic changes. Laboratory findings revealed very low critically low potassium levels in the serum, measured at a value of only 1.8 mmol/L. Potassium supplementation through the femoral CVC at a rate of infusion of only 10 mEq each hour for ten hours accompanied by monitoring through continuous ECG observation for possible cardiac changes during correction. Throughout the replacement period, no electrocardiographic signs of hyperkalemia, such as tall peaked T waves, were observed. After correction, the patient's serum potassium increased to 2.6 mmol/L, accompanied by a marked improvement in motor strength and overall neurological function. The patient remained hemodynamically stable and in normal sinus rhythm following therapy.

Case 4

A 65-kg, 45-year-old male presented with complaints of anorexia, generalized weakness, and symptoms of acute gastroenteritis (AGE). On admission, he was alert and fully oriented, with a Glasgow Coma Scale (GCS) score of 4-5-6, though he reported significant weakness in all limbs. His vital signs were stable, with a blood pressure of 105/60 mmHg, heart rate of 72 beats per minute, respiratory rate of 22 breaths per minute, and oxygen saturation

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of 98% on 3L/min nasal cannula oxygen. Electrocardiographic examination revealed a normal sinus rhythm (NSR) without any arrhythmic abnormalities. Laboratory investigations showed severe hypokalemia, with a serum potassium level of 1.7 mmol/L. Potassium replacement therapy was initiated through a femoral central venous catheter (CVC) at a rate of 10 for 10 hours, accompanied by continuous ECG monitoring throughout the infusion. No electrocardiographic signs of hyperkalemia, such as tall peaked T waves, were observed during treatment. Following potassium correction, the serum potassium level increased to 2.4 mmol/L, and the patient exhibited marked clinical improvement, including resolution of weakness and overall symptomatic relief.nction betterment and ECG now showing only sinus tachycardia. The patient stabilized.

Case 5

A 27-year-old male patient weighing 56 kg with a known history of hyperthyroidism and recurrent episodes of hypokalemia presented to the emergency department with respiratory distress and altered consciousness. On arrival, he was unresponsive, with a Glasgow Coma Scale (GCS) score of 1-1-1, and exhibited signs of circulatory compromise, including hypotension with a blood pressure of 86/50 mmHg, tachycardia with a heart rate of 135 beats per minute, and tachypnea with a respiratory rate of 36 breaths per minute. His oxygen saturation was 93% while receiving 15 L/min of oxygen via a non-rebreathing mask. Initial laboratory analysis revealed severe hypokalemia with a serum potassium level of 1.7 mmol/L. The patient was immediately intubated for airway protection, and a femoral central venous catheter (CVC) was inserted to facilitate potassium replacement therapy, which was initiated at a rate of 10 mEq per hour under continuous cardiac monitoring. Despite aggressive resuscitative efforts and correction attempts, his hemodynamic condition continued to deteriorate, and he succumbed six hours after admission.

Case 6

A 17-year-old male patient weighing 50 kg with a known history of hyperthyroidism was admitted to the intensive care unit (ICU) following return of spontaneous circulation (ROSC) after a cardiac arrest. On arrival, he was apneic and unresponsive, with a Glasgow Coma Scale (GCS) score of 1-1-1. His vital signs revealed hypotension with a blood pressure of 82/40 mmHg, tachycardia with a heart rate of 127 beats per minute, and oxygen saturation of 93% while being manually ventilated. Initial laboratory evaluation showed severe hypokalemia, with a serum potassium level of 1.4 mmol/L. The patient was intubated for airway and respiratory support, and a femoral central venous catheter (CVC) was inserted for potassium replacement therapy, which was initiated at a rate of 10 mEq per hour under continuous cardiac monitoring. Despite prompt intervention and supportive management, his clinical condition continued to deteriorate, and he passed away four hours after admission.

Discussions

Potassium correction can be achieved through various methods and modalities.[1,2,5–7] In asymptomatic cases, including mild to moderate hypokalemia, oral potassium supplementation is recommended as a long-term strategy.[1,7] In severe cases, particularlythose with life-threatening arrhythmias on ECG, rapid correction via the intravenous (IV) route is required.[5,7,8] V potassium administration can be performed through either a peripheral or central vein, depending on the correction rate.[5–7]

For correction rates exceeding 10 mEq/hour, IV potassium chloride should be delivered through a central venous catheter.[3,5] In life-threatening hypokalemia with ventricular arrhythmias or cardiac arrest, IV potassium at 2 mEq/min followed by 10 mEq over 5–10 minutes is recommended as baseline therapy.[6] The initial intravenous potassium treatment can be maintained until the life-threatening

condition resolves (cessation of malignant arrhythmias on ECG).[6] The maximum IV potassium infusion rate is 20 mEq per hour.[7] When administering IV potassium via continuous infusion, a glucose-free solution can be used as a diluent.[6,7]

Regardless of the correction strategy, continuous monitoring of serum potassium is essential. Monitoring should include bedside ECG and periodic electrolyte panels.[5] n severe cases requiring rapid correction, electrolyte monitoring must be performed alongside continuous cardiac rhythm assessment.[2,5] One study reported electrolyte checks at 2, 5, 10, 30, and 50 minutes during rapid correction.[5] For correction lasting longer than one hour, serum potassium should be rechecked every 2–4 hours until levels reach ≥3.5 mmol/L.7 This prevents overcorrection, which may result in hyperkalemia, malignant arrhythmias, or cardiac arrest.[7]

In cases 1 and 2, rapid potassium correction (>40 mEq/hour) produced a prompt clinical response, although Case 2 showed persistent ECG abnormalities. In Case 1, IV potassium was administered according to American Heart Association (AHA) guidelines for malignant ventricular arrhythmia. Alternative references suggest an initial regimen of 20 mEq IV over 10 minutes (60–120 mEq/hour), followed by rapid down-titration as the ECG improves and hemodynamics stabilize.[6,9] In contrast, Cases 3 and 4, managed with slower correction (10 mEq/hour for 10 hours), demonstrated gradual but uneventful recovery. Cases 5 and 6 received the same correction rate but had poor prognoses due to hyperthyroidism, low initial GCS, and post-ROSC status. Patients without comorbidities (Cases 3 and 4) achieved full recovery, whereas those with risk factors (Cases 5 and 6) did not. Severe ECG abnormalities were most evident in Cases 1 and 2, especially with potassium <1.5 mmol/L, highlighting the increased risk of fatal arrhythmias.

In case 1, hypokalemia can be caused by the physiological effects of pregnancy, such as hemodilution and increased progesterone levels. Pregnancy can cause a dilutional effect, leading to lower serum electrolyte concentrations. High progesterone levels in pregnancy can activate mutated mineralocorticoid receptors, causing sodium retention and potassium wasting.[10,11]

Case 2 involved prolonged inadequate intake without clear etiology, corrected with 40 mEq/hour due to post-cardiac arrest but without ventricular arrhythmia. Rapid correction in such cases is best delivered via a central venous catheter, as peripheral infusion risks phlebitis and extravasation.[1]

Case 3 involved sudden onset without identifiable cause, managed successfully with gradual correction. Cases 5 and 6, both with hyperthyroidism, deteriorated despite therapy. Thyrotoxic periodic paralysis is a known mechanism of hypokalemia in hyperthyroidism, where increased Na^+/K^+-ATPase activity shifts potassium intracellularly, causing paralysis.[12] Treatment requires simultaneous management of thyroid dysfunction, as potassium replacement alone is insufficient. A patient with serum potassium of 2.0 mEq/L may have a deficit of 400–800 mEq, though accurate quantification is difficult due to transcellular shifts.[1] The variability in clinical response between patients illustrates the need for individualized correction strategies.[1]

One study emphasized the importance of the administration route in achieving effective correction.[6] Potassium administered peripherally may be partly sequestered by surrounding tissues before reaching the circulation, reducing effective cardiac delivery.[6] This explains why even high local concentrations may not necessarily result in systemic hyperkalemia.[6]

The risks of IV potassium therapy include hyperkalemia, arrhythmias, and cardiac arrest.[7] Outcomes depend not only on correction strategy and administration route but also on baseline patient condition and the underlying etiology of hypokalemia.

Conclusion

Correction of severe hypokalemia must be individualized according to the patient's

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clinical status. Rapid, high-dose intravenous potassium is essential in patients with severe electrolyte disturbances complicated by arrhythmias, and must always be accompanied by close monitoring. In patients without significant cardiovascular compromise, a slower correction strategy is generally safer and effective. Prognosis is notably poorer in patients with hyperthyroidism and those admitted post-ROSC, even with timely potassium replacement.

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