Case Report

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Management of Hyperkalemia in Patients with Acute Kidney Injury Post Septic Shock: A Case Report

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ABSTRACT

Introduction: Hyperkalemia often occurs in patients with acute kidney injury due to decreased ability to excrete potassium. This case report aims to describe treating hyperkalemia in patients with acute kidney injury (AKI) after septic shock. **Method:** Case reports detailed the management of hyperkalemia correction using a combination of insulin with 40% Dextrose solution, Ca Gluconate, and hemodialysis therapy. **Results:** A 64-year-old male patient was admitted to the GICU due to septic shock and AKI after undergoing surgery to extract the 36th and 38th teeth accompanied by a retropharyngeal abscess. The physical examination results showed palpitations, tachycardia, hypertension, and grade 1 pitting edema in the upper extremities. The laboratory checks showed hyperkalemia, and an ECG showed sinus tachycardia. The nurse carried out collaborative therapy for potassium correction using a combination of 10-unit insulin and 40% Dextrose, as much as 2 flacons, and Ca Gluconate. The intervention results showed that after 3 hours post-correction, there was a decrease in potassium levels from 5.5 mmol/L to 5 mmol/L. **Conclusion:** Correction of hyperkalemia used a combination of 10-unit insulin and D40%, and Ca Gluconate therapy was the most widely used intervention because it had a rapid onset and hypokalemic effect. Unfortunately, this therapy could not overcome refractory hyperkalemia in these cases, so the patient was advised to undergo hemodialysis therapy.

Introduction

Sepsis is defined as life-threatening organ dysfunction due to an irregular host response to infection (Mahapatra and Heffner, 2020). WHO sets sepsis as one of its global health priorities (Evans, 2018). Sepsis is one of ICU patients' highest causes of morbidity and mortality. Globally, sepsis in hospitalized adults is 270 per 100 000 cases, with an estimated overall mortality of up to 26% (Evans, 2018). Based on data from WHO, in 2017 cases of sepsis in the world reached 48.9 million and caused 20% of deaths at the global level, reaching 11 million (WHO, 2023). This data also shows the fact that as many as 89% of sepsis cases occur in low and middle-income countries (WHO, 2023). In Indonesia, in 2020, 110 patients were diagnosed with sepsis in the ICU (Wicaksono, Adisasmita and Harijanto, 2022). The most frequent sources of infection causing sepsis were pulmonary problems (39.1%) and intra-abdominal disorders (31.8%) (Wicaksono, Adisasmita and Harijanto, 2022).

In sepsis, cytokine release due to activating the systemic inflammatory response alters the patient's blood pressure and decreases organ perfusion (Hotchkiss *et al.*, 2016). As a result of decreased perfusion, AKI (Acute Kidney Injury) is often found in patients treated in the ICU (Ko *et al.*, 2021). Patients with kidney injury will experience a decreased ability to excrete electrolytes, one of which is potassium, so they often experience hyperkalemia. In AKI patients, there is often a decrease in GFR and an increase in tubular flow accompanied by tissue injury, hypercatabolism, and hyperkalemia (Kovesdy, 2014). The hyperkalemia mechanism differs between AKI patients with comorbid CKD and cardiovascular problems. Insulin deficiency and hyperglycemia in diabetic patients cause a decrease in potassium transport into the intracellular space (Watanabe, 2020). Cardiovascular disease, such as hypertension, requires drugs that can cause hyperkalemia due to inhibition of the Na+/K+ ATPase pump, thus exchanging intracellular sodium with extracellular potassium. The drugs most relevant to hyperkalemia are β2-adrenergic receptor blockers and RAAS (Renin-angiotensin-aldosterone system) inhibitors which prevent renin production and reduce the capacity of potassium redistribution in the intracellular space (Kovesdy, 2014).

Hyperkalemia is a medical emergency because it can cause life-threatening arrhythmias (Montford and Linas, 2017). It is due to the role of potassium in forming and spreading impulses in the heart. Hyperkalemia causes an extension of the membrane depolarization time and a shortening of the repolarization time (Martinez-Vea *et al.*, 1999; Gaba *et al.*, 2020) (Martinez-Vea et al., 1999 in Gaba et al., 2020). Acute increases in potassium produce ECG changes. Fatal rhythms such as ventricular tachycardia, ventricular fibrillation, complete heart block, and asystole can occur suddenly, leaving minimal time for treatment (Gaba *et al.*, 2020). Health workers must immediately manage hyperkalemia treatment to reduce the risk of death in patients (Sarnowski *et al.*, 2022). Hyperkalemia is a condition that can cause death, so this case report helps readers understand the condition of hyperkalemia in more advanced ways, to help with prevention, early diagnosis, and determining effective management to reduce more serious complications and of course improve the quality of patient care. From the description that has been presented, this case report aims to describe the process of treating hyperkalemia in AKI patients after septic shock.

Case Presentation

Patient Mr. T (64 years) was admitted to the GICU (General Intensive Care Unit) with a diagnosis of retropharyngeal abscesses before finally having the 36th and 38th teeth extracted. The postoperative patient's condition worsened to experiencing sepsis and developing into septic shock, and the patient was rushed from the postoperative treatment room to the GICU. During the first day of observation, the patient was sedated using Dexemedetomidine 500 gr/8 hours. The patient was installed on a VC SIMV (Volume Control-Synchronized Intermittent Mandatory Ventilation) ventilator, installed DC (Douwer Catheter) and Urometer, OGT (Orogastric Tube), CVC (Central Venous

Catheter) on the Dextra Femoral, CDL (Double Lumen Catheter) on the Left Femoral, and attached monitor leads.

The study focuses on the cardiovascular system; PMI (Point of Maximal impulse) palpation results are palpable with HR in the 65-103 x/minute range and ECG sinus rhythm. On the next day of observation, the patient had tachycardia with HR in the range of 110-123x/min. At its peak, on the third day of observation, the patient's potassium value reached 5.9-6,2 mmol/L, marked by the patient's HR reaching 230x/minute, and the ECG showed sinus tachycardia. The results of the assessment of vital signs during the three days of observation are attached in Table 1. In the first day of observation, the patient's blood pressure which was initially assessed as 145/80 mmHg, continued to increase until it reached 152/90 mmHg until the nurse consulted the doctor on duty to reduce the dose of vasopressin given from 0.04 units/hour to be reduced to 0.02 units/hour. After one hour of observation of the dose reduction, blood pressure gradually decreased to 150/90 mmHg and two hours later reached 144/84 mmHg. The results of the physical examination showed no enlargement of the JVP (Jugular Venous Pressure), no visible shift of the ictus cordis, no peripheral or central cyanosis; anemia in the conjunctival area, no additional heart sounds, PMI (Point of Maximal impulse) tended to palpitations, and there is no dilation of flat sounds in the heart area. Examining the neurological system is difficult except for the oculomotor nerve, which shows the pupil is still isochronous, and the vagus nerve, where the patient's swallowing reflex is absent. The patient has grade 1 pitting edema in both upper extremities and good skin turgor, but muscle strength cannot be assessed because the patient is sedated. Examination results of urine output ± 270 cc/hour with a characteristic dark yellow colour and no findings of abdominal distention.

The patient experienced hyperkalemia on day 1, reaching 5.5 mmol/L (ECG in Figure 1); on day 2, 5.8 mmol; on day 3, 5.9 mmol/L, and continued to rise to 6.2 mmol/L (ECG in Figure 2), RBG (Random Blood Glucose) values in the range of 122 mg/dl in day one, 150 mg/dl in day two, and 93 mg/dl in day three The ECG on the first day showed sinus rhythm, but the second and the last day of observation showed sinus tachycardia. To treat this condition, the patient received therapy D40% 2 flacons+insulin 10-units, and Ca Gluconate 4 gr. In addition, the patient also received other medical therapy such as Fentanyl 25 mg/hour, Dexmedetomidine 0.4 mg/hour, insulin 0.5 units/hour, vasopressin 0.04 units/hour, vasoconstrictor 0.1 mcg, paracetamol 1 gr/6 hours, omeprazole, metronidazole 40 gr/12 hours, meropenem 500 mg/8 hours, levofloxacin 750 mg/24 hours, NaCl 1x0.4 ml, NAC 200 mg/day.

Results

Based on the priority nursing issues raised, nurses collaborate with doctors regarding potassium correction therapy to treat hyperkalemia. Correction is carried out by titration of 40% Dextrose mixed with 10 units of insulin for 30 minutes and titrating Ca gluconate 50 cc. The intervention results showed that after 3 hours post-correction, there was a decrease in potassium levels from 5.5 mmol/L to 5 mmol/L. However, the lab results revealed another problem, the patient had hypocalcemia with a calcium value of only 1.9 mmol/L. Potassium levels rose again one day after correction, where potassium increased to 5.8 mmol/L and sodium increased to 155 mEq/L. The patient also experienced respiratory acidosis with a pH of 7.330, pCO2 48.9 mmHg, and HCO3 26.1 mmol/L. The following day, the patient experienced increased potassium at 5.9 mmol/L and sodium at 149 mEq/L. Nurses carry out 2 cycles of collaborative therapy for potassium correction using D40% + 10 units of insulin with an evaluation of fluctuations in potassium values reaching 6.2 mmol/L marked by changes in the ECG picture to sinus tachycardia and HR reaching 230x/minute. Nurses and doctors have discussions where potassium correction will be carried out again for 3 cycles. If the evaluation results still show refractory, then the corrective action is to do dialysis (hemodialysis). Arterial blood gas analysis ABG) checks are routinely

carried out after corrective therapy to evaluate patients' potassium and blood sugar values. The results of the evaluation, the patient's random blood sugar (RBG) value is normal in the range of 95-122 mg/dl.

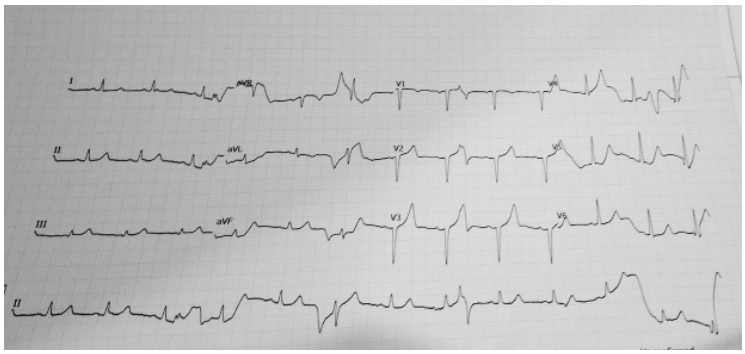

Figure 1. The patient's ECG on the first day of observation, potassium value 5.5 mmol/L

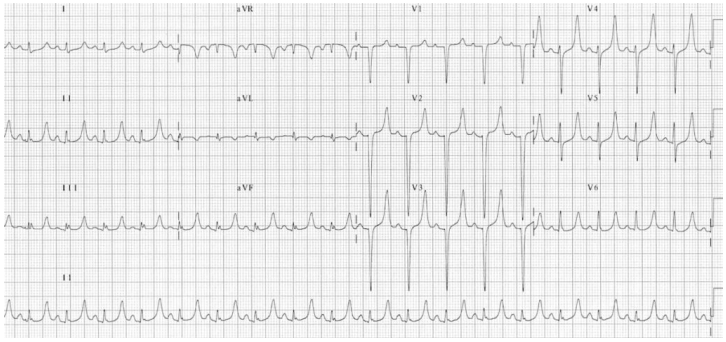

Figure 2. The patient's ECG on the last day of observation, the potassium value was 6.2 mmol/L

Table 1. Vital Signs and Electrolyte Observation Results

Day	Ventilator Mode	BP (mmHg)	HR (x/min)	RR (x/min)	SpO2 (%)	°C	MAP	Na+ (mmol/ L)	K+ (mmol/ L)	RBG (mg/ dl)	Lacta te
D-1	VC SIMV (RR 18, PEEP 6, FiO2 50%)	Lowest: 145/85 Highest: 152/90	65-103	20-60	95-100	35,8 - 38,4	105-110,7	146	5,5	122	2,5
D-2	VC SIMV (RR 20, PEEP 6, FiO2 70%)	Lowest: 110/72 Highest: 150/90	110-123	35-45	95-100	35,7 - 36,5	84,7-110	155	5,8	150	2,1
D-3	PC-AC (FiO2 60% Pinsp 22, Ti 1,30, RR 20 PEEP 6 VT 475 ml/insp)	Lowest: 90/50 Highest: 113/65	123-230	20-25	91-99	35,4 - 38,6	81-63,3	151	5,9-6,2	93	2,7

Discussion

AKI frequently occurs in patients admitted to the ICU as part of the development of multi-organ system failure. Patients with sepsis often experience AKI, with recorded incidence rates reaching 19% in septic patients, 23% in patients with severe sepsis, and 51% in patients with septic shock (Makris and Spanou, 2016). Patients with AKI often experience imbalances in electrolyte and mineral levels (Jung *et al.*, 2016). One of the electrolyte problems often disturbed in AKI patients is hyperkalemia due to impaired excretion of potassium by the kidneys, such as in patients with CKD, heart failure, uncontrolled hypertension, diabetes, or complications from these conditions (Kovesdy, 2014).

Hyperkalemia is when potassium levels are more than 5 mmol/L in adult patients, more than 5.5 mmol/L in pediatric patients, and more than 6 mmol/L in neonatal patients (Simon, Hashmi and Farrel, 2023). Hyperkalemia can be caused by excess potassium intake, impaired potassium excretion, or transcellular shifts (Ohkuma *et al.*, 2022). The etiology of hyperkalemia is often multifactorial, with impaired renal function, drug use, and hyperglycemia being the most common contributors (Fordjour, Walton and Doran, 2014). Intravenous insulin, glucose-inhaled beta-agonists, and dialysis are effective for treating acute hyperkalemia (Sterns, Grieff and Bernstein, 2016).

Many alternative therapies to treat hyperkalemia include administering agents to stabilize the cardiac membrane, distributing potassium into cells, and increasing the elimination of potassium from the body (Moussavi *et al.*, 2019).IV insulin is one of the most widely used treatments for hyperkalemia, considering it has a fast onset and a hypokalemic effect (Nguyen *et al.*, 2011).

Insulin will shift potassium into cells by stimulating the activity of Na^+ and H^+ antiporters on the cell membrane, encouraging the entry of sodium into cells which leads to the activation of Na+ and K+ ATPase, causing electrogenic entry of potassium (Li and Vijayan, 2014). Combining IV insulin doses of 10 units with 2 flacons D40% can reduce potassium levels by 1 mmol/L within 10-20 minutes, and the effect can last within 4-6 hours (Emmett, 2000; Kim and Han, 2002). One of the side effects of IV insulin therapy is hypoglycemia, with a prevalence of 8.7% to 75% (Chothia *et al.*, 2014).Therefore, in the correction of potassium with insulin, it is highly recommended to be accompanied by the administration of dextrose fluids. One of the other effects of hyperkalemia correction therapy with insulin is a shift in intracellular potassium that stimulates Na^+ and H^+, thereby increasing sodium levels that enter cells (Li and Vijayan, 2014).

The study results indicate that it is important to monitor blood glucose intensively after administration of insulin therapy (Apel, Reutrakul and Baldwin, 2014). The same study also explained that adding Dextrose solution to potassium correction therapy using insulin increases the risk of hyperglycemia 1 hour after adding 25 grams of Dextrose. Therefore, Apple recommends checking glucose levels at the beginning and end of potassium correction. If the initial RBG result is ≥250 mg/dL, then there is no need to give a 40% Dextrose solution (Apel, Reutrakul and Baldwin, 2014). Apart from routine RBG checks, another way to reduce the risk of hypoglycemia in potassium correction with insulin is to use a weight-based insulin regimen, especially in patients with low body mass index and older people. The recommended insulin regimen is insulin IV 0.1 unit/KgBW followed by 250 ml of D10W administration in 2 hours (Ljutić and Rumboldt, 1993).

Correction of hyperkalemia using 10 units of IV insulin is considered to reduce serum potassium to 0.6-1.2 mmol/L. The effect can be seen within 15 minutes after administration (McNicholas *et al.*, 2018). The results of a recent study stated that the use of a dose of 5 units of insulin compared to 10 units of insulin with both accompanied by administration of 25 grams of dextrose had results that were not much different (0.91 - 1.16 mEq/L after 5 units vs. 0.89 - 1.08 mEq/L after 10 units of therapy, p=0.89) (Chothia *et al.*, 2014).

The use of dextrose infusion to prevent hypoglycemia after insulin therapy was

described by Coca et al. (2017), where patients were given 10 units of insulin followed by dextrose 50 gram IV therapy (500 mL dextrose 10%), which was infused for 4 hours indicating that only 6 .1% of cases develop hypoglycemia and new onset occurs after 8 hours after the end of dextrose therapy (Coca *et al.*, 2017).

Patients who received sedation therapy experienced hyperkalemia with a value of 5.5 mmol/L and continued to increase to 5.9 mmol/L despite corrective therapy using a mixture of D40% 2 flacons with 10-unit insulin. The results of the first correction only showed a decrease from 5.5 mmol/L to 5 mmol/L, which then rose again to 5.8 mmol/L, and the last day of monitoring reached 5.9 mmol/L marked by cardiac arrhythmias and pulse rates reaching 230x/minute. Seeing this problem, the nurse consults the doctor in charge of the patient and gets advice to do another 3 cycles of correction. The dialysis procedure must be done immediately if the results are still refractory. After correction, the patient is always routinely checked for blood glucose levels using a mixture of D40% with 10 units of insulin. The results of checking the patient's RBG (Blood Sugar) are always within the normal range, from 93 mg/dl-122 mg/dl. During observation, the patient never experienced hypoglycemia due to the hyperkalemia correction that had been made.

Another intervention applied for correcting hyperkalemia is using Ca Gluconate. Unfortunately, research on the effectiveness of Ca Gluconate therapy against hyperkalemia in patients is still being tested on animals. In animals, Ca Gluconate is considered to cause membrane stabilization by reducing cell membrane depolarization, thereby preventing the effects of hyperkalemia on the cardiac conduction (Celebi Yamanoglu and Yamanoglu, 2022). However, research in humans has yet to be proven by clinical trials and is almost entirely discussed only at the level of case reports and studies with low evidence (Zeytin *et al.*, 2010). Recent research states that applying Ca Gluconate in hyperkalemia patients is beneficial in two ways. First, to correct ECG pathology due to hyperkalemia, and second, to ensure existing pathology does not worsen to a fatal rhythm. Potassium correction therapy with insulin is often started simultaneously as Ca gluconate. In correcting hyperkalemia, the Ca gluconate effect begins within 5-10 minutes, while the insulin effect begins within 20-30 minutes and will reach a maximum in 30-60 minutes (Celebi Yamanoglu and Yamanoglu, 2022).

In addition to the therapies already mentioned, hemodialysis is believed to be the fastest way to reduce serum potassium concentrations (Lameire *et al.*, 1998). There is one study that examined 9 hemodialysis patients with a duration of 5 hours and using potassium with a concentration of 1.5 mmol/L, which showed results where 2/3 of extracellular potassium was released after the first hour, and 15% was removed after the last 2 hours (Abuelo, 2015). Approximately 28%-47% of potassium is dialyzed in a standard treatment duration of 4 hours (Basile and Lomonte, 2015).

Seeing that hemodialysis therapy is quite effective, patients who always experience post-correction refractory are planning to undergo hemodialysis as electrolyte correction therapy, especially potassium which is still above the normal threshold with the condition that the patient's urine must be <100 cc/hour. On the last day of observation, the patient's urine output decreased, where at 10.00, it was still in the range of 95cc/hour, and at 13.00, the urine output was only 30 cc/hour, so the patient was consulted for hemodialysis therapy.

Conclusions

Hyperkalemia in AKI patients occurs due to a decrease in the ability to excretion of potassium. Combining insulin 10 units with D40% 2 flacons and Ca Gluconate is the most widely applied choice to treat hyperkalemia because of its rapid onset and hypokalemic effect. If these interventions still do not produce significant results, the last option for correcting potassium or other electrolytes is to use hemodialysis therapy as given to the patient. In the ICU setting, nurses can perform a collaborative intervention. Before correction therapy, nurses can assess potassium and glucose values, carrying out corrective therapy, to post-correction monitoring. In addition, by compiling

this case report, it is hoped that readers, especially nurses, will be motivated to develop research on the same topic or more complex topics with designs that have a higher level of evidence, such as case studies, RCTs, and systematic reviews so its effective to be applied on the patient.

Conflict of Interests

There was no conflict of interest during the process of writing this case report.

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