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Review

The Impact of Hyperkalemia on Cardiac Function in ICU Patients

Mazen Alaadeen Nassar[1], Ahmad Hassan Albargi[2], Amani Mohammed Alsulami[2], Hissah Saud Alotaibi[3], Areej Ahmad Abulela[4], Aghsan Adel Badawe[5], Raghad Hassan Almusui[6], Abdullah Mohammed Alnejidi[7], Mohammed Hussain Al bather[7]

[1] *ICU, Al Thager Hospital, Jeddah, Saudi Arabia*
[2] *Emergency Department, Al Thager Hospital, Jeddah, Saudi Arabia*
[3] *College of Medicine, Visions Colleges, Riyadh, Saudi Arabia*
[4] *ICU, King Abdullah Medical City, Mecca, Saudi Arabia*
[5] *College of Medicine, Al Rayan Colleges, Medina, Saudi Arabia*
[6] *Department of Emergency Medicine, King Faisal Medical Complex, Taif, Saudi Arabia*
[7] *College of Medicine, Imam Abdulrahman Bin Faisal University, Dammam, Saudi Arabia*

Correspondence should be addressed to **Mazen Alaadeen Nassar**, ICU, Al Thager Hospital, Jeddah, Saudi Arabia, email: maze1985@hotmail.com

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Abstract

Hyperkalemia, characterized by elevated serum potassium levels, poses a critical risk to ICU patients, especially those with chronic kidney disease, heart failure, or receiving renin-angiotensin-aldosterone system inhibitors. This review examines hyperkalemia's impact on cardiac function, focusing on its role in arrhythmogenesis, myocardial conduction changes, and ECG abnormalities such as peaked T waves, QRS widening, and bradycardia, which can escalate to fatal outcomes if untreated. The U-shaped mortality curve associated with hypo- and hyperkalemia highlights the risks of both extremes of potassium levels. Major contributors to hyperkalemia in critically ill patients include impaired renal clearance, cellular potassium release due to tissue injury, and medications. Diagnostic complexities arise due to the variability of ECG findings and overlapping symptoms with other conditions. Effective management strategies discussed include calcium for myocardial stabilization, insulin and albuterol to shift potassium intracellularly, and potassium-binding agents like patiromer and sodium zirconium cyclosilicate. Further research is essential to refine treatment thresholds and enhance management strategies for hyperkalemia in ICU patients.

Keywords: *Hyperkalemia, ICU, cardiac function, electrolyte disturbance, myocardial conduction, ECG abnormalities*

Introduction

Hyperkalemia is a potentially dangerous electrolyte disturbance. The term "hyperkalemia" is derived from "hyper-" meaning "high," "kalium" meaning "potassium," and "-emia," which refers to a blood condition (1). Normal serum potassium is typically defined between 3.5 and 5.3 mmol/l (2). While a universal definition for hyperkalemia is lacking, the European Resuscitation Council characterizes it as a plasma potassium level exceeding 5.5 mmol/L, with severe hyperkalemia defined as levels above 6.5 mmol/L (3). Hyperkalemia is linked to adverse outcomes across various clinical contexts, particularly in acutely ill patients (4, 5). In cases of acute hyperkalemia, primary mortality risks include cardiac rhythm and conduction abnormalities. However, the specific causes of death among hyperkalemic patients are not well documented, and the direct relationship between hyperkalemia and clinical outcomes remains debated (3).

Previous studies involving patients with both acute and chronic cardiovascular conditions have shown a U-shaped link between admission potassium levels and mortality in those hospitalized with heart failure (HF) or myocardial infarction (6, 7), and in general intensive care unit (ICU) patients (8, 9). Research on patients with acute myocardial infarction has repeatedly found hyperkalemia to be an indicator of increased mortality, though findings on hypokalemia's relationship with mortality are more varied (7). While many earlier studies in acutely ill cardiac patients concentrated on a single primary admission diagnosis, today's cardiac intensive care unit (CICU) patients frequently present with complex or mixed disease profiles. Given these evolving characteristics of the CICU patient population, there is a need to reassess established risk predictors for the contemporary CICU (10, 11).

In effective doses for treating HF, inhibitors of the renin-angiotensin-aldosterone system (RAAS) such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin-receptor blockers, and mineralocorticoid receptor antagonists reduce morbidity and mortality but can elevate serum potassium levels, even with concurrent use of potassium-depleting diuretics. Optimal medical management of symptomatic HF often involves multiple neurohormonal antagonists, which independently and collectively increase the hyperkalemia risk. The Candesartan in Heart Failure-Assessment of Reduction in Mortality and Morbidity (CHARM) Program studied the effects of the angiotensin receptor blocker (ARB) candesartan, both alone and with an ACE inhibitor, in a wide range of symptomatic HF patients, encompassing those with both reduced and preserved left ventricular ejection fraction (LVEF) and those receiving varied neurohormonal antagonist combinations (12).

Hyperkalemia is a frequently encountered yet potentially life-threatening electrolyte disturbance in patients with chronic kidney disease (CKD), HF, diabetes mellitus (DM), or hypertension. Its reported incidence in the general population ranges from 2.6% to 7%,4–7 and can be as high as 73% in patients with CKD (13).

Patients admitted with acute HF face significant risks of mortality and rehospitalization (14). Among the various metabolic disturbances, serum potassium levels (SK^+) have garnered attention in this context. In acute HF patients, SK^+ is influenced by several factors that may be altered, such as renal function and the activation of the renin–angiotensin–aldosterone system (15).

Methodology

This study is based on a comprehensive literature search conducted on 30 October 2024, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the impact of hyperkalemia on cardiac function in ICU patients. There were no restrictions on date, language, participant age, or type of publication.

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Discussion

Potassium (K^+), the body's primary cation, has a total reserve of 3000–4000 mmol, with only 2% in the extracellular space. Hyperkalemia increases mortality in the general population, and in cardiac, renal, and critically ill patients. A U-shaped mortality curve exists for hypo- and hyperkalemia, though treatment thresholds remain debated (3).

Cardiac Manifestations of Hyperkalemia

Hyperkalemia rarely presents symptoms like weakness, flaccid paralysis, paresthesias, or decreased tendon reflexes; instead, clinical signs often remain mild until cardiac rhythm or conduction disturbances arise. Elevated extracellular potassium significantly impacts myocardial electrophysiology, leading to intracardiac conduction abnormalities. As extracellular potassium levels rise, the potassium gradient across the cell membrane reduces, diminishing the resting membrane potential and increasing potassium membrane permeability, reducing resistance, and increasing repolarizing currents. These effects shorten the transmembrane action potential, with lower potassium elevations initially accelerating conduction but higher levels ultimately slowing it (3).

Characteristic electrocardiogram (ECG) signs in hyperkalemia include indicators of heightened excitability, such as peaked T waves, PR prolongation, loss of P waves, QRS widening, and bradycardia; severe cases may even show a sine wave rhythm due to prolonged diastolic depolarization. The correlation between potassium levels and ECG abnormalities, however, is inconsistent; severe hyperkalemia may have minimal or atypical ECG findings, including nonspecific ST segment changes or pseudo-Brugada syndrome (wide QRS, ST elevation, J-point elevation, T-wave inversion). In contrast, moderate hyperkalemia (<6 mmol/L) can produce life-threatening ECG changes. ECG changes in hyperkalemia are influenced by rapid plasma potassium fluctuations, the myocardial potassium gradient, other ions (e.g., sodium, calcium), and existing cardiac conditions (16). Retrospective studies show higher mortality in hyperkalemic patients with abnormal ECG findings (17). Chronically dialyzed patients, however, may not show ECG changes despite elevated potassium levels, suggesting that therapeutic decisions should prioritize ECG findings over serum potassium levels alone.

Causes of hyperkalemia in acutely ill patients

The factors contributing to hyperkalemia can be grouped into three categories: altered renal potassium clearance (e.g., chronic kidney disease, acute kidney injury, renin–angiotensin–aldosterone system inhibitors), potassium release from cells (e.g., hemolysis, rhabdomyolysis, tissue injury), and impaired transfer of potassium into cells (e.g., acidosis, insulin deficiency, β-adrenergic blockers, heparin) **(Table 1)** (3).

Hyperkalemia in patients with normal renal function is unusual and, without ECG changes, should prompt investigation for pseudo-hyperkalemia, a falsely elevated potassium reading often due to blood sample hemolysis, which does not reflect true plasma levels. While drugs like potassium supplements, penicillin G, digoxin, NSAIDs, RAAS inhibitors, amiloride, triamterene, trimethoprim, and pentamidine can contribute to hyperkalemia, they are seldom the only cause in acute settings. Potassium primarily exists within cells, so impaired cellular uptake is a significant factor in hyperkalemia (18). Hyperchloremic acidosis, common in acutely ill patients (19), is explained by Stewart's theory, where acid-base balance is defined by the strong ion difference (SID), calculated between major cations (e.g., sodium) and anions (e.g., chloride) (20). In hyperchloremic acidosis, mineral acids like hydrochloric acid reduce extracellular pH, inhibiting Na+–H+ exchange and decreasing Na^+–K^+ ATPase activity, limiting potassium transport into cells and raising extracellular potassium levels. Balanced solutions with physiological chloride levels (e.g., Ringer's lactate) rather than NaCl 0.9% help lower serum potassium (3). Organic acidosis (e.g., lactate, phosphate) affects hyperkalemia less since these acids diffuse into cells, maintaining ATPase function and limiting extracellular potassium (21).

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Succinylcholine, used for rapid intubation, induces potassium efflux by muscle depolarization, raising serum potassium by 0.4 mmol/L; it is contraindicated in hyperkalemic patients or those with conditions like anatomical denervation or prolonged immobilization, where rocuronium is preferable (3).

Table 1. Mechanisms contributing to the development of hyperkalemia (3)

Mechanism	Description	Examples
Increased Extracellular K^+	Elevated potassium levels in extracellular fluid	Tissue injury, hemolysis, rhabdomyolysis, tumor lysis syndrome
	K^+ shift to extracellular space	Mineral acidosis (e.g., hyperchloremic acidosis), succinylcholine
	Impaired potassium entry into cells	Diabetes mellitus, hyperglycemia, hypertonicity, β_2-receptor antagonists, aldosterone blockers, cardiac glycosides
	High acute potassium load	Increased dietary intake, blood transfusion, injection error
Decreased K^+ Elimination	Reduced potassium clearance	Acute kidney injury (AKI), hypovolemia, sepsis, acidosis treatment, Renin-Angiotensin-Aldosterone System (RAAS) inhibitors, calcineurin inhibitors, cardiac glycosides

Note: K^+ = potassium, RAAS = renin–angiotensin–aldosterone system.

Diagnosis

When diagnosing hyperkalemia, an initial ECG of a patient with a potassium level of 5.7 mmol/l may show prominent T waves and reduced P waves. Since elevated potassium can result from hemolysis in the initial sample, repeating the potassium measurement is essential. Normal potassium levels range from 3.5 to 5 mmol/l. Diagnostic tests often include blood tests for kidney function (e.g., creatinine, blood urea nitrogen), glucose, and occasionally creatine kinase and cortisol. Additionally, calculating the trans-tubular potassium gradient may help identify the hyperkalemia's cause. An ECG can assess the risk of abnormal heart rhythms, while a thorough medical history will likely focus on kidney disease and medications like potassium-sparing diuretics, common contributors to hyperkalemia (22).

Electrocardiogram (ECG) findings

ECG findings are critical in assessing hyperkalemia severity, showing characteristic changes from mild to severe stages, such as PR prolongation, QRS widening, and tented T waves. However, potassium levels required for these changes vary due to factors like electrolyte and catecholamine levels. **Table 2** outlines key ECG changes linked to hyperkalemia and the limitations in its diagnostic reliability.

Management

The acute management of hyperkalemia involves multiple strategies, including myocardial stabilization, shifting potassium intracellularly, enhancing excretion through urine or feces, and hemodialysis for refractory cases. Calcium is a primary intervention to mitigate hyperkalemia's effects on cardiac cells, especially in severe cases with significant ECG changes like absent P waves, peaked T waves, or widened QRS complexes (25). The European Resuscitation Council and the American Heart Association recommend using calcium for such cases. Commonly administered as calcium gluconate or calcium chloride, these forms differ in calcium content; calcium gluconate (1–3 g) contains 4.6 mEq of calcium per gram, whereas calcium chloride (500–1000 mg) contains 13.6 mEq per gram (26, 27). Calcium offers myocardial protection within 5 minutes, although repeated doses may be necessary if severe ECG abnormalities persist (26). Potential side effects include hypotension, peripheral vasodilation, and

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bradycardia, with calcium chloride posing a greater risk of tissue necrosis if extravasation occurs, thus requiring central vein administration. Calcium should not be mixed with bicarbonate due to precipitation risk and is cautiously used in patients with digoxin toxicity (25).

Table 2. Key ECG changes and limitations for hyperkalemia diagnosis

ECG Findings	Description
Mild to Moderate Hyperkalemia	Causes PR interval prolongation and peaked T waves (22).
Severe Hyperkalemia	Leads to a widened QRS complex, which can progress to a sinusoidal shape.
Potassium Channel Effects	Elevated potassium accelerates membrane repolarization, causing tented T waves, while membrane depolarization inactivates sodium channels, leading to slower conduction, smaller P waves, and wider QRS (23).
Classic Progression of ECG Changes	Peaked T waves → Shortened QT interval → Prolonged PR interval → Widened QRS complex → Loss of P wave → Sinusoidal QRS. Bradycardia and junctional rhythms increase adverse outcome risks (24).
Potassium Threshold for ECG Changes	Variable; depends on other electrolytes and catecholamine levels.
Reliability	ECG findings are inconsistent for hyperkalemia diagnosis; a review found peaked T waves in only 3 out of 90 cases, with sensitivity from 0.18 to 0.52.

Insulin and albuterol are frequently used to shift potassium into cells. Insulin activates sodium-potassium ATPase, moving potassium into cells with an onset of 15 minutes, a peak effect around 45–60 minutes, and duration lasting up to 6 hours (28, 29). Since hypoglycemia is a primary side effect, it is often managed with IV dextrose, and weight-based dosing (0.1 units/kg) may further reduce hypoglycemia risk. Albuterol, a beta-2 agonist, also promotes potassium shift by ATPase activation, lowering potassium by roughly 1 mmol/L within 15–30 minutes. Both nebulized and IV forms are effective, though IV albuterol can increase the risk of tachycardia. Sodium bicarbonate is sometimes used but is more effective in cases of hyperkalemia accompanied by metabolic acidosis, as its use in stable hyperkalemia is generally not recommended (25).

Potassium elimination in acute hyperkalemia management commonly includes diuretics and potassium-binding resins. Loop diuretics are often used, promoting potassium excretion by blocking sodium reabsorption in the loop of Henle and enhancing distal excretion (30, 31). They can be combined with thiazide diuretics for a synergistic effect but are contraindicated in hypovolemic patients and may exacerbate AKI (32). Acetazolamide also increases potassium excretion through bicarbonate delivery to the distal nephron but is not recommended alone due to metabolic acidosis risk, potentially worsening hyperkalemia (33).

Sodium polystyrene sulfonate (SPS), or Kayexalate, is a longstanding adjunct that binds potassium in the large intestine in exchange for sodium; 1-gram exchanges 0.5–1 mEq of potassium (34, 35). Recent studies support its effectiveness in lowering potassium, but acute use is limited by side effects like hypokalemia, hypomagnesemia, and severe gastrointestinal issues, especially in patients with slowed bowel transit (25). Patiromer, FDA-approved for chronic hyperkalemia, effectively reduces potassium by 1 mEq/L within three days at typical doses of 8.4-25.2 g/day. It is generally well-tolerated, though its role in acute hyperkalemia requires further investigation. Sodium zirconium cyclosilicate (ZS-9), another potassium binder, reduces potassium in a dose-dependent manner,

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with the HARMONIZE trial showing effects within an hour and normalization in 2.2 hours, with common adverse effects being edema and gastrointestinal symptoms (25).

Prevention

To prevent hyperkalemia recurrence, strategies generally include reducing dietary potassium intake, discontinuing any causative medication, and/or adding a diuretic, such as furosemide or hydrochlorothiazide (22). Sodium polystyrene sulfonate combined with sorbitol (kayexalate) is sometimes used long-term to keep serum potassium levels low, although the safety of prolonged sodium polystyrene sulfonate use remains uncertain for this purpose. Common high-potassium foods to limit include vegetables like avocados (36).

Future directions

Future research on hyperkalemia in ICU patients should focus on establishing precise treatment thresholds and protocols tailored to critically ill populations. Key areas for investigation include determining the most effective dosing and timing for therapies such as calcium, insulin, and potassium binders, particularly in acute hyperkalemia. Additionally, exploring newer potassium binders like patiromer and sodium zirconium cyclosilicate for rapid potassium reduction in emergency settings could improve immediate outcomes. Understanding the relationship between hyperkalemia and cardiac electrophysiology—especially with variable ECG presentations—may aid in refining diagnostic approaches and risk stratification methods. Since hyperkalemia recurrence is common in patients with chronic kidney disease or heart failure, studies should also investigate long-term prevention strategies, including dietary management, medication adjustment, and innovative drug combinations. Developing targeted guidelines for hyperkalemia in ICU patients could significantly enhance cardiac outcomes, reduce mortality, and provide a clearer framework for managing this high-risk condition.

Conclusion

Hyperkalemia significantly impacts cardiac function in ICU patients, increasing mortality risk through arrhythmias and conduction disturbances. Effective management using calcium, insulin, and potassium binders—can mitigate these risks, but optimal treatment thresholds remain unclear. Further research is essential to establish precise protocols for hyperkalemia management, improving outcomes for critically ill patients facing this electrolyte imbalance.

Disclosure

Conflict of interest

There is no conflict of interest.

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Ethical Consideration

Not applicable.

Data availability

Data that supports the findings of this study are embedded within the manuscript which is based on a comprehensive literature search conducted on October 2024, in the Medline and Cochrane databases.

Author Contribution

The authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

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