

Review

Diagnosis of and Management Strategies for Electrolyte Imbalances in Critically Ill Patients

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Abstract

Electrolyte imbalances are a frequent and serious concern in critically ill patients, often contributing to morbidity and mortality in this population. These imbalances, including disturbances in sodium, potassium, calcium, and magnesium levels, can arise from various factors such as underlying disease processes, therapeutic interventions, and fluid shifts within the body. Understanding the pathophysiology of these disorders is crucial, as they can have profound effects on organ systems, particularly the cardiovascular, neuromuscular, and renal systems. Accurate diagnosis of electrolyte disorders in critically ill patients relies on a combination of laboratory tests, clinical assessment, and continuous monitoring. Laboratory measurements of serum electrolytes and arterial blood gases provide essential data, while electrocardiography (ECG) and point-of-care testing offer real-time insights into the patient's status. The interpretation of these results must consider the dynamic nature of the patient's condition, as well as the influence of medications and fluid management. Management strategies for electrolyte imbalances are multifaceted and must be tailored to the specific disorder and patient context. For hyperkalemia, immediate interventions include the administration of calcium gluconate and measures to shift potassium intracellularly, while promoting its elimination. Hyponatremia management requires careful correction to avoid complications such as osmotic demyelination syndrome, often involving the use of hypertonic saline or fluid restriction. Hypocalcemia treatment focuses on intravenous calcium supplementation, with attention to underlying causes like hypomagnesemia. Magnesium imbalances, particularly hypomagnesemia, necessitate intravenous magnesium sulfate administration, with vigilant monitoring to prevent overcorrection. Electrolyte management in critically ill patients is complex, requiring prompt recognition and appropriate interventions to mitigate risks. An integrated approach that includes ongoing research and clinical vigilance is essential for improving patient outcomes and reducing the burden of these life-threatening imbalances in critical care settings.

Keywords: *electrolyte imbalances, critical care, hyperkalemia, hypocalcemia, magnesium disorders*

Introduction

Electrolyte imbalances are a common and potentially life-threatening complication in critically ill patients, often resulting from underlying disease processes, therapeutic interventions, or a combination of both. These imbalances, which include disturbances in sodium, potassium, calcium, magnesium, and phosphate levels, can significantly affect cellular function, leading to a cascade of metabolic derangements that may exacerbate a patient's critical condition. The delicate equilibrium of electrolytes is crucial for maintaining cellular homeostasis, and any perturbations can have far-reaching effects on organ function, particularly in the heart, kidneys, and nervous system (1).

The management of electrolyte imbalances in critically ill patients is a complex and multifaceted challenge that requires careful monitoring and timely intervention. Early detection and correction of these imbalances are critical to preventing severe complications such as cardiac arrhythmias, neuromuscular disturbances, and renal dysfunction. The pathophysiology of electrolyte disorders in critically ill patients is often multifactorial, involving a combination of fluid shifts, impaired organ function, and the effects of medications such as diuretics and vasopressors. For example, hypokalemia, commonly seen in this patient population, can result from excessive gastrointestinal losses, renal excretion, or inadequate intake, and may be further exacerbated by the use of certain medications (2). In addition to the intrinsic challenges posed by the patient's underlying condition, the diagnostic approach to electrolyte imbalances in the critically ill is complicated by the dynamic nature of their clinical status. Frequent monitoring of electrolyte levels, along with a thorough assessment of clinical signs and symptoms, is essential for guiding treatment decisions. Furthermore, the management strategies must be tailored to the individual patient's needs, taking into account the etiology of the imbalance, the severity of the disorder, and the potential risks and benefits of the intervention. For instance, the administration of intravenous potassium must be

carefully balanced to avoid the dangers of rapid correction, which can lead to life-threatening hyperkalemia (3).

Despite advancements in critical care, the optimal management of electrolyte imbalances remains a subject of ongoing research and debate. Emerging evidence suggests that a more individualized approach to therapy, informed by a better understanding of the underlying mechanisms of these disorders, may improve outcomes for critically ill patients (4). This review aims to provide a comprehensive overview of the current strategies for diagnosing and managing electrolyte imbalances in critically ill patients, with a focus on the latest evidence-based practices.

Review

The management of electrolyte imbalances in critically ill patients requires a nuanced approach, considering the intricate interplay between the patient's underlying conditions and the therapeutic interventions employed. Electrolyte disturbances, such as hyperkalemia, hyponatremia, and hypocalcemia, are particularly challenging due to their potential to cause rapid deterioration in a patient's condition. The dynamic nature of electrolyte shifts in critical illness often necessitates continuous monitoring and prompt adjustment of treatment strategies. For example, the use of potassium-binding resins or intravenous insulin with glucose is a common practice to manage hyperkalemia, yet these interventions must be meticulously dosed to prevent adverse outcomes like hypoglycemia or rebound hyperkalemia (5). Furthermore, the choice of fluid therapy plays a significant role in managing electrolyte disorders. Isotonic saline, often used to correct hyponatremia, must be administered with caution, as rapid correction can lead to osmotic demyelination syndrome, a severe complication associated with overly aggressive sodium repletion (6). The complexity of electrolyte management in critical care underscores the importance of a personalized treatment approach, integrating both the patient's clinical presentation and the latest evidence-based guidelines to optimize outcomes. Continued

research is needed to refine these strategies, ensuring that interventions not only correct imbalances but also mitigate potential complications.

Pathophysiology and Clinical Implications of Electrolyte Imbalances in Critical Care

Electrolyte imbalances in critically ill patients are complex and multifactorial, with significant implications for patient outcomes. The pathophysiology of these disturbances often involves a combination of factors including fluid shifts, organ dysfunction, and the effects of medications commonly used in intensive care units (ICUs). These imbalances can affect various organ systems, leading to severe complications if not promptly addressed.

One of the most common electrolyte imbalances in critically ill patients is hyperkalemia, which can result from conditions such as acute kidney injury, tissue breakdown (as seen in rhabdomyolysis), and the use of potassium-sparing diuretics or ACE inhibitors. Hyperkalemia poses a significant risk due to its potential to cause cardiac arrhythmias, which can be life-threatening if not managed properly. The pathophysiological mechanism involves the accumulation of potassium in the extracellular space, which alters the normal electrochemical gradient across cell membranes, leading to impaired myocardial conduction and muscle weakness (7). This highlights the need for vigilant monitoring and timely intervention in patients at risk for hyperkalemia.

Hyponatremia, another common electrolyte disorder in critical care, is often associated with a high mortality rate, particularly in patients with neurological conditions such as traumatic brain injury or subarachnoid hemorrhage. The pathophysiology of hyponatremia typically involves an excess of free water relative to sodium, which can result from the inappropriate secretion of antidiuretic hormone (SIADH), excessive fluid administration, or renal dysfunction. Clinically, hyponatremia can lead to cerebral edema, which manifests as headache, confusion, seizures, and, in severe cases, coma. The rapid correction of

hyponatremia must be avoided, as it can precipitate osmotic demyelination syndrome, a devastating condition characterized by irreversible neurological damage (8).

Hypocalcemia is another critical electrolyte disturbance that often goes unrecognized in the ICU setting. It can arise from a variety of causes, including sepsis, acute pancreatitis, and massive transfusion of blood products containing citrate. Hypocalcemia affects both cardiovascular and neuromuscular function, leading to symptoms such as hypotension, prolonged QT interval on ECG, and tetany. The pathophysiological basis of hypocalcemia in critical illness is multifaceted, often involving a combination of decreased ionized calcium levels due to hypoalbuminemia, impaired parathyroid hormone secretion, and resistance to the effects of vitamin D. These disturbances can exacerbate the underlying critical condition, increasing the risk of morbidity and mortality (9). The pathophysiology of electrolyte imbalances in critically ill patients is complex and contributes significantly to the clinical challenges in this population. Understanding the underlying mechanisms and potential complications of these imbalances is essential for effective management and improving patient outcomes.

Hypomagnesemia is more frequently encountered in the ICU setting and is often associated with other electrolyte disturbances, such as hypokalemia and hypocalcemia. The pathophysiology of hypomagnesemia involves a variety of mechanisms, including inadequate intake, gastrointestinal losses (such as from diarrhea or nasogastric suction), renal wasting due to diuretics, and shifts between intracellular and extracellular compartments. Magnesium is a cofactor for many enzymes, including those involved in ATP metabolism, and its deficiency can impair cellular energy production, leading to muscle weakness, seizures, and cardiac arrhythmias, particularly torsades de pointes, a life-threatening form of ventricular tachycardia (10). In contrast, hypermagnesemia is less common but can occur in patients with renal failure, particularly if they receive magnesium-containing medications such as antacids or laxatives. The pathophysiology

of hypermagnesemia involves impaired renal excretion leading to an accumulation of magnesium in the bloodstream. Clinically, hypermagnesemia can cause hypotension, bradycardia, and respiratory depression due to its depressant effects on the neuromuscular and cardiovascular systems. Severe hypermagnesemia can lead to cardiac arrest if not promptly recognized and treated (11). Both hypomagnesemia and hypermagnesemia require prompt identification and management in critically ill patients. Hypomagnesemia is typically treated with intravenous magnesium sulfate, with careful monitoring to avoid overcorrection, particularly in patients with renal impairment. Hypermagnesemia, on the other hand, may require the use of intravenous calcium to counteract the effects of excess magnesium, along with measures to enhance renal excretion, such as diuretics or dialysis (12).

Diagnostic Approaches for Identifying Electrolyte Disorders in Critically Ill Patients

The accurate diagnosis of electrolyte disorders in critically ill patients is essential for guiding appropriate treatment and improving patient outcomes. Given the complexity and rapid changes in the clinical status of these patients, a multifaceted diagnostic approach is necessary. This approach involves a combination of laboratory testing, clinical assessment, and monitoring technologies to identify and track electrolyte imbalances effectively. Laboratory tests remain the cornerstone of diagnosing electrolyte disorders in the ICU. Serum electrolyte measurements, including sodium, potassium, calcium, magnesium, and phosphate, are routinely performed to assess and monitor the electrolyte status of critically ill patients. In addition to these basic tests, arterial blood gas (ABG) analysis is often used to evaluate acid-base balance, which can indirectly indicate electrolyte disturbances, such as the presence of anion gap acidosis in cases of hyperkalemia or lactic acidosis. Advanced laboratory techniques, such as ion-selective electrode (ISE) technology, have improved the accuracy and speed of electrolyte measurement, which is critical in the acute setting (13).

However, laboratory values must be interpreted within the context of the patient's clinical condition. Factors such as fluid status, ongoing losses (e.g., from diarrhea or vomiting), renal function, and medication use can all influence electrolyte levels. For example, hypokalemia in a patient receiving diuretics might indicate excessive renal potassium loss, necessitating a careful evaluation of diuretic dosage and the potential need for potassium supplementation. Similarly, hyponatremia may require differentiation between hypovolemic, euvolemic, and hypervolemic states to determine the underlying cause and appropriate treatment strategy (14). In addition to laboratory tests, continuous monitoring technologies, such as electrocardiography (ECG), play a vital role in detecting electrolyte disorders, particularly those that affect cardiac function. ECG changes, such as peaked T waves in hyperkalemia or prolonged QT intervals in hypocalcemia, can provide early indications of potentially life-threatening imbalances before they manifest in laboratory results. This underscores the importance of integrating clinical monitoring with laboratory testing to ensure timely and accurate diagnosis (15).

Moreover, the use of point-of-care testing (POCT) in the ICU has become increasingly common, allowing for rapid bedside assessment of electrolyte levels. This approach can significantly reduce the time between sample collection and result availability, enabling quicker decision-making in critical situations. However, it is essential to validate POCT results with conventional laboratory methods, particularly in cases of significant electrolyte disturbances or when clinical correlation is unclear. The diagnostic approach to electrolyte disorders in critically ill patients requires a comprehensive strategy that combines laboratory testing, clinical assessment, and continuous monitoring. This integrated approach is vital for the timely identification and management of electrolyte imbalances, ultimately improving patient outcomes.

Management Strategies and Therapeutic Interventions for Electrolyte Imbalances

Effective management of electrolyte imbalances in critically ill patients requires a tailored approach,

considering the underlying causes, the severity of the imbalance, and the potential for rapid changes in the patient's condition. The therapeutic interventions must be both prompt and precise to prevent complications such as arrhythmias, neuromuscular dysfunction, and organ failure.

For hyperkalemia, one of the most common and potentially life-threatening electrolyte imbalances, the management strategy typically includes a combination of methods to stabilize the cardiac membrane, shift potassium intracellularly, and enhance potassium elimination. Immediate administration of intravenous calcium gluconate is often used to counteract the effects of elevated potassium on cardiac myocytes, thereby reducing the risk of arrhythmias. Concurrently, agents such as insulin with glucose or beta-agonists (e.g., albuterol) are administered to promote the intracellular shift of potassium, temporarily lowering serum levels. Additionally, potassium-binding resins like sodium polystyrene sulfonate, along with loop diuretics or dialysis, may be utilized to remove excess potassium from the body, depending on the severity of hyperkalemia and renal function (16).

Hyponatremia management depends on the etiology and severity of the sodium deficit. In cases of acute symptomatic hyponatremia, where patients may present with seizures or altered mental status, hypertonic saline (3% NaCl) is often administered to rapidly increase serum sodium levels and alleviate cerebral edema. However, the rate of sodium correction must be carefully controlled to avoid the risk of osmotic demyelination syndrome, a serious and often irreversible neurological condition caused by overly rapid sodium correction. For chronic or mild hyponatremia, fluid restriction and the use of vasopressin receptor antagonists (vaptans) are commonly employed to manage euvolemic or hypervolemic hyponatremia (17).

Hypocalcemia management is another critical aspect of care in critically ill patients, particularly in those with sepsis, pancreatitis, or massive transfusions. Intravenous calcium gluconate or calcium chloride is the treatment of choice for acute

symptomatic hypocalcemia, with the latter being preferred in cases requiring rapid correction due to its higher calcium content. However, the administration must be done cautiously to avoid complications such as tissue necrosis if extravasation occurs. Additionally, underlying causes such as hypomagnesemia or vitamin D deficiency should be corrected to ensure sustained normalization of calcium levels (9).

Magnesium imbalances, specifically hypomagnesemia, are also a significant concern in critically ill patients. Hypomagnesemia often coexists with other electrolyte disturbances, such as hypokalemia and hypocalcemia, and can exacerbate their effects. Correction of hypomagnesemia typically involves the administration of intravenous magnesium sulfate. The dose and rate of administration should be adjusted based on the severity of the deficiency and the presence of renal impairment. For example, in patients with mild hypomagnesemia, oral supplementation may be sufficient, while more severe cases may require intravenous therapy. Continuous monitoring is essential to avoid overcorrection, which could lead to hypermagnesemia, particularly in patients with renal insufficiency. Hypermagnesemia, though less common, can be managed with intravenous calcium to stabilize cardiac and neuromuscular functions, along with measures to enhance magnesium excretion, such as diuretics or dialysis (18). In summary, the management of electrolyte imbalances in critically ill patients involves a combination of targeted therapeutic interventions tailored to the specific electrolyte disturbance. Rapid identification and correction of these imbalances are crucial to preventing further complications and ensuring the stabilization of critically ill patients.

Conclusion

Electrolyte imbalances in critically ill patients are complex and require prompt, individualized management to prevent severe complications. An integrated approach combining timely diagnosis, continuous monitoring, and targeted therapeutic interventions is essential for optimizing patient

outcomes. Addressing underlying causes and closely monitoring treatment responses are critical to ensuring safe and effective correction of these imbalances. Ongoing research and clinical vigilance remain vital in refining these management strategies to improve care in critical settings.

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Author Contributions

The author has reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Consent for publications

Not applicable

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Conflict of interest

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References

1. Rose BD. Clinical physiology of acid-base and electrolyte disorders. (No Title). 2001.
2. Adrogué HJ, Madias NE. Sodium and potassium in the pathogenesis of hypertension. *New England journal of medicine*. 2007;356(19):1966-78.
3. Sterns RH, Grieff M, Bernstein PL. Treatment of hyperkalemia: something old, something new. *Kidney international*. 2016;89(3):546-54.
4. Halperin ML, Goldstein MB, Stark J. Fluid, electrolyte and acid-base physiology: a problem-based approach. *Critical Care Nursing Quarterly*. 1994;17(3):88-9.
5. Langston C. Managing fluid and electrolyte disorders in kidney disease. *Veterinary Clinics: Small Animal Practice*. 2017;47(2):471-90.

6. Adrogué HJ, Madias NE. Hyponatremia. *New England Journal of Medicine*. 2000;342(21):1581-9.
7. Palmer BF, Clegg DJ. Electrolyte and acid-base disturbances in patients with diabetes mellitus. *New England Journal of Medicine*. 2015;373(6):548-59.
8. Zenenberg RD, Carluccio AL, Merlin MA. Hyponatremia: evaluation and management. *Hospital Practice*. 2010;38(1):89-96.
9. Kelly A, Levine MA. Hypocalcemia in the Critically Ill patient. *Journal of Intensive Care Medicine*. 2011;28(3):166-77.
10. Reinhart RA. Clinical correlates of the molecular and cellular actions of magnesium on the cardiovascular system. *American heart journal*. 1991;121(5):1513-21.
11. Tong GM, Rude RK. Magnesium deficiency in critical illness. *Journal of intensive care medicine*. 2005;20(1):3-17.
12. Goldman J, Choure GS. Metabolic disturbances of acid-base and electrolytes. *Critical Care Study Guide: Text and Review*. 2010:691-713.
13. Palmer BF. Managing hyperkalemia caused by inhibitors of the renin-angiotensin-aldosterone system. *New England Journal of Medicine*. 2004;351(6):585-92.
14. Ayus JC, Arieff AI. Pathogenesis and prevention of hyponatremic encephalopathy. *Endocrinology and metabolism clinics of North America*. 1993;22(2):425-46.
15. Surawicz B, Knilans T. *Chou's electrocardiography in clinical practice: adult and pediatric*: Elsevier Health Sciences; 2008.
16. Kim MJ, Valerio C, Knobloch GK. Potassium disorders: hypokalemia and hyperkalemia. *American Family Physician*. 2023;107(1):59-70A.
17. Sterns RH, Hix JK, Silver SM. Management of hyponatremia in the ICU. *Chest*. 2013;144(2):672-9.
18. Rylander R, Arnaud MJ. Mineral water intake reduces blood pressure among subjects with low urinary magnesium and calcium levels. *BMC public health*. 2004;4:1-5.