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Review Article

Medication-induced electrolyte imbalances: a call for increased awareness and vigilance

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ABSTRACT

Electrolyte imbalances are a significant concern in clinical practice, often caused by medications such as diuretics, cardiovascular drugs, chemotherapy agents, and antibiotics. These imbalances can lead to serious complications, including cardiac arrhythmias and increased mortality. Key electrolytes like sodium, potassium, calcium, and magnesium play crucial roles in maintaining fluid balance, nerve function, and muscle contraction. Medications can disrupt electrolyte balance, causing hyponatremia, hyperkalemia, hypomagnesemia, and other imbalances. Risk factors include pre-existing kidney disease, heart failure, and concomitant use of multiple medications. Treatment involves discontinuing the causative medication, correcting electrolyte imbalances, and managing underlying conditions. This review analyzes drug induced electrolyte disorders, their consequences, and treatment options. Understanding the mechanisms of drug-induced electrolyte imbalances can help clinicians manage complex cases and provide effective care.

Keywords: Electrolyte imbalance, Diuretics, Cardiovascular drugs, Chemotherapy agents, Corticosteroids, NSAIDs and electrolyte management

INTRODUCTION

Electrolytes are the essential mineral in human body which is required for fundamental life processes. They regulate various critical functions, including heart rhythm, nerve function, fluid balance, oxygen transport, and acid-base equilibrium. Key electrolytes that play a crucial role in these processes include calcium, chloride, magnesium, phosphate, potassium, and sodium. When the high retention or excretions of mineral occur that leads to electrolyte.[1-3] As there are the certain medications that can significantly impact electrolyte balance in the body, affecting the intake, elimination, regulation, and distribution. These disruptions can be severe and life-threatening, presenting challenges for healthcare providers who are not aware of them. By understanding how specific drugs alter electrolyte levels and the underlying mechanisms, clinicians can better manage complex cases and provide more effective care.[4]

Electrolyte imbalances, such as hyponatremia, hyperkalemia, and hypomagnesemia, are prevalent in up to 50% of intensive care unit (ICU) patients, often triggered by medications.

Common ICU medications, including diuretics, corticosteroids, and antimicrobials, can disrupt electrolyte balance, leading to serious complications like cardiac arrhythmias, prolonged ventilation, and increased mortality.[5] The review analyzes all the electrolyte disorder with its consequences caused by which drug with its treatment.

WHAT DOES ELECTROLYTE DO TO THE BODY?

Electrolytes like sodium, potassium, calcium, bicarbonate, magnesium, chloride, and phosphorus play crucial roles in maintaining fluid balance, nerve function, muscle contraction, and acid-base regulation.

Sodium

It is crucial for maintaining extracellular fluid volume, regulating cell membrane potential, and supporting nerve function.

Hyponatremia (low sodium, <135 mmol/L): headaches, confusion, nausea. Hypernatremia (high sodium, >145 mmol/L): tachypnea, restlessness, sleeping difficulty.

Potassium

An intracellular ion that regulates heart rhythm and muscle function.

Hypokalemia (low potassium, <3.6 mmol/L): weakness, fatigue, muscle twitching. Hyperkalemia (high potassium, >5.5 mmol/L): arrhythmias, muscle weakness, cramps.

Calcium

Plays a vital role in bone health muscle contraction nerve transmission. Hypocalcemia (low calcium, <8.8 mg/dl): muscle cramps, weakness. Hypercalcemia (high calcium, >10.7 mg/dl): bone pain, kidney stones, arrhythmias.

Magnesium

It is crucial for energy production (ATP metabolism), muscle function, nerve transmission.[2]

Hypomagnesemia (serum magnesium concentration of <1.5 mg/dl) is frequently observed in critically ill patients and has been associated with increased mortality. Severe hypomagnesemia (serum magnesium concentration of <1.0 mg/dl) can result in ECG changes, seizures, coma, and even death.

Hypermagnesemia is defined as a serum magnesium level >2.4 mg/dl. Mild cases (2.5-4 mg/dl) are often asymptomatic. Moderate hypermagnesemia (4-12.5 mg/dl) can cause symptoms like nausea, vomiting, loss of deep tendon reflexes, hypotension, bradycardia, and ECG changes. Severe cases (>12.5 mg/dl) can lead to respiratory paralysis, refractory hypotension, AV block, cardiac arrest, and death.[6]

Chloride

Plays a key role in regulating fluid balance and acid-base balance. Causes hyperchloremia (high chloride) and hypochloremia (low chloride). Often associated with gastrointestinal losses (vomiting, diarrhea), congestive heart failure.

Bicarbonate

Crucial for maintaining acid-base balance, primarily regulated by the kidneys.

Acidosis (low bicarbonate): often due to kidney disease or diarrhea. Alkalosis (high bicarbonate): often due to kidney disorders or excessive bicarbonate intake.

Phosphorus

It is crucial for energy production (ATP), Bone health and also involve in metabolic pathways.

Hypophosphatemia (low phosphate): often due to poor dietary intake, GI disorders, or excessive renal excretion. Hyperphosphatemia (high phosphate): often due to kidney disease or excessive intake.[7]

WHAT ARE THE DRUGS THAT CAUSES ELECTROLYTE IMBALANCE?

Diuretics like loop diuretics (e.g., furosemide, bumetanide), thiazide diuretics (e.g., hydrochlorothiazide) and potassium-sparing diuretics (e.g., spironolactone, amiloride).[8] Cardiovascular drugs i.e. ACE inhibitors (e.g., lisinopril), arbs (e.g., losartan) and beta-blockers (e.g., metoprolol).[9] Chemotherapy agents like cisplatin, ifosfamide.[10] Psychotropic medications include SSRIS (e.g., sertraline, fluoxetine and Carbamazepine).[11] Gastrointestinal drugs as proton pump inhibitors (PPIS) (e.g., omeprazole, pantoprazole and Chronic laxative use.[12] Antibiotics and antifungals include amphotericin B and trimethoprim-sulfamethoxazole.[13] Other drugs may include corticosteroids, NSAIDS (e.g., ibuprofen, naproxen), and Bisphosphonates and denosumab.[8]

HOW DOES THE DRUG CAUSES ELECTROLYTE IMBALANCE?

Diuretics

Here, imbalances like hyponatremia and hypokalaemia can emerge within days to weeks of initiating therapy or increasing the dose, but can also appear years later. It disrupts electrolyte balance by blocking reabsorption in the kidneys, increasing urinary excretion. The type and onset of imbalance depend on the diuretic class, dose, and patient factors like age, diet, and concomitant medications.[14]

Loop diuretics

Loop diuretics like furosemide inhibit the sodium-potassium-chloride cotransporter ($NKCC_2$) in the kidney's thick ascending limb of the loop of Henle, a key area for sodium reabsorption.[15]

Hypokalaemia

This is a major concern with loop diuretic use. By blocking sodium reabsorption, these drugs increase the delivery of sodium to the distal tubules. The ensuing increase in sodium-for-potassium exchange in the distal nephron promotes excessive potassium excretion. Studies have

documented that the electrolyte imbalances caused by loop diuretics, particularly hypokalaemia, significantly increase the risk of digoxin toxicity, especially in heart failure patients. One nested case-control study found that loop diuretics carried the greatest risk of digoxin intoxication compared to other diuretic types, making co-prescribing them a practice to avoid if possible.

Hypocalcaemia and hypomagnesemia

Loop diuretics reduce the positive electrical potential in the renal tubule that drives the passive reabsorption of calcium and magnesium. This leads to their increased excretion in the urine.

Hyperchloremic metabolic alkalosis

The loss of fluid and chloride, combined with increased hydrogen ion secretion, can cause a metabolic alkalosis.[14,16,17]

Thiazide diuretics (e.g., Hydrochlorothiazide)

This work by blocking the sodium-chloride cotransporter (NCC) in the kidney's distal convoluted tubule, reducing sodium and chloride reabsorption and increasing their excretion in urine.[18,19] It can have a significant impact on electrolyte balance, leading to several notable effects:

Hyponatremia

Thiazides can cause dilutional hyponatremia, particularly in susceptible individuals such as the elderly and those with lower body mass, by impairing the kidney's ability to excrete free water while preserving the urine-concentrating mechanism. This is especially pronounced with excessive fluid intake. Research has confirmed that thiazide diuretics are associated with a higher risk of hyponatremia compared to other diuretics. A 2024 study in elderly adults reported a markedly increased risk of hyponatremia associated with thiazide diuretic use, with the greatest risk occurring within the first 60 days of therapy.

A review of thiazide-induced hyponatremia further noted that most cases develop within the first two weeks after initiation of treatment. In a study of 114 elderly patients, the incidence of hyponatremia was approximately 11%. Hospital-based research in geriatric inpatients also shows that hyponatremia is a common adverse effect, affecting 11% to 33% of patients taking a thiazide diuretic at the time of admission.[18,20,21]

Hypokalaemia

Similar to loop diuretics, thiazides promote increased potassium secretion further down the nephron, leading to potassium loss and depletion. The extent of potassium depletion is dose-dependent, making it a significant concern.[20]

Hypercalcemia

Thiazides have a unique effect on calcium levels, paradoxically enhancing calcium reabsorption and leading to increased calcium levels in the blood.[22-24]

Potassium-sparing diuretics

This are the mildest type, working on the kidney's collecting tubule to increase sodium and water excretion while preserving potassium. They're often used with other diuretics to prevent low potassium levels. There are two main types: Aldosterone antagonists (e.g., spironolactone), alock mineralocorticoid receptors, reducing sodium reabsorption and potassium excretion and ENAC blockers (e.g., amiloride, triamterene): Directly block sodium reabsorption channels.[25,26]

Hyperkalaemia

Both mechanisms inhibit sodium reabsorption and decrease the secretion of potassium into the urine. The risk of dangerously high potassium is increased in patients with kidney dysfunction or those also taking ACE inhibitors or arbs.

Hyperchloremic metabolic acidosis

By inhibiting the excretion of both potassium and hydrogen, potassium-sparing diuretics can cause a type of metabolic acidosis.[27]

Cardiovascular agents

RAAS inhibitors

Including ACE inhibitors and arbs, block angiotensin II and aldosterone, affecting blood pressure and electrolyte balance. ACE inhibitors (e.g., ramipril) and arbs (e.g., losartan) inhibit aldosterone, reducing potassium excretion and sodium reabsorption in collecting ducts. Monitor potassium levels, especially with kidney issues or combined therapy. Risk of hyperkalaemia increases with potassium-sparing diuretics or renal impairment.[28]

Hyperkalaemia

Risk starts soon after treatment begins, increasing over time, especially with kidney issues. Aldosterone suppression reduces potassium excretion, elevating serum levels. Risk increases with potassium-sparing diuretics or renal impairment. Hyponatremia is less common than with diuretics, but possible.[29]

Beta-blockers

It works by blocking stress hormones like adrenaline, treating various heart conditions. It may cause Hyperkalemia, blocking beta-2 receptors can slightly increase serum potassium by reducing cellular uptake.

This effect is usually minor, but can be significant if combined with other risk factors or in overdose situations. Acute use or overdose can cause rapid potassium shifts. Chronic use typically has minimal impact unless combined with other agents.[28,29]

Digoxin

Digoxin is a cardiac glycoside used to treat heart failure and certain heart rhythm problems.[28] This causes mild electrolyte wasting and limits magnesium reabsorption, increasing excretion. Low potassium and magnesium levels amplify digoxin's effects, increasing toxicity risk. Hypokalaemia and hypomagnesemia, this may lead increase digoxin toxicity risk, potentially leading to arrhythmias. Low potassium and magnesium levels enhance digoxin's effect. Toxicity risk exists throughout therapy.[28,30] Monitoring electrolyte levels is key when patients are on cardiovascular meds. Imbalances can lead to serious cardiovascular disease like arrhythmias, and other complications.[28,31] The risk of developing these electrolyte abnormalities is heightened in older patients often have reduced renal function and are more susceptible to medication-induced electrolyte shifts. Impaired kidney function directly compromises the body's ability to regulate electrolyte balance, exacerbating the effects of cardiovascular drugs. Concurrent use of multiple cardiovascular medications, especially those that affect electrolytes (e.g., diuretics, RAAS inhibitors), increases the potential for adverse interactions. Congestive heart failure (CHF) condition involves ongoing neurohumoral activation (including the RAAS and sympathetic nervous system), which can predispose patients to both electrolyte disturbances and medication-related adverse events.[28] Mild or chronic electrolyte imbalances can develop subtly, causing non-specific symptoms such as muscle weakness, fatigue, or generalized malaise, which may be mistaken for other conditions. Rapid or severe electrolyte shifts, particularly in potassium levels, can lead to life-threatening cardiac arrhythmias. This is a recognized risk, especially in the context of perioperative management, such as following cardiac surgery, where electrolyte depletion is common.[28,32,33]

Chemotherapy agent

Chemo drugs like cisplatin and Ifosfamide can cause electrolyte imbalances by nephrotoxicity, Gastrointestinal losses and Tumor lysis syndrome. Onset and type of imbalance depend on the specific drug and cumulative dose.[34-36] Cisplatin, a platinum-based chemo drug, damages renal tubules, causing significant electrolyte deficiencies.[35]

Cisplatin

Hypomagnesemia

Most common, up to 90% of patients. Cisplatin damages renal tubules, impairing magnesium reabsorption. Cumulative and dose-dependent, can persist years after treatment.

Hypokalemia

Often coexists with hypomagnesemia. Magnesium depletion increases potassium excretion. Refractory to potassium replacement unless hypomagnesemia is corrected. Cisplatin can also cause.

Hypophosphatemia

It occurs due to proximal tubule damage. Hypocalcemia secondary to the hypomagnesemia-induced impairment of parathyroid hormone (PTH) release. Hyponatremia through the syndrome of inappropriate antidiuretic hormone secretion (SIADH) or renal salt-wasting syndrome (RSWS).[34-37]

Ifosfamide

An alkylating agent, is a prodrug metabolized in the liver into therapeutic and toxic metabolites, including the highly nephrotoxic chloroacetaldehyde.[38] This drug can cause significant kidney damage, leading to Fanconi syndrome, a defect in the proximal renal tubules. Fanconi Syndrome impairs reabsorption of filtered substances, leading to urinary wasting of key electrolytes. Fanconi syndrome causes Hypokalemia, Hypophosphatemia, Metabolic acidosis, Hypomagnesemia and Hyponatremia. This may cause due to higher cumulative doses, prior cisplatin treatment, damage can occur during therapy or persist after completion.[39-41]

Psychotic drugs

SSRIS (like sertraline and fluoxetine)

Can cause hyponatremia (low sodium) by triggering SIADH. It Increased serotonin levels stimulate ADH (antidiuretic hormone) release, ADH increases water reabsorption in kidneys, leading to water retention and dilutional hyponatremia.

Typically, within first few weeks of starting or increasing dose hyponatremia occurs. This can be treated usually within 1-2 weeks after stopping medication. Elderly, females, low body mass, or taking other sodium-affecting meds (like diuretics) have a high risk of hyponatremia.[42-44]

Carbamazepine

It can cause hyponatremia (low sodium) by triggering SIADH.[45] It increases ADH secretion or renal sensitivity to ADH and enhances water reabsorption in kidneys, leading to dilutional hyponatremia.

Hyponatremia occurs within first few days to weeks of starting treatment. Higher doses, older patients and Concomitant diuretics are the risk factors.[46,47,48]

LITERATURE REVIEW

This review article examines the effects of medications on electrolyte balance through a comprehensive literature review. Electronic databases (PubMed, MEDLINE, Google Scholar) were searched using relevant keywords. Reference lists of selected articles were manually searched to identify additional studies. Inclusion criteria included human studies (clinical trials, case reports, observational studies) published in English language journals, focusing on medications affecting electrolyte balance (e.g., diuretics, cardiovascular drugs, chemotherapy agents, antibiotics). Relevant data were extracted, including medication name and class, electrolyte imbalance type and severity, mechanisms of disturbance, clinical manifestations, and treatment strategies. The review aims to summarize current knowledge on medication-induced electrolyte imbalances, highlighting risk factors, clinical implications, and management approaches.

OBSERVATIONS

The review identified numerous medications that disrupt electrolyte balance, leading to imbalances such as hyponatremia, hyperkalemia, hypokalemia, hypomagnesemia, and hypocalcemia. Diuretics, SSRIs, and carbamazepine can cause hyponatremia, while potassium-sparing diuretics, ACE inhibitors, and ARBs can cause hyperkalemia. Loop diuretics and thiazide diuretics can lead to hypokalemia, and proton pump inhibitors and amphotericin B can cause hypomagnesemia. Medications affect electrolyte balance by altering renal excretion, hormone regulation, gut absorption, or inducing nephrotoxicity. Clinical manifestations range from mild to severe, including muscle weakness, cardiac arrhythmias, seizures, and respiratory depression. The severity of imbalances varies depending on medication dose, duration, and patient factors. Recognizing and managing medication-induced electrolyte imbalances is crucial to prevent serious complications and improve patient outcomes.

DISCUSSION

Medication-induced electrolyte imbalances are a significant concern, as they can lead to serious complications and increased mortality. The review highlights the importance of recognizing and managing these imbalances. Clinicians should be aware of the potential effects of medications on electrolyte balance, particularly in high-risk patients. Monitoring electrolyte levels and adjusting medication regimens can help prevent imbalances. Further research is needed to understand the mechanisms of medication-induced electrolyte disturbances and to develop strategies for prevention and treatment. By acknowledging the risks and taking proactive steps, healthcare providers can improve patient outcomes and reduce the burden of electrolyte-related complications.

CONCLUSION

Medication-induced electrolyte imbalances are a significant concern in clinical practice, leading to serious complications and increased mortality. This review highlights the importance of recognizing and managing these imbalances. Clinicians should be aware of the potential effects of medications on electrolyte balance, particularly in high-risk patients. By monitoring electrolyte levels and adjusting medication regimens, healthcare providers can prevent and treat imbalances, improving patient outcomes. Increased awareness and proactive management can reduce the burden of electrolyte-related complications, ultimately enhancing patient care and safety. Vigilance and informed decision-making are essential in mitigating these risks.

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