

Electrolyte Disturbances and Clinical Outcomes in Chronic Kidney Disease: A Comprehensive Narrative Review

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ABSTRACT

Chronic kidney disease (CKD) can be described as an advancing condition where there is a reduction in renal function leading to several biochemical alterations in the blood, including electrolytes imbalance, and this increases the chances of acceleration in continuous, gradual decline of illness. Impaired renal tubular function in CKD disrupts the homeostasis of sodium, potassium, calcium, phosphorus, magnesium, and uric acid; this disruption leads to conditions like hyperkalemia, hypocalcemia, hyperphosphatemia, hyperuricemia, and hypermagnesemia^[1,3]. The prevalence and severity of these electrolyte disturbances rise as the glomerular filtration rate (GFR) drops, and are strongly associated with comorbidities like diabetes mellitus and hypertension^[2]. Electrolyte imbalance in CKD are tied to cardiovascular issues, mineral and bone disorders, nerve and muscle dysfunction, lower quality of life, and advancement towards end-stage renal disease (ESRD)^[1,3]. Recent long term studies have shown that ongoing patterns in electrolyte levels - especially persistent hyperkalemia and hyperphosphatemia, may predict adverse renal outcomes and hospitalization risk among pre-dialysis CKD patients^[1]. Early identification, continuous biochemical monitoring, individualized dietary modification, and appropriate pharmacological management are therefore essential to slowing the progression of the disease and enhancing patient care in CKD patients^[2,3].

Keywords: Chronic kidney disease, electrolyte imbalance, hyperkalemia, hyponatremia, hypocalcemia, hyperphosphatemia, dialysis, renal failure, quality of life

1. INTRODUCTION

Chronic kidney disease (CKD) is a global epidemic that significantly impacts the health status of populations worldwide. It is estimated that approximately 10% of the world's population suffers from CKD, imposing a substantial economic burden on healthcare systems^[3]. CKD progression is commonly associated with diabetes mellitus, hypertension, cardiovascular diseases, and metabolic imbalance^[1,3]. The progressive loss of kidney function in CKD leads to impaired fluid, electrolyte, and acid-base homeostasis, resulting in severe electrolyte imbalances^[2].

Electrolytes like sodium, potassium, calcium, and phosphorus are critical for heart stability, bone health, nerve transmission, and cellular functions. Impaired renal excretion and altered hormonal regulation in CKD frequently lead to hyponatremia, hyperkalemia, hypocalcemia, and hyperphosphatemia. These disruptions can result in severe complications, including abnormal heart rhythms, cognitive deterioration, calcified blood vessels, mineral bone disorders, and a high risk of death^[4].

Recent studies have demonstrated that the temporal patterns of electrolyte levels can serve as powerful predictive biomarkers for CKD progression. For instance, an analysis of longitudinal electrolyte trends in patients with predialysis chronic kidney disease identified specific patterns of sodium, potassium, calcium, and phosphorus levels associated with an increased risk of dialysis initiation. Patients who presented with consistently elevated potassium and phosphorus levels alongside decreased sodium and calcium levels were found to have a significantly higher likelihood of progressing to ESRD ^[1,5].

Beyond physical health, CKD substantially impacts patients' health-related quality of life (HRQoL). Patients with chronic kidney disease frequently report physical limitations, fatigue, emotional distress, and social dysfunction. Reduced glomerular filtration rate (GFR) is strongly correlated with diminished HRQoL scores ^[3,6].

CKD is classified into five stages based on eGFR, with stage 1 representing normal or increased GFR (≥ 90 mL/min/1.73 m²) in the presence of markers of kidney damage, and stage 5 (< 15 mL/min/1.73 m²) representing kidney failure, at which point renal replacement therapy - dialysis or transplantation—is typically required. Stage 3 is further subdivided into 3a and 3b to reflect the clinically meaningful difference in risk between moderately and moderately-to-severely reduced GFR^[6,36]. This staging framework, summarized in Table 1 below, underpins clinical decision-making regarding the frequency of biochemical monitoring and the intensity of electrolyte and mineral-bone disorder management, since the risk of clinically significant electrolyte disturbance rises sharply from stage 3b onward.

Table 1: KDIGO Staging of Chronic Kidney Disease by Estimated Glomerular Filtration Rate

CKD Stage	Description	eGFR (mL/min/1.73 m ²)	Typical Electrolyte Monitoring Frequency
Stage 1	Normal or high GFR with evidence of kidney damage	≥ 90	Annual
Stage 2	Mildly decreased GFR with evidence of kidney damage	60–89	Annual
Stage 3a	Mildly to moderately decreased GFR	45–59	Every 6–12 months
Stage 3b	Moderately to severely decreased GFR	30–44	Every 3–6 months
Stage 4	Severely decreased GFR	15–29	Every 1–3 months
Stage 5	Kidney failure	< 15 (or dialysis)	Monthly or per dialysis schedule

This review aims to provide an in-depth exploration of electrolyte imbalances in CKD, including their clinical manifestations, mechanisms, prognostic implications, and therapeutic management.

2. MATERIALS AND METHODS

This narrative review was developed through a structured search of the peer-reviewed literature published in electronic databases, including PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar, supplemented by relevant clinical practice guidelines from KDIGO and other professional nephrology bodies. Search terms included combinations

of “chronic kidney disease,” “electrolyte imbalance,” “hyponatremia,” “hyperkalemia,” “hypocalcemia,” “hyperphosphatemia,” “hypermagnesemia,” “metabolic acidosis,” “uric acid,” “CKD-mineral and bone disorder,” “CKD progression,” and “quality of life.” Original research articles, cohort studies, systematic reviews, meta-analyses, and clinical guidelines published predominantly within the last decade were prioritized, while several foundational studies of continued clinical relevance were also included to provide historical and mechanistic context. Articles were screened for relevance to the pathophysiology, epidemiology, clinical outcomes, and management of electrolyte disturbances in CKD. Findings were synthesized narratively and organized thematically by electrolyte and by clinical domain (pathophysiology, prognosis, quality of life, and management) rather than through a formal systematic or meta-analytic methodology, consistent with the scope and purpose of a narrative review.

3. ELECTROLYTE DISTURBANCES IN CKD

3.1 Sodium Imbalance in CKD

3.1.1 Pathophysiology

Sodium balance is principally regulated by the kidneys through control of renal tubular reabsorption and free water excretion. Reduced GFR in CKD impairs the kidney’s capacity to excrete free water, predisposing to dilutional hyponatremia, particularly in the setting of excess fluid intake or impaired urinary dilution. In addition, CKD patients frequently exhibit an inappropriate increase in circulating ADH and secondary fluid retention, further contributing to sodium imbalance ^[7]. Conversely, in advanced CKD with reduced urinary concentrating capacity, or in the setting of inadequate free water intake, osmotic diuresis, or hypernatremia may occur, although this is less common than hyponatremia in the CKD population.

3.1.2 Clinical Manifestations

Hyponatremia is defined as a serum sodium concentration below 135 mmol/L. Neurological symptoms are the most prominent clinical manifestations and range from mild confusion and impaired concentration to severe cases involving seizures, cerebral edema, and coma. Chronic, even mild, hyponatremia has been associated with subtle gait instability, attention deficits, and an increased risk of falls and fractures, particularly among older adults ^[4]. Hyponatremia has also been linked to higher hospitalization rates and increased mortality in patients with CKD and ESRD.

3.1.3 Prognostic Significance

Longitudinal studies have shown that abnormal serum sodium levels are independently associated with poor renal outcomes, and that progressive declines in serum sodium over time are predictive of accelerated decline in kidney function and earlier development of ESRD ^[2,8]. A cross-sectional analysis further demonstrated that sodium balance, as reflected by dietary sodium intake and fluid status, correlates with quality-of-life indices in people living with CKD, reinforcing the clinical relevance of sodium homeostasis beyond its biochemical implications ^[21].

3.2 Potassium Imbalance in CKD

3.2.1 Pathophysiology

Potassium homeostasis is predominantly regulated by the kidneys, with the distal nephron responsible for fine adjustment of urinary potassium excretion under the influence of aldosterone. As renal function declines, the capacity for potassium excretion diminishes, predisposing patients to hyperkalemia. This risk is substantially amplified by the widespread use of RAAS inhibitors—including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, mineralocorticoid receptor antagonists, and, increasingly, angiotensin receptor-neprilysin inhibitors - which are cornerstone therapies for CKD and cardiovascular risk reduction but simultaneously blunt the kidney’s capacity to excrete potassium ^[1,9]. Concurrent metabolic acidosis, commonly present in CKD, further impairs cellular potassium uptake and exacerbates hyperkalemia.

3.2.2 Clinical Manifestations

Hyperkalemia is defined as a serum potassium concentration exceeding 5.0 mmol/L. Its clinical manifestations range from subtle findings, such as muscle weakness, fatigue, and paresthesia, to life-threatening cardiac arrhythmias and sudden cardiac death. Characteristic electrocardiographic (ECG) changes include peaked T-waves, PR-interval prolongation, QRS widening, and, in severe cases, a sine-wave pattern that may herald ventricular fibrillation. Notably, the correlation between serum potassium concentration and ECG changes is imperfect, and severe arrhythmias can occur even in the absence of classic ECG findings, underscoring the need for vigilance ^[10].

3.2.3 Prognostic Significance

Several large cohort studies have demonstrated that elevated serum potassium levels are independently predictive of CKD progression and dialysis initiation ^[1,12,13]. In a Danish population-based cohort, elevated potassium levels were associated with increased risk of major adverse cardiovascular events, hospitalization, and mortality ^[14], findings that were subsequently corroborated in the Fukushima CKD cohort study ^[15]. Hyperkalemia has been reported to occur in nearly 45% of patients with CKD, underscoring its high prevalence and clinical significance ^[2], and has also been shown to predict adverse outcomes in patients with concomitant heart failure ^[23]. Population-based data further indicate that a substantial proportion of patients receiving RAAS inhibitor therapy experience at least one episode of hyperkalemia sufficient to prompt dose reduction or discontinuation, a phenomenon that may paradoxically worsen long-term cardio-renal outcomes by depriving patients of guideline-directed therapy ^[26].

3.3 Calcium and Phosphorus Imbalance in CKD

3.3.1 Pathophysiology

Disturbances of calcium metabolism in CKD arise from a complex interplay of reduced renal synthesis of active vitamin D, phosphate retention, and secondary hyperparathyroidism. As GFR declines, renal 1-alpha-hydroxylase activity diminishes, reducing conversion of 25-hydroxyvitamin D to its active form and impairing intestinal calcium absorption. Concurrently, phosphate retention stimulates parathyroid hormone (PTH) and FGF-23 secretion, both of which act, in part, to promote phosphaturia at the expense of further suppressing calcitriol synthesis. The net effect is a tendency toward hypocalcemia, compensatory secondary hyperparathyroidism, and, over time, tertiary hyperparathyroidism with autonomous PTH secretion ^[18].

3.3.2 Clinical Manifestations

Hypocalcemia presents with neuromuscular irritability, including paraesthesia, muscle cramps, tetany, and, in severe cases, seizures and prolongation of the QT interval predisposing to arrhythmia. Chronic disturbances in calcium and PTH homeostasis contribute to renal osteodystrophy, a spectrum of bone disorders encompassing osteitis fibrosa cystica (high-turnover bone disease), adynamic bone disease (low-turnover), and osteomalacia, all of which increase fracture risk.

3.3.3 Prognostic Significance

Low serum calcium has been independently associated with poorer renal outcomes in patients with CKD stages 3–4, including a faster decline in eGFR ^[16]. Similarly, disturbed calcium-phosphate homeostasis has been linked with increased risk of acute coronary syndrome in CKD patients ^[17], and joint longitudinal trajectories of low calcium combined with high phosphate have been shown to associate with accelerated CKD progression, cardiovascular events, and mortality ^[20]. Vitamin D deficiency itself is highly prevalent across all CKD stages and correlates with both biochemical and clinical markers of disease severity ^[19].

3.3.4 Phosphorus Disorders and Ckd-Mineral and Bone Disorder

Phosphate homeostasis is maintained through a delicate balance of intestinal absorption, bone turnover, and renal excretion, the latter regulated principally by PTH and FGF-23. As nephron mass declines, phosphate excretion per nephron initially increases to maintain normophosphatemia; however, this adaptive response is itself mediated by rising FGF-23 and PTH concentrations, which, over time, contribute to vascular calcification, left ventricular hypertrophy, and other manifestations of the broader CKD-mineral and bone disorder (CKD-MBD) syndrome. Overt hyperphosphatemia typically becomes evident once GFR falls below approximately 30 mL/min/1.73 m², when compensatory mechanisms are exhausted^[16,17,18,19].

Hyperphosphatemia is strongly associated with vascular and soft-tissue calcification, accelerated atherosclerosis, and increased cardiovascular morbidity and mortality in CKD. Elevated serum phosphate has been shown, across multiple cohorts, to independently predict faster CKD progression, more rapid decline in eGFR, and increased risk of ESRD and dialysis initiation^[16,17]. Patients with the combination of low calcium and high phosphate consistently demonstrate the poorest renal outcomes and the lowest eGFR values among CKD subgroups^[16,17], and the FGF-23–calcium-phosphate axis has emerged as a key mediator linking mineral metabolism disturbances to cardiovascular risk in young and older CKD populations alike^[18].

Phosphate binders are broadly divided into calcium-based agents (such as calcium carbonate and calcium acetate) and non-calcium-based agents (such as sevelamer, lanthanum carbonate, and iron-based binders). While calcium-based binders remain widely used owing to their low cost and efficacy, concerns regarding cumulative calcium loading and its potential contribution to vascular calcification have driven increased interest in non-calcium-based alternatives, particularly in patients with evidence of pre-existing vascular calcification, adynamic bone disease, or persistently low PTH^[19,20,21].

3.4 Magnesium Imbalance in CKD

Interest in magnesium as a modifiable factor in CKD has grown substantially in recent years, driven in large part by evidence linking magnesium status to vascular calcification, a major contributor to cardiovascular morbidity in this population. Magnesium is thought to inhibit vascular smooth muscle cell calcification through several mechanisms, including inhibition of hydroxyapatite crystal formation and modulation of osteogenic transdifferentiation of vascular smooth muscle cells, providing biological plausibility for the observed inverse association between serum magnesium concentration and vascular calcification burden in several CKD cohorts^[31,32].

Magnesium is the second most abundant intracellular cation and plays a critical role in enzymatic reactions, neuromuscular transmission, cardiac conduction, and bone mineral metabolism. The kidney is the principal organ responsible for magnesium homeostasis, with reabsorption occurring predominantly in the thick ascending limb of the loop of Henle via a paracellular pathway and, to a lesser extent, in the distal convoluted tubule via the transient receptor potential melastatin 6 (TRPM6) channel^[31]. In early to moderate CKD, fractional excretion of magnesium increases to compensate for reduced filtered load, maintaining normomagnesemia; however, once GFR falls substantially, this compensatory mechanism is overwhelmed, and hypermagnesemia may develop, particularly in patients using magnesium-containing antacids, laxatives, or phosphate binders^[32].

Hypomagnesemia, though less common in advanced CKD, may occur in earlier stages, particularly in the setting of diuretic use, calcineurin inhibitor therapy, or malabsorption, and has been associated epidemiologically with more rapid CKD progression, increased vascular calcification, and adverse cardiovascular outcomes, potentially reflecting magnesium's physiological role as an endogenous inhibitor of vascular smooth muscle calcification^[31,32]. Conversely, symptomatic hypermagnesemia, though relatively rare, may manifest with hyporeflexia, muscle weakness, hypotension, bradyarrhythmia, and, in severe cases, respiratory depression and cardiac arrest, and should be considered in any CKD patient presenting with unexplained neuromuscular or cardiovascular depression, especially after exposure to magnesium-containing medications^[32]. Given the emerging evidence linking magnesium status to vascular calcification and mortality risk in CKD, several authors have advocated for more routine serum magnesium monitoring, although this has not yet been universally incorporated into standard CKD biochemical panels.

4. ELECTROLYTE PATTERNS AND CKD PROGRESSION

Recent advances in electronic medical records and group-based trajectory modeling (GBTM) have improved the understanding of longitudinal electrolyte changes in patients with chronic kidney disease. GBTM is useful for identifying groups of patients who share similar biochemical patterns over time and helps in predicting disease progression. Studies involving CKD patients identified electrolyte pattern based on serum sodium, potassium, calcium, and phosphorus levels [1,8]. Among these groups, patients with persistently elevated potassium and phosphorus levels along with lower sodium and calcium concentrations were found to have a significantly higher risk of progression to dialysis compared with patients showing stable electrolyte profiles. Individuals belonging to this high-risk electrolyte pattern also exhibited a more rapid annual decline in estimated glomerular filtration rate (eGFR) and poorer renal outcomes. Furthermore, electrolyte changes observed during the initial years of follow-up were found to predict future dialysis risk, highlighting the prognostic value of continuous electrolyte monitoring in CKD management. These findings suggest that early identification of abnormal electrolyte patterns may facilitate timely therapeutic interventions and potentially delay progression to end-stage renal disease [1,8,20].

5. DIAGNOSTIC EVALUATION AND MONITORING

A structured approach to biochemical monitoring is essential for the timely detection of electrolyte disturbances across the CKD trajectory. At a minimum, serum electrolytes (sodium, potassium, chloride, bicarbonate), calcium, phosphate, magnesium, uric acid, and PTH should be assessed at intervals determined by CKD stage, with more frequent monitoring warranted in advanced disease (stages 4–5), during acute intercurrent illness, and following initiation or dose adjustment of RAAS inhibitors, diuretics, or other electrolyte-active medications [17,18,31,32].

Table 2: Representative Reference Ranges and Typical CKD-Associated Derangements

Electrolyte Parameter	Normal Range	Typical Derangement in CKD	Key Contributing Mechanism
Sodium	135–145 mmol/L	Hyponatremia (more common); occasional hypernatremia	Impaired free-water excretion; elevated ADH
Potassium	3.5 - 5.0 mmol/L	Hyperkalemia	Reduced distal excretion; RAAS-inhibitor use; acidosis
Calcium	8.5 - 10.5 mg/dL	Hypocalcemia	Reduced calcitriol; phosphate retention
Phosphate	2.5 - 4.5 mg/dL	Hyperphosphatemia	Reduced renal excretion; FGF-23/PTH resistance
Magnesium	1.7 - 2.2 mg/dL	Hypermagnesemia (advanced CKD); occasional hypomagnesemia	Reduced excretion; exogenous Mg exposure

Beyond standard chemistry panels, ECG assessment is a valuable adjunct in suspected hyperkalemia, particularly in patients with rapidly rising potassium or coexisting cardiac disease, although normal ECG findings do not exclude clinically significant hyperkalemia and should not delay treatment when clinical suspicion is high¹⁰. Trends over serial measurements, rather than isolated values, provide superior prognostic information, supporting a shift toward longitudinal, trajectory-based monitoring frameworks in routine nephrology practice^[1,5,8,17,34,35,36].

Practical monitoring protocols should also account for pre-analytical factors that can spuriously alter measured electrolyte values, most notably pseudohyperkalemia resulting from haemolysis during venepuncture, prolonged tourniquet application, or delayed sample processing, which can lead to unnecessary and potentially harmful treatment if not recognised. Clinicians are therefore advised to correlate unexpected or discordant electrolyte results with the clinical picture and, where feasible, to repeat testing on a fresh, properly handled sample before initiating emergency treatment for an isolated, unexpected laboratory abnormality, particularly in an otherwise asymptomatic patient^[14,15,23,26].

6. BIOMARKERS AND NOVEL DIAGNOSTIC APPROACHES

Beyond conventional serum electrolyte measurement, several emerging biomarkers offer additional insight into mineral and electrolyte disturbance in CKD. Intact FGF-23 rises early in the course of CKD, often before overt hyperphosphatemia is detectable, and has been proposed as a sensitive early marker of disordered phosphate handling and an independent predictor of cardiovascular events and mortality^[18]. Similarly, trajectory-based composite biomarker panels, incorporating serial measurements of potassium, phosphate, sodium, and calcium analysed via statistical trajectory modelling, have shown superior discriminative capacity for predicting dialysis initiation compared with single time-point measurements^[1,5,20].

Point-of-care testing devices for rapid potassium measurement have also gained clinical traction, particularly in emergency and dialysis settings, enabling faster therapeutic decision-making in suspected severe hyperkalemia. Continuous or near-continuous electrolyte monitoring technologies, though not yet in routine clinical use, represent an active area of research and may, in the future, allow real-time detection of dangerous electrolyte excursions in high-risk CKD and dialysis populations.

7. IMPACT OF ELECTROLYTE IMBALANCE ON QUALITY OF LIFE

CKD significantly affects physical, psychological, and social well-being. Electrolyte abnormalities contribute to fatigue, cognitive dysfunction, sleep disturbances, muscle cramps, and reduced functional capacity. Several studies evaluated HRQoL among CKD patients using the KDQoL SF-36 questionnaire and demonstrated progressive deterioration in quality-of-life scores with advancing CKD stages. Patients with stage 5 CKD had significantly lower scores in:

- Physical functioning,
- Emotional well-being,
- Sleep quality,
- Social functioning,
- Cognitive function,

Reduced GFR and hemoglobin levels were strongly associated with poor physical and mental composite scores. Electrolyte imbalance, particularly hyperkalemia and hypocalcemia, further worsened symptom burden and impaired daily functioning^[2,3,6,21].

These findings emphasize the need for comprehensive management approaches targeting both biochemical abnormalities and psychosocial health in CKD patients.

8. MANAGEMENT OF ELECTROLYTE DISTURBANCES IN CKD

Management of electrolyte disturbances in CKD requires an individualized approach that integrates dietary modification, pharmacological therapy, careful review of concurrent medications, and, where indicated, renal replacement therapy, all within the broader context of comprehensive CKD care aimed at slowing disease progression and reducing cardiovascular risk.

8.1 Management of Hyponatremia

Treatment depends on the severity and underlying cause of hyponatremia. Fluid restriction remains the cornerstone of therapy in dilutional hyponatremia. Vasopressin receptor antagonists such as tolvaptan may be beneficial in selected cases. Correction of sodium imbalance should be gradual to avoid osmotic demyelination syndrome. Monitoring volume status and avoiding excessive free water intake are essential ^[2,7,21].

8.2 Management of Hyperkalemia

Management of hyperkalemia in chronic kidney disease primarily involves dietary potassium restriction, discontinuation or dose adjustment of potassium-retaining medications, and the use of potassium-lowering therapies. Acute severe hyperkalemia is considered a medical emergency because of the risk of life-threatening cardiac arrhythmias and requires immediate treatment ^[2]. Regular monitoring of serum potassium and renal function is essential, especially in patients receiving renin-angiotensin-aldosterone system (RAAS) blockers. Dietary potassium restriction, correction of metabolic acidosis, and careful adjustment of medications that increase potassium levels are commonly recommended. In severe cases, emergency treatment such as intravenous calcium, insulin with glucose, beta-agonists, potassium-binding agents, or dialysis may be required.

Early detection and close follow-up are important because hyperkalemia is associated with increased short-term mortality in CKD patients. Proper patient education and individualized therapy can significantly reduce complications and improve patient safety in CKD management ^[2,22].

8.3 Management of Calcium, Phosphate, and CKD-MBD

Management of calcium and phosphate disturbances in CKD focuses on controlling hyperphosphatemia while correcting hypocalcemia and avoiding over suppression of PTH. Dietary phosphate restriction and phosphate-binding agents (calcium-based and non-calcium-based) form the cornerstone of hyperphosphatemia management, while vitamin D analogues and, where necessary, calcimimetic agents are used to normalize serum calcium and PTH concentrations ^[1,2,6]. Current KDIGO guidance recommends maintaining serum phosphate and calcium within, or as close as reasonably possible to, the normal range, together with individualized control of PTH, rather than pursuing rigid, one-size-fits-all biochemical targets, in order to reduce the risk of CKD-MBD-related fractures and cardiovascular calcification ^[1,2,6].

8.4 Management of Magnesium Disturbances

Given the relative rarity of severe, symptomatic magnesium disturbance in CKD, management is largely preventive: avoidance of magnesium-containing antacids and laxatives in patients with advanced CKD, cautious use of magnesium-containing phosphate binders, and periodic monitoring of serum magnesium, particularly in symptomatic patients or those on multiple magnesium-containing preparations^[31,32]. Symptomatic hypermagnesemia may require intravenous calcium gluconate for cardiac protection and, in severe cases, dialysis for definitive removal.

Table 3: First-Line and Emergency Management Strategies by Electrolyte Disturbance

Disturbance	First-Line / Chronic Management	Emergency Management
Hyponatremia	Fluid restriction; treat underlying cause; vasopressin antagonists in select cases [2,7,21]	Hypertonic saline (severe, symptomatic cases; specialist-guided, gradual correction)
Hyperkalemia	Dietary potassium restriction; RAAS-inhibitor dose review; correction of acidosis; potassium binders [2,22,26]	IV calcium, insulin-glucose, beta-2 agonists, potassium binders, dialysis
Hypocalcemia / Hyperphosphatemia	Phosphate restriction; phosphate binders; vitamin D analogues [1,2,6]	IV calcium gluconate for symptomatic severe hypocalcemia
Hypermagnesemia	Avoid magnesium-containing antacids/laxatives; monitor levels [31,32]	IV calcium gluconate; dialysis in severe cases

9. DIETARY AND NUTRITIONAL CONSIDERATIONS

Dietary counselling remains a cornerstone of electrolyte management across all stages of CKD and should be individualized by a renal dietitian wherever possible, balancing the need for electrolyte restriction against the risk of protein-energy wasting, which itself carries independent mortality risk in this population. Blanket dietary restriction without individualised assessment is discouraged, as excessive restriction of potassium- or phosphate-rich foods may inadvertently reduce intake of fruits, vegetables, and other nutrient-dense foods associated with favourable outcomes in CKD.

10. CONCLUSION

Electrolyte disturbances are common and clinically significant complications of chronic kidney disease. Abnormalities in sodium, potassium, calcium, and phosphorus contribute to cardiovascular morbidity, bone disease, neurological complications, poor quality of life, and accelerated progression to ESRD. Recent studies demonstrate that longitudinal electrolyte temporal pattern provide important prognostic information beyond single laboratory measurements. Early recognition, continuous monitoring, and individualized management of electrolyte abnormalities are essential for improving patient outcomes and delaying dialysis initiation. Comprehensive multidisciplinary care incorporating biochemical, clinical, and psychosocial interventions may substantially improve the overall health and quality of life of CKD patients.

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