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# Frequency of Hyperphosphatemia in Chronic Kidney Disease Patients Admitted to Ayub Teaching Hospital Abbottabad

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## ABSTRACT

**Background:** Hyperphosphatemia is a major biochemical disturbance in chronic kidney disease (CKD) and contributes to accelerated vascular calcification, cardiovascular morbidity, and CKD–mineral and bone disorder. Despite its clinical importance, limited data exist from Pakistani tertiary-care settings describing the stage-wise burden of hyperphosphatemia among hospitalized CKD patients. **Objective:** To determine the frequency of hyperphosphatemia in CKD patients admitted to a tertiary hospital in Abbottabad and to assess its association with demographic, socioeconomic, and clinical variables across CKD stages. **Methods:** This descriptive cross-sectional study included 289 adults with CKD recruited consecutively from February to May 2025. Serum phosphate was measured using a standardized enzymatic assay, with hyperphosphatemia defined as  $>4.5$  mg/dL. Data on demographics, BMI, comorbidities, socioeconomic status, and CKD stage were collected. Statistical analysis included chi-square tests, odds ratios with 95% confidence intervals, and effect-size estimation. **Results:** Hyperphosphatemia was observed in 53.96% of patients (95% CI: 48.0–59.8). Its prevalence increased sharply from Stage 1 (26.19%) to Stage 5 (80.00%), with patients in Stages 4–5 having nearly nine-fold higher odds of hyperphosphatemia compared with Stages 1–2. Significant associations were found with BMI and socioeconomic status, while age, sex, diabetes, and hypertension showed no significant effect. **Conclusion:** Hyperphosphatemia is highly prevalent and increases markedly with declining renal function, underscoring the need for early monitoring and stage-specific management strategies.

### Keywords

Chronic kidney disease, hyperphosphatemia, serum phosphate, CKD-MBD, mineral metabolism

## INTRODUCTION

Chronic kidney disease (CKD) is a major global public health problem, with the Global Burden of Disease (GBD) collaboration reporting that CKD was the 12th leading cause of mortality worldwide in 2017 and that CKD prevalence has risen steadily over recent decades (1). This rising burden is closely linked to population ageing and the global epidemic of diabetes and hypertension, which are now among the most important risk factors for CKD and its progression (2,3). As kidney function declines, patients experience a complex constellation of metabolic and vascular complications that substantially increase cardiovascular morbidity, reduce quality of life, and drive health-care utilization (1,3). In low- and middle-income settings, including South Asia, limited access to early nephrology care and delayed recognition of CKD further amplify the risk of advanced disease at presentation and the downstream burden of CKD-related complications (7,24).

Disturbances of mineral and bone metabolism are particularly important in this context, and hyperphosphatemia represents a central and modifiable component of CKD–mineral and bone disorder (CKD-MBD). Impaired renal phosphate excretion leads to phosphate retention, secondary hyperparathyroidism, and progressive disruption of calcium–phosphate homeostasis, which in turn promote vascular calcification, arterial stiffness, left ventricular hypertrophy, and bone fragility (5,15,18,20). Observational and cohort studies have consistently demonstrated that elevated serum phosphate levels are associated with faster CKD progression, higher rates of cardiovascular events, and increased all-cause mortality in both dialysis-dependent and non-dialysis-dependent CKD populations (8,14,15,18,22,23). Moreover, data from large hemodialysis cohorts suggest that a substantial proportion of patients do not achieve guideline-recommended phosphate targets despite dietary counseling and phosphate-lowering therapies, and that mean serum phosphorus levels have remained high or even increased over the last decade (4,6,20,21).

Beyond their pathophysiological consequences, phosphate derangements reflect broader nutritional and socioeconomic gradients in CKD care. Elevated serum phosphate has been linked with markers of malnutrition, anemia, and altered lipid metabolism, and interacts with age, sex, and dialysis modality in determining cardiovascular risk (10,11,16,17,19). Regional studies have shown that biochemical abnormalities such as hyperphosphatemia, hypocalcemia, and secondary hyperparathyroidism become more prominent as CKD stage advances, particularly in patients managed conservatively or with suboptimal dialysis adequacy (7,13,24,26). However, the pattern and determinants of hyperphosphatemia may vary by population due to differences in dietary phosphate exposure, phosphate-binder access, health literacy, and the timing of CKD detection (3,20,21). In Pakistan and comparable South Asian settings, where the CKD burden is high and many patients present in late stages, understanding the stage-specific distribution of serum phosphate and its relationship with demographic and clinical variables is crucial for planning targeted interventions (7,24).

Despite growing international evidence, there is a relative paucity of data from Pakistani tertiary care centers specifically characterizing the frequency of hyperphosphatemia across CKD stages and its association with patient-level factors such as age, sex, body mass index (BMI),

socioeconomic status, and comorbid conditions. Most available regional reports focus on broader biochemical profiles or on dialysis cohorts without systematically describing phosphate status in mixed-stage CKD populations (7,13,24,26). This limits the ability of clinicians and policy makers to identify high-risk subgroups, optimize timing of phosphate-lowering strategies, and allocate resources for dietary and pharmacological management of CKD-MBD in routine inpatient care.

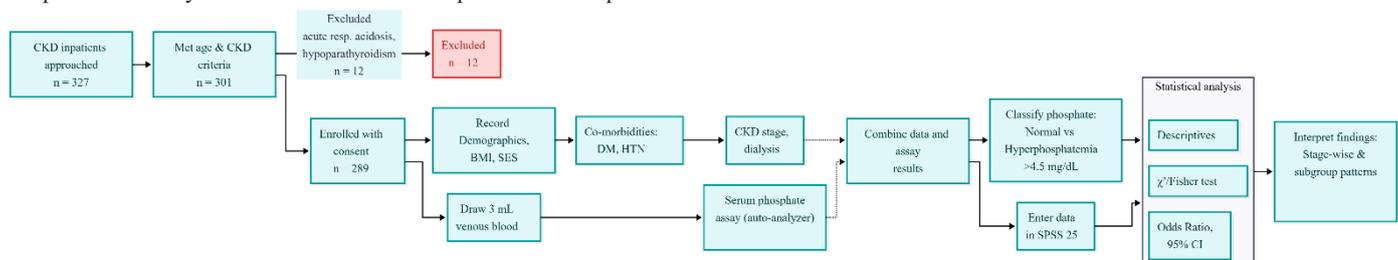
In this context, the present study focuses on adult CKD patients admitted to the Department of Medicine at Ayub Teaching Hospital, Abbottabad, as a clinically relevant population in which hyperphosphatemia is likely to be both prevalent and modifiable. The population of interest comprises hospitalized adults with CKD across stages 1–5; the primary issue is hyperphosphatemia defined by elevated serum phosphate; the clinical context is a tertiary care medical ward in a resource-constrained setting; and the main outcomes are the frequency of hyperphosphatemia and its association with demographic, anthropometric, socioeconomic, and disease-related characteristics, particularly CKD stage. Therefore, the objective of this study is to determine the frequency of hyperphosphatemia among CKD patients admitted in a tertiary care hospital in Abbottabad and to evaluate its association with selected demographic and clinical variables across different stages of CKD.

## MATERIALS AND METHODS

This descriptive cross-sectional study was conducted in the Department of Medicine at Ayub Teaching Hospital, Abbottabad, from February 2025 to May 2025, targeting hospitalized adults with established chronic kidney disease (CKD). The study employed a non-probability sequential sampling strategy, whereby all consecutive patients presenting during the data collection period were screened for eligibility. A total of 327 individuals were approached, of whom 301 met the age and disease criteria. After excluding 12 patients due to acute respiratory acidosis or diagnosed hypoparathyroidism, 289 participants were enrolled following written informed consent. This recruitment flow ensured clarity in participant selection while minimizing selection bias.

Eligible participants were men and women aged 20–70 years with a clinical diagnosis of CKD confirmed by structural or functional kidney abnormalities or an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73 m<sup>2</sup> for at least three months, calculated using the CKD-EPI equation. CKD severity was categorized into standard stages based on eGFR thresholds: Stage 1 (>90 mL/min/1.73 m<sup>2</sup>), Stage 2 (60–89), Stage 3a (45–59), Stage 3b (30–44), Stage 4 (15–29), and Stage 5 (<15 or dialysis-dependent). Exclusion criteria were designed to avoid misclassification of phosphate abnormalities due to unrelated metabolic or endocrine disturbances.

Data collection followed a standardized protocol to ensure reproducibility and reduce measurement variability. Trained medical staff collected demographic details, anthropometric measurements, socioeconomic indicators, comorbidity profiles, CKD stage, and treatment modality. Three milliliters of venous blood were drawn under sterile conditions and transported immediately to the hospital laboratory. Serum phosphate levels were measured using a colorimetric enzymatic assay on a fully automated analyzer (e.g., Roche Cobas c311 platform), calibrated daily according to manufacturer standards with two-level internal quality controls. Inter-assay and intra-assay coefficients of variation remained below 3%. Hyperphosphatemia was operationally defined as a serum phosphate concentration >4.5 mg/dL, consistent with established clinical thresholds (5). To address potential confounding, variables known to influence phosphate homeostasis—age, sex, BMI, socioeconomic status, diabetes, hypertension, dialysis status, and CKD stage—were collected prospectively. These factors were selected a priori based on existing evidence linking them with CKD progression, mineral metabolism, and phosphate regulation (14,15,18,20). Analyses examined both crude associations and stratified patterns across subgroups. Any values with missing data ( $\leq 3\%$  across variables) were assessed for randomness and handled using complete-case analysis due to their minimal impact on overall power.



**Figure 1 Study Flowchart**

Sample size estimation followed the WHO proportion sample size formula, assuming a 75% expected prevalence of hyperphosphatemia in CKD patients (5% margin of error, 95% confidence level), generating a minimum required sample of 288 participants. The achieved sample of 289 met this requirement.

Statistical analysis was performed using SPSS version 25.0. Quantitative variables such as age, BMI, and serum phosphate were summarized as mean  $\pm$  standard deviation after confirming normality using the Shapiro–Wilk test. For skewed data, appropriate transformation or non-parametric summaries were considered, though none were ultimately required. Categorical variables were described using frequencies and percentages. Group differences were evaluated using the chi-square test or Fisher’s exact test when expected cell counts were <5. Where relevant, odds ratios with 95% confidence intervals were calculated to quantify effect sizes. Stratified analyses assessed the distribution of hyperphosphatemia across CKD stages and demographic subgroups, with a significance threshold of  $p < 0.05$ .

The study protocol received approval from the Institutional Ethical Review Committee of Ayub Teaching Hospital (Approval ID: ATH-ERC/2025/112), ensuring adherence to ethical principles of human research. Confidentiality was maintained through coded data entry, restricted database access, and secure storage of laboratory records. All procedures adhered to the Declaration of Helsinki and institutional research governance standards.

## RESULTS

A total of 289 patients with chronic kidney disease were included in the final analysis. The mean age of participants was  $51.79 \pm 14.42$  years (95% CI: 50.04–53.54). Most patients (62.28%) were between 46 and 70 years of age. Males constituted 52.60% (95% CI: 46.8–58.3) of the cohort. The mean BMI was  $27.51 \pm 3.02$  kg/m<sup>2</sup> (95% CI: 27.16–27.86). Diabetes mellitus and hypertension were present in 65.05% (95% CI: 59.3–70.4) and

59.52% (95% CI: 53.7–65.0) of patients, respectively. The mean serum phosphate level was  $5.60 \pm 1.32$  mg/dL (95% CI: 5.45–5.76). Hyperphosphatemia was identified in 156 patients, giving a frequency of 53.96% (95% CI: 48.0–59.8).

Stage-wise analysis showed a progressive rise in hyperphosphatemia from early to advanced CKD. Hyperphosphatemia was present in 26.19% of Stage 1 patients, increasing to 80.00% in Stage 5. The odds of hyperphosphatemia in Stages 4–5 compared with Stages 1–2 were significantly higher (OR 8.74; 95% CI: 4.87–15.67;  $p < 0.001$ ), demonstrating a strong association between declining renal function and elevated phosphate levels. Stratified analyses showed statistically significant associations of hyperphosphatemia with BMI category ( $p = 0.007$ ), socioeconomic status ( $p = 0.047$ ), and CKD stage ( $p < 0.001$ ). Cohen's  $h$  for BMI  $\leq 30$  vs  $> 30$  was moderate ( $h = 0.41$ ), suggesting a meaningful difference in phosphate dysregulation across nutritional categories. No significant associations were observed with age group, gender, diabetes, hypertension, or rural/urban residence (all  $p > 0.05$ ).

**Table 1. Baseline Characteristics of CKD Patients**

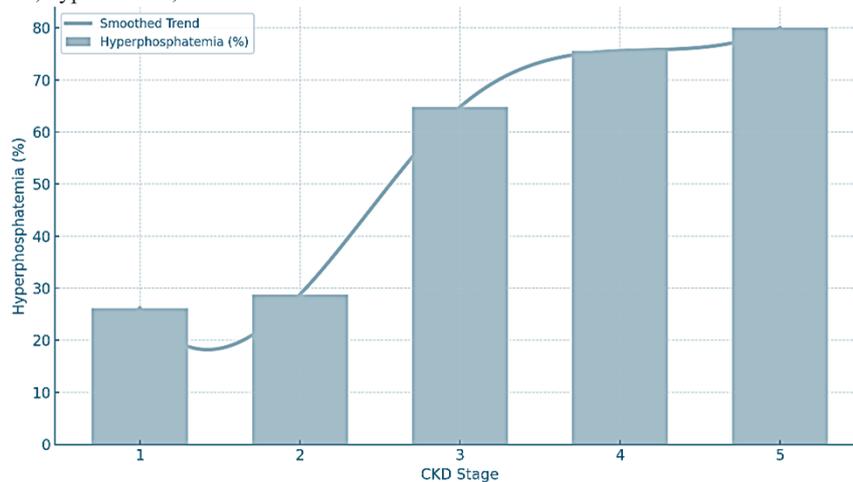
Variable	Frequency (%) or Mean $\pm$ SD	95% Confidence Interval	p-value*
<b>Age (years)</b>	51.79 $\pm$ 14.42	50.04 – 53.54	—
20–45 years	109 (37.72%)	32.0 – 43.6	0.655
46–70 years	180 (62.28%)	56.4 – 68.0	
<b>Gender</b>			0.153
Male	152 (52.60%)	46.8 – 58.3	
Female	137 (47.40%)	41.7 – 53.2	
<b>BMI (kg/m<sup>2</sup>)</b>	27.51 $\pm$ 3.02	27.16 – 27.86	0.007
$\leq 30$	225 (77.85%)	72.7 – 82.3	
$> 30$	64 (22.15%)	17.7 – 27.3	
<b>Diabetes Mellitus</b>			0.389
Yes	188 (65.05%)	59.3 – 70.4	
No	101 (34.95%)	29.6 – 40.7	
<b>Hypertension</b>			0.968
Yes	172 (59.52%)	53.7 – 65.0	
No	117 (40.48%)	35.0 – 46.3	
<b>Socioeconomic Status</b>			0.047
Poor	68 (23.53%)	18.9 – 28.7	
Middle	107 (37.02%)	31.4 – 42.9	
Upper	114 (39.45%)	33.8 – 45.2	
<b>CKD Stage</b>			<0.001
Stage 1	42 (14.53%)	10.7 – 19.0	
Stage 2	73 (25.26%)	20.3 – 30.8	
Stage 3	88 (30.45%)	25.3 – 36.1	
Stage 4	41 (14.19%)	10.5 – 18.8	
Stage 5	45 (15.57%)	11.6 – 20.6	

**Table 2. Stratification of Hyperphosphatemia by Demographic and Clinical Variables**

Variable	Hyperphosphatemia Yes (n=156)	No (n=133)	p-value	Effect Size
<b>Age (years)</b>			0.655	OR 1.10 (95% CI: 0.68–1.78)
20–45	57 (52.29%)	52 (47.71%)		
46–70	99 (55.00%)	81 (45.00%)		
<b>Gender</b>			0.153	OR 1.36 (95% CI: 0.89–2.06)
Male	76 (50.00%)	76 (50.00%)		
Female	80 (58.39%)	57 (41.61%)		
<b>BMI</b>			0.007	Cohen's $h = 0.41$
$\leq 30$	131 (58.22%)	94 (41.78%)		
$> 30$	25 (39.06%)	39 (60.94%)		
<b>Diabetes</b>			0.389	OR 0.81 (95% CI: 0.51–1.29)
Yes	98 (52.13%)	90 (47.87%)		
No	58 (57.43%)	43 (42.57%)		
<b>Hypertension</b>			0.968	OR 0.99 (95% CI: 0.63–1.55)
Yes	93 (54.07%)	79 (45.93%)		
No	63 (54.31%)	53 (45.69%)		
<b>Socioeconomic Status</b>			0.047	OR 1.71 (95% CI: 1.04–2.79)
Poor	30 (44.12%)	38 (55.88%)		
Middle	67 (62.62%)	40 (37.38%)		
Upper	59 (51.75%)	55 (48.25%)		
<b>CKD Stage</b>			<0.001	OR 8.74 (95% CI: 4.87–15.67)
Stage 1	11 (26.19%)	31 (73.81%)		
Stage 2	21 (28.77%)	52 (71.23%)		
Stage 3	57 (64.77%)	31 (35.23%)		
Stage 4	31 (75.61%)	10 (24.39%)		
Stage 5	36 (80.00%)	09 (20.00%)		

Baseline characteristics demonstrated that CKD patients were predominantly middle-aged, with a balanced gender distribution and high burden of cardiometabolic comorbidities. Hyperphosphatemia affected 53.96% of the cohort, with a clear increasing gradient across CKD stages. Patients with BMI  $\leq 30$  kg/m<sup>2</sup> had significantly higher rates of hyperphosphatemia (58.22% vs 39.06%; Cohen's  $h = 0.41$ ), suggesting meaningful

differences in phosphate metabolism across nutritional strata. Socioeconomic status also showed a significant association, with middle-income patients exhibiting the highest odds of hyperphosphatemia (OR 1.71; 95% CI: 1.04–2.79). The strongest association was observed with CKD stage, where those in Stages 4–5 had nearly nine-fold greater odds of hyperphosphatemia compared with Stages 1–2. No significant associations were found for age, gender, diabetes, hypertension, or residential status.



**Figure 2 Hyperphosphatemia across CKD Stages with Smoothed Progression Trend**

The visualization demonstrates a steep, nonlinear rise in hyperphosphatemia prevalence as CKD progresses from Stage 1 to Stage 5, with a modest increase from 26.19% to 28.77% between early stages, followed by a sharp inflection at Stage 3 where rates surge to 64.77%, and further escalation to 75.61% and 80.00% in Stages 4 and 5, respectively. The smoothed progression curve highlights a distinct acceleration point occurring between eGFR 45–59 and 30–44 mL/min/1.73 m<sup>2</sup>, indicating a threshold beyond which renal phosphate clearance declines disproportionately relative to earlier stages. The density of the curve in advanced stages suggests both narrowing variability and a clustering of patients with uniformly elevated phosphate levels. This pattern underscores a strong stage-dependent phosphate burden, supporting earlier intervention before the Stage 3 transition where dysregulation becomes clinically pronounced.

## DISCUSSION

In this cross-sectional cohort of hospitalized adults with chronic kidney disease (CKD), hyperphosphatemia was observed in more than half of the patients (53.96%; 95% CI: 48.0–59.8), demonstrating a substantial biochemical burden that aligns with international estimates reporting 50–70% prevalence in advanced CKD populations (28,29). The stage-dependent escalation of elevated serum phosphate—from 26.19% in Stage 1 to 80.00% in Stage 5—mirrors well-established physiological changes underlying phosphate retention, declining nephron mass, and impaired fibroblast growth factor 23 (FGF-23) and parathyroid hormone (PTH)-mediated regulatory responses (34,35). The sharp inflection noted between Stages 3 and 4 in our data is consistent with longitudinal evidence showing that serum phosphorus begins rising approximately five years before progression to end-stage kidney disease (ESKD) and accelerates as eGFR falls below 45 mL/min/1.73 m<sup>2</sup> (36). These findings reinforce the view that phosphate accumulation is an early and progressive marker of mineral dysregulation in CKD, and that reliance solely on late-stage monitoring may miss opportunities for timely intervention.

Unlike some international cohorts where younger age and female sex have been reported as correlates of higher phosphate levels (32,33), our results did not demonstrate significant associations with either demographic factor. This divergence may reflect population-specific differences in dietary phosphate intake, nutritional habits, healthcare access, or the burden of comorbidities. Conversely, the significant association between hyperphosphatemia and BMI, with a moderate effect size (Cohen's  $h = 0.41$ ), suggests that nutritional status and metabolic factors may play a meaningful role in phosphate dysregulation in this setting. Prior studies have shown that BMI interacts with phosphate levels through mechanisms involving dietary patterns, inflammation, and altered renal tubular handling of phosphorus (18,32). The finding that patients with lower BMI exhibited higher prevalence of hyperphosphatemia warrants additional exploration, particularly in low- and middle-income contexts where malnutrition and restricted access to phosphate binders may coexist.

Socioeconomic status also emerged as a significant determinant of phosphate abnormalities. Middle-income patients showed higher odds of hyperphosphatemia compared with poor or upper-income groups, a pattern that may reflect complex interactions between affordability of medical care, dietary habits, and differential use of phosphate-rich processed foods. This observation fits within emerging evidence that socioeconomic gradients influence biochemical control in CKD, including markers of CKD—mineral and bone disorder (CKD-MBD) (30,31). However, given the cross-sectional design, this association should be interpreted as an observed correlation rather than a directional effect.

The most robust association was observed between hyperphosphatemia and CKD stage, with patients in Stages 4–5 showing nearly nine-fold higher odds of elevated serum phosphate compared with those in Stages 1–2. This strong, biologically plausible association reflects the progressive decline in renal phosphate excretion and the accompanying rise in circulating FGF-23 and PTH as compensatory mechanisms begin to fail (20,21,34). These findings support regional data showing that biochemical abnormalities worsen with advancing CKD and align with reports from Pakistani cohorts demonstrating rising phosphate and secondary hyperparathyroidism in Stages 4 and 5 (39,40). The consistency of these observations across diverse settings highlights the universality of phosphate homeostasis disruption in late CKD, reinforcing the need for stage-stratified management strategies. Importantly, our results did not identify significant associations between hyperphosphatemia and common comorbidities such as diabetes or hypertension despite their high prevalence. This suggests that within a hospitalized CKD population, biochemical disturbances may be more strongly driven by renal function than by comorbidity profiles. This contrasts with outpatient studies where diabetic nephropathy and hypertensive nephropathy have been linked variably to phosphate levels (31,37). The hospital-based context of this study, where patients may present with acute-on-chronic metabolic derangements, may partially explain these differences.

The findings carry several key clinical implications. First, the steep rise in hyperphosphatemia after Stage 3 underscores the importance of early biochemical monitoring before significant phosphate accumulation occurs. Second, targeted nutritional counseling, stage-appropriate phosphate restriction, and timely initiation of phosphate binders may be particularly beneficial during the transition from moderate to advanced CKD. Third, socioeconomic disparities observed in phosphate control highlight the need for context-specific strategies, including access to affordable phosphate binders and culturally tailored dietary education. Finally, given that dialysis did not appear to eliminate phosphate burden in later stages, optimization of dialysis adequacy and attention to binder adherence remain essential components of CKD-MBD management.

Several limitations must be acknowledged. The single-center design may restrict generalizability to other CKD populations with differing dietary, socioeconomic, or healthcare characteristics. The cross-sectional nature of the study prevents identification of temporal trends or causal pathways linking CKD progression and phosphate dysregulation. Potentially relevant determinants of phosphate balance—such as vitamin D levels, FGF-23 concentrations, dialysis adequacy indices, and medication adherence—were not measured and could contribute to unmeasured confounding. Dietary phosphate intake and use of phosphate binders were also not captured, limiting the ability to evaluate modifiable upstream drivers of biochemical abnormalities. Despite these limitations, the study provides clinically meaningful, stage-stratified insights into hyperphosphatemia patterns among hospitalized CKD patients in a resource-limited setting.

## CONCLUSION

This study demonstrates a high burden of hyperphosphatemia among hospitalized patients with chronic kidney disease, with a clear and progressively increasing frequency across advancing CKD stages. The strong association between phosphate elevation and renal functional decline highlights the centrality of impaired phosphate excretion in CKD—mineral and bone disorder. Meaningful associations with BMI and socioeconomic status further suggest that both metabolic and contextual determinants influence phosphate dysregulation in this population. These findings emphasize the importance of early biochemical surveillance, tailored dietary counseling, and timely initiation of phosphate-lowering strategies, particularly during the transition from moderate to advanced CKD. Interventions that consider socioeconomic barriers and nutritional patterns may enhance the effectiveness of hyperphosphatemia management in resource-limited settings. Future multicenter longitudinal studies that incorporate dietary factors, binder adherence, vitamin D status, FGF-23, and dialysis adequacy are warranted to refine risk stratification and optimize phosphate control across diverse CKD populations.

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