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Original Research Article

Frequency of Serum Calcium Imbalance in Chronic Kidney Disease Patients

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Abstract: *Chronic kidney disease (CKD) is frequently accompanied by disturbances in mineral metabolism, particularly abnormalities in serum calcium homeostasis. Calcium imbalance, including hypocalcemia and hypercalcemia, contributes to chronic kidney disease-mineral and bone disorder and may worsen skeletal and cardiovascular outcomes. Local evidence regarding the burden of serum calcium imbalance in Pakistani CKD patients remains limited.* ***Objective:*** *To determine the frequency of serum calcium imbalance in patients with chronic kidney disease and to evaluate its association with CKD stage and selected biochemical and clinical variables.* ***Methods:*** *This cross-sectional study was conducted in the Department of Chemical Pathology in collaboration with the Kidney Center, Quaid-e-Azam Medical College/B.V. Hospital, Bahawalpur, Pakistan, over six months from 19 February 2025 to 19 August 2025. A total of 97 patients aged 18–60 years with CKD stages I–IV were enrolled through non-probability consecutive sampling. Patients with CKD stage V, dialysis-dependent disease, acute kidney injury, or conditions and medications affecting calcium metabolism were excluded. Demographic and clinical data were recorded, and laboratory investigations included serum calcium, phosphate, albumin, renal function tests, and estimated glomerular filtration rate (eGFR). Corrected calcium was calculated in patients with hypoalbuminemia. Data were analyzed using SPSS version 26. Associations were assessed using the chi-square test, with p<0.05 considered statistically significant.* ***Results:*** *The mean age of participants was 44.18 ± 10.27 years, and 55.7% were male. Overall, 52 of 97 patients (53.6%) had serum calcium imbalance. Hypocalcemia was observed in 41 patients (42.3%), whereas hypercalcemia was found in 11 patients (11.3%). The frequency of calcium imbalance increased progressively with worsening CKD stage, from 22.2% in stage I to 66.7% in stage IV. Serum calcium imbalance was significantly associated with elevated serum phosphate levels (p=0.003) and advanced CKD stage (p=0.009). Patients with calcium imbalance also had lower mean eGFR and serum albumin levels than those of normocalcemic patients.* ***Conclusion:*** *Serum calcium imbalance was highly prevalent in non-dialysis CKD patients, with hypocalcemia as the predominant abnormality. Advanced CKD stage and hyperphosphatemia were significant correlates, supporting the need for routine surveillance of mineral metabolism in CKD patients to enable earlier intervention and reduce CKD-related complications.*
Keywords: Chronic Kidney Disease; Hypocalcemia; Hypercalcemia; Calcium; Phosphates; Glomerular Filtration Rate

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Introduction

Chronic kidney disease (CKD) is a progressive and irreversible condition characterized by a gradual decline in renal function, culminating in end-stage renal disease (ESRD) if left unmanaged. It is defined by the presence of kidney damage or a glomerular filtration rate (GFR) below 60 mL/min/1.73 m² for more than three months, and has emerged as a major public health burden globally (1). The global prevalence of CKD is estimated to exceed 10–15%, affecting hundreds of millions of individuals and imposing substantial morbidity, mortality, and healthcare costs (2). In Pakistan, CKD represents an escalating epidemic, with an estimated prevalence of 12.5% in the adult population. However, it remains largely underdiagnosed due to limited health infrastructure, inadequate awareness, and resource constraints (3).

As renal function deteriorates, a cascade of metabolic derangements ensues, among which disturbances in mineral metabolism are particularly prominent and clinically consequential. Serum calcium imbalance, encompassing both hypocalcemia and hypercalcemia, is a well-recognized complication of CKD and plays a central role in the pathogenesis of chronic kidney disease-mineral and bone disorder (CKD-MBD) (4). The kidneys are vital for calcium homeostasis through their roles in the hydroxylation of vitamin D to its active form, 1,25-dihydroxyvitamin D (calcitriol), in regulating parathyroid hormone (PTH), and in modulating phosphate excretion. As CKD progresses, these regulatory mechanisms become progressively impaired, leading to reduced intestinal calcium absorption, secondary hyperparathyroidism, and altered calcium-phosphate dynamics (5).

Hypocalcemia is the predominant form of calcium imbalance in non-dialysis-dependent CKD patients. It is attributable to impaired calcitriol synthesis, phosphate retention leading to calcium binding and precipitation, and skeletal resistance to PTH (6). Hypercalcemia, though less common, may arise from over-suppression of PTH by pharmacological doses of active vitamin D analogues, adynamic bone disease, or excessive calcium supplementation (7). Both ends of the calcium imbalance spectrum are associated with adverse outcomes, including cardiovascular calcification, renal osteodystrophy, increased fracture risk, and higher all-cause mortality (8).

The relationship between CKD stage and the degree of calcium dysregulation is well established in high-income countries; however, data from low- and middle-income countries (LMICs), including Pakistan, remain sparse. Studies have shown that serum phosphate levels, albumin concentrations, and the estimated glomerular filtration rate (eGFR) are closely interlinked with calcium status in CKD patients (9). Hyperphosphatemia, which develops due to diminished renal phosphate clearance in advanced CKD, directly lowers ionized calcium through physicochemical interactions and further stimulates PTH secretion, thereby perpetuating secondary hyperparathyroidism and contributing to hypocalcemia (10).

Despite the clinical importance of monitoring serum calcium in CKD, there is a paucity of local evidence on the frequency and determinants of calcium imbalance in Pakistani patients with CKD. Tertiary care hospitals in Pakistan frequently manage patients presenting in moderate-to-advanced CKD stages due to delayed diagnosis, limited access to nephrology services, and poor follow-up. Understanding the prevalence of calcium dysregulation across CKD stages and its association with key biochemical parameters, such as serum phosphate, albumin, and eGFR, is essential for guiding evidence-based clinical management in resource-limited settings. The present study was therefore conducted to determine the frequency of serum calcium imbalance in patients with CKD presenting to a tertiary care hospital in Pakistan, and to explore its associations with CKD stage and relevant biochemical and clinical variables. These findings are expected to inform local clinical guidelines and highlight the need for early surveillance of mineral metabolism in Pakistani patients with CKD.

Methodology

This cross-sectional study was conducted in the Department of Chemical Pathology in collaboration with the Kidney Center, Quaid-e-Azam Medical College/B.V. Hospital, Bahawalpur, over a period of six months, from 19 February 2025 to 19 August 2025, after approval of the synopsis. The study aimed to determine the frequency of serum calcium imbalance in patients with chronic kidney disease. The sample size was calculated using the WHO sample size calculator. It yielded 97 patients, based on a 95% confidence level, 10% absolute precision, and an expected mean serum calcium level of 9.5 ± 0.5 mg/dL. Participants were enrolled through non-probability consecutive sampling.

Male and female patients aged 18–60 years with chronic kidney disease stages I–IV were included. Chronic kidney disease was defined as kidney damage or decreased kidney function persisting for at least three months, as evidenced by albuminuria or an estimated glomerular filtration rate below 90 mL/min/1.73 m². Patients with CKD stage V, dialysis-dependent disease, or acute kidney injury were excluded. Patients receiving medications known to significantly affect calcium metabolism within the previous month, including calcium or vitamin D supplements, bisphosphonates, calcimimetics, and thiazide diuretics, were also excluded. Similarly, patients with comorbid disorders that could independently alter calcium homeostasis or influence CKD progression, including primary hyperparathyroidism, hypercalcemia of malignancy, and malabsorption syndromes, were not enrolled.

Following institutional review board ethical approval, written informed consent was obtained from all participants prior to enrollment. Demographic and clinical details, including age, sex, body mass index, smoking status, hypertension, diabetes mellitus, cardiovascular disease, and duration of chronic kidney disease, were recorded on a predesigned proforma. Laboratory investigations included renal function tests, serum albumin, serum calcium, and serum phosphate levels. The estimated glomerular filtration rate was calculated from serum creatinine values, and patients were categorized into CKD stages according to standard eGFR-based criteria. Blood samples were collected via standard venipuncture, allowed to clot, and centrifuged to separate serum. All biochemical analyses were performed using the Roche Cobas c303 chemistry analyzer.

Serum calcium imbalance was defined as either hypocalcemia or hypercalcemia. Hypocalcemia was defined as serum calcium <8.5 mg/dL, normocalcemia as 8.5–10.5 mg/dL, and hypercalcemia as serum calcium >10.5 mg/dL. In patients with serum albumin levels below 3.5 g/dL, corrected calcium was calculated using the formula: corrected calcium = measured serum calcium + 0.8 × (4 − serum albumin). The frequency of calcium imbalance was recorded, and affected patients were further classified into hypocalcemia or hypercalcemia.

Data were analyzed using SPSS version 26. Quantitative variables were summarized as mean ± standard deviation, whereas qualitative variables were presented as frequencies and percentages. Stratified analyses were carried out by age, sex, body mass index, duration of CKD, serum phosphate level, and CKD stage to assess their associations with serum calcium imbalance. Post-stratification chi-square testing was applied, and a p-value of <0.05 was considered statistically significant.

Results

A total of 97 patients with chronic kidney disease were included in the analysis. The mean age of the participants was 44.18 ± 10.27 years, and the mean body mass index was 26.11 ± 4.36 kg/m². There were 54 (55.7%) males and 43 (44.3%) females. Hypertension was the most frequent comorbidity, followed by diabetes mellitus and cardiovascular disease. The mean duration of CKD was 3.94 ± 1.88 years. Most patients had CKD stage III or IV, reflecting the usual tertiary-care presentation pattern in Pakistani hospital settings (Table 1).

Table 1: Baseline demographic and clinical characteristics of patients with chronic kidney disease (n=97)

Variable	Value
Age (years), mean ± SD	44.18 ± 10.27
BMI (kg/m²), mean ± SD	26.11 ± 4.36
Duration of CKD (years), mean ± SD	3.94 ± 1.88
Serum albumin (g/dL), mean ± SD	3.61 ± 0.49
Serum phosphate (mg/dL), mean ± SD	4.87 ± 0.93
Serum calcium (mg/dL), mean ± SD	8.63 ± 0.92
eGFR (mL/min/1.73 m²), mean ± SD	38.74 ± 17.65
Male	54 (55.7%)
Female	43 (44.3%)
Hypertension	63 (64.9%)
Diabetes mellitus	39 (40.2%)
Cardiovascular disease	18 (18.6%)
Smoking	24 (24.7%)
CKD stage I	9 (9.3%)
CKD stage II	18 (18.6%)
CKD stage III	37 (38.1%)
CKD stage IV	33 (34.0%)

Overall, 52 (53.6%) patients had serum calcium imbalance, while 45 (46.4%) had normocalcemia. Among patients with calcium imbalance, hypocalcemia was more frequent than hypercalcemia, observed in 41 (42.3%) patients, whereas 11 (11.3%) had hypercalcemia (Table 2). This pattern is consistent with the expected predominance of hypocalcemia in non-dialysis CKD patients (Table 2).

Table 2: Frequency of serum calcium imbalance in chronic kidney disease patients (n=97)

Calcium status	Frequency	Percentage
Normocalcemia	45	46.4
Hypocalcemia	41	42.3
Hypercalcemia	11	11.3
Any calcium imbalance	52	53.6

Biochemical analysis showed that patients with calcium imbalance had lower mean serum albumin and eGFR and higher mean serum phosphate levels than normocalcemic patients. Patients with hypocalcemia had the lowest mean calcium levels, whereas those with hypercalcemia had the highest corrected calcium values (Table 3).

When CKD stage was analyzed, the frequency of calcium imbalance increased progressively with worsening renal function. Calcium imbalance was noted in 22.2% of stage I patients, 38.9% of stage II patients, 56.8% of stage III patients, and 72.7% of stage IV patients. Hypocalcemia showed a clear increasing trend across CKD stages, whereas hypercalcemia remained less common across all stages (Table 4).

In stratified analysis, serum calcium imbalance was more frequent among patients aged over 40 years, those with higher BMI, longer duration of CKD, elevated serum phosphate, and advanced CKD stage. Statistically significant associations were observed between serum phosphate level and CKD stage, whereas age, gender, BMI, and duration of CKD showed non-significant or borderline associations (Table 5). This suggests that worsening renal dysfunction and phosphate retention may be the strongest correlates of calcium imbalance in this cohort (Table 5).

Table 3: Comparison of selected biochemical parameters according to calcium status

Variable	Normocalcemia (n=45)	Hypocalcemia (n=41)	Hypercalcemia (n=11)	Overall
Serum calcium (mg/dL), mean ± SD	9.21 ± 0.44	7.92 ± 0.39	10.94 ± 0.31	8.63 ± 0.92
Serum albumin (g/dL), mean ± SD	3.73 ± 0.42	3.48 ± 0.51	3.57 ± 0.47	3.61 ± 0.49
Serum phosphate (mg/dL), mean ± SD	4.36 ± 0.71	5.29 ± 0.88	5.12 ± 0.79	4.87 ± 0.93
eGFR (mL/min/1.73 m²), mean ± SD	47.62 ± 15.11	31.85 ± 13.94	29.73 ± 11.62	38.74 ± 17.65

Table 4: Distribution of calcium status according to CKD stage (n=97)

CKD stage	Total n	Normocalcemia	Hypocalcemia	Hypercalcemia	Any imbalance
Stage I	9	7 (77.8%)	2 (22.2%)	0 (0.0%)	2 (22.2%)
Stage II	18	11 (61.1%)	6 (33.3%)	1 (5.6%)	7 (38.9%)
Stage III	37	16 (43.2%)	17 (45.9%)	4 (10.8%)	21 (56.8%)
Stage IV	33	11 (33.3%)	16 (48.5%)	6 (18.2%)	22 (66.7%)
Total	97	45 (46.4%)	41 (42.3%)	11 (11.3%)	52 (53.6%)

Table 5: Stratification of serum calcium imbalance with effect modifiers (n=97)

Variable	Category	Calcium imbalance present n (%)	Calcium imbalance absent n (%)	p-value
Age	18–40 years (n=35)	15 (42.9)	20 (57.1)	0.091
	41–60 years (n=62)	37 (59.7)	25 (40.3)	
Gender	Male (n=54)	30 (55.6)	24 (44.4)	0.712
	Female (n=43)	22 (51.2)	21 (48.8)	
BMI	<25 kg/m² (n=39)	17 (43.6)	22 (56.4)	0.118
	≥25 kg/m² (n=58)	35 (60.3)	23 (39.7)	
Duration of CKD	≤3 years (n=42)	18 (42.9)	24 (57.1)	0.084
	>3 years (n=55)	34 (61.8)	21 (38.2)	
Serum phosphate	≤4.5 mg/dL (n=36)	12 (33.3)	24 (66.7)	0.003
	>4.5 mg/dL (n=61)	40 (65.6)	21 (34.4)	
CKD stage	I–II (n=27)	9 (33.3)	18 (66.7)	0.009
	III–IV (n=70)	43 (61.4)	27 (38.6)	

Discussion

The present study examined the frequency of serum calcium imbalance in 97 patients with CKD attending a tertiary care hospital in Pakistan. Our findings reveal that more than half of the cohort (53.6%) demonstrated some form of calcium imbalance, with hypocalcemia being considerably more prevalent (42.3%) than hypercalcemia (11.3%). Furthermore, the burden of calcium dysregulation increased progressively with advancing CKD stage, and serum phosphate levels and CKD stage were identified as statistically significant correlates of calcium imbalance.

The overall frequency of hypocalcemia observed in our study (42.3%) is consistent with reports from other LMICs. It aligns with the pathophysiological expectation of diminished calcitriol synthesis and phosphate retention in non-dialysis CKD populations. Moe et al. (11) demonstrated in a large multinational cohort that hypocalcemia is the predominant mineral disorder in CKD stages III–IV, with a prevalence approaching 40–50%, attributable primarily to impaired renal 1-alpha-hydroxylase activity. Our data corroborate these findings, particularly in the subgroup with stage III and IV disease.

The progressive increase in calcium imbalance from CKD stage I (22.2%) to stage IV (72.7%) observed in our cohort mirrors findings reported by Ketteler et al. (12), who documented a stepwise worsening of mineral and bone disorder parameters with declining eGFR. Similarly, Isakova et al. (13) in the CRIC study reported that serum calcium abnormalities became increasingly prevalent as kidney function declined, reinforcing the utility of CKD staging for predicting mineral metabolism disturbances.

Serum phosphate levels greater than 4.5 mg/dL were significantly associated with calcium imbalance in our analysis ($p = 0.003$). This finding is supported by Gutiérrez et al. (14), who demonstrated that hyperphosphatemia directly suppresses serum calcium by promoting calcium-phosphate precipitation and stimulating fibroblast growth factor-23 (FGF-23) secretion, which in turn inhibits calcitriol production. The inverse relationship between serum phosphate and calcium observed in our cohort aligns with this mechanistic framework.

Patients with calcium imbalance in our study exhibited lower mean eGFR (31.85 ± 13.94 mL/min/1.73 m² in hypocalcemic patients) and lower mean serum albumin (3.48 ± 0.51 g/dL) compared with normocalcemic counterparts. This is consistent with the findings of Vervloet et al. (15), who reported that hypoalbuminemia, by affecting corrected calcium calculations, frequently masks true calcium abnormalities in CKD patients, underscoring the importance of using albumin-corrected serum calcium values in clinical practice.

Hypercalcemia, though less frequent (11.3%) in our cohort, was disproportionately concentrated in CKD stage IV patients (18.2%). Cunningham et al. (16) reported that hypercalcemia in advanced CKD is commonly associated with adynamic bone disease and excessive use of calcium-based phosphate binders or calcitriol. This pattern may be emerging in Pakistani tertiary care settings where supplementation practices are not always protocol-driven.

Gender and BMI were not significantly associated with calcium imbalance in our study, which is consistent with observations by Cheung et al. (17), who found that sex-based differences in mineral metabolism in CKD were not clinically meaningful after adjusting for renal function and nutritional status. The borderline association of longer CKD duration with calcium imbalance ($p = 0.084$) in our cohort suggests a temporal dimension to mineral dysregulation that warrants longitudinal evaluation in future studies.

In the Pakistani context, a study by Naqvi et al. (18) reported a high burden of mineral metabolism disorders among CKD patients at a Karachi-based tertiary centre, with hypocalcemia and hyperphosphatemia noted in over 45% of patients at presentation. Our findings extend this evidence to a different geographic region and reinforce the conclusion that calcium dysregulation is a common and clinically significant complication in Pakistani CKD patients, particularly at advanced stages. The predominance of CKD stage III and IV in our cohort reflects the typical delayed presentation pattern at tertiary care hospitals in Pakistan, where primary nephrology referral infrastructure remains underdeveloped.

Taken together, our findings underscore the need for routine monitoring of serum calcium, phosphate, and albumin in CKD patients across all stages, with heightened vigilance in those with advanced disease and hyperphosphatemia. Early identification of calcium dysregulation can facilitate timely intervention with dietary phosphate restriction, appropriate vitamin D supplementation, and optimization of calcium-based therapies, all of which are endorsed by current KDIGO guidelines (19, 20). Integrating standardized mineral metabolism surveillance protocols into Pakistani nephrology practice is essential to reducing the burden of CKD-MBD and its associated cardiovascular and skeletal complications.

Conclusion

More than half of the studied CKD patients had serum calcium imbalance, predominantly hypocalcemia. The risk increased with worsening renal function and higher serum phosphate, suggesting that early biochemical monitoring may improve clinical management in CKD.

Declarations

Data Availability statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Approved by the department concerned. (IRBEC-DBVH-91-24)

Consent for publication

Approved

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Conflict of interest

The authors declared no conflict of interest.

Author Contribution

SUD (PGR)

Contributed to study design, data collection and initial manuscript drafting

Assisted in data acquisition, literature review and manuscript editing

Performed statistical analysis and contributed to the interpretation of results

SH (Professor)

Contributed to patient recruitment, data entry and results compilation

Assisted in referencing, proofreading and final revisions of the manuscript

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the study's integrity.

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