

## Stage-Related Changes in iPTH, Phosphate, and Corrected Calcium in Chronic Kidney Disease: A Retrospective Observational Study

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### Abstract

### Original Research Article

**Background:** Chronic kidney disease-mineral and bone disorder (CKD-MBD) and secondary hyperparathyroidism (SHPT) are common complications of non-dialysis chronic kidney disease (CKD) and generally become more pronounced as kidney function declines. This study aimed to describe stage-stratified patterns of intact parathyroid hormone (iPTH), phosphate, and albumin-corrected calcium in a hospital-based CKD cohort. **Methods:** We conducted a retrospective single-center laboratory-based study at the Clinical Biochemistry Laboratory of Arrazi Hospital, CHU Mohammed VI, Marrakech, Morocco, between January and December 2025. We included 150 non-dialysis patients with eGFR <60 mL/min/1.73 m<sup>2</sup> (CKD stages G3a–G5) and retained one index sample per patient. Eligible patients had concurrent measurements of iPTH, total calcium, albumin, phosphate, and creatinine. Corrected calcium was calculated as total calcium (mg/L) + 0.8 × (40 – albumin [g/L]), and eGFR was estimated using the CKD-EPI 2021 equation. Stage comparisons were performed with non-parametric tests, and correlations were assessed using Spearman's rho. **Results:** The median age was 46.50 years (IQR 33.25–63.75), and 54.0% of patients were male. Median eGFR was 12.24 mL/min/1.73 m<sup>2</sup> (IQR 6.26–31.93), indicating a cohort largely composed of advanced CKD, with G5 accounting for 56.0% of cases. Median iPTH was 269.00 pg/mL (IQR 159.25–612.25), corrected calcium was 86.71 mg/L (IQR 82.85–89.63), and phosphate was 46.00 mg/L (IQR 37.00–58.00). Hyperphosphatemia (>45 mg/L) was present in 52.7% of patients, and corrected-calcium-defined hypocalcemia (<85 mg/L) in 38.7%. Across CKD stages, iPTH and phosphate increased significantly, whereas corrected calcium declined modestly. Median iPTH rose from 144.00 pg/mL in G3a to 448.50 pg/mL in G5 (p < 0.0001), while hyperphosphatemia increased from 11.8% to 70.2% (p < 0.0001). iPTH correlated inversely with eGFR (rho = -0.545; p < 0.001). **Conclusion:** In this predominantly advanced non-dialysis CKD cohort, CKD-MBD abnormalities were frequent and strongly stage-dependent. Hyperphosphatemia became highly prevalent from G4 onward, and corrected-calcium-defined hypocalcemia was common in G5. Overall, declining kidney function was closely associated with worsening CKD-MBD abnormalities, particularly higher iPTH and phosphate levels.

**Keywords:** chronic kidney disease; CKD-MBD; secondary hyperparathyroidism; phosphate; corrected calcium; non-dialysis CKD.

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## INTRODUCTION

Chronic kidney disease (CKD) is often complicated by chronic kidney disease-mineral and bone disorder (CKD-MBD), a systemic syndrome characterized by disturbances in calcium, phosphate, parathyroid hormone (PTH), and vitamin D, together with abnormalities of bone turnover and extra-skeletal calcification. Secondary hyperparathyroidism (SHPT) is a major endocrine component of CKD-MBD and generally becomes more frequent and more severe as

kidney function declines. Current guidelines therefore recommend interpreting PTH, phosphate, and calcium together, especially in advanced CKD. [1]

As renal phosphate excretion falls, phosphate retention develops and triggers compensatory endocrine responses, including increased PTH secretion. At the same time, impaired renal activation of vitamin D and disturbances in calcium balance further stimulate parathyroid activity. Although biochemical profiles are influenced by comorbidities and treatment exposure

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(e.g., phosphate binders, vitamin D analogs, and calcimimetics), advanced CKD is generally associated with higher PTH concentrations and a greater burden of hyperphosphatemia. [1]

CKD staging based on estimated glomerular filtration rate (eGFR) provides a practical framework for stratifying disease severity and examining stage-related gradients in CKD-MBD abnormalities. KDIGO defines CKD categories using eGFR thresholds, including stages G3a–G5, which makes stage-based comparisons clinically meaningful and supports structured reporting of biomarker distributions across disease severity. [2]

This study aimed to describe the stage-stratified distribution of intact PTH (iPTH), phosphate, and albumin-corrected calcium in a hospital-based cohort of non-dialysis CKD patients and to assess the prevalence of major mineral abnormalities across CKD stages. We also explored correlations between kidney function and CKD-MBD biochemical markers in order to provide local descriptive data relevant to routine laboratory practice. [1,2]

## MATERIALS AND METHODS

### Study design, setting, and period

We conducted a single-center retrospective laboratory-based observational study in the Clinical Biochemistry Laboratory of Arrazi Hospital, CHU Mohammed VI, Marrakech, Morocco, covering the period from January to December 2025. The manuscript was prepared in accordance with STROBE reporting principles for observational studies. [3]

### Study population and eligibility criteria

We included 150 adult patients with eGFR <60 mL/min/1.73 m<sup>2</sup>, corresponding to CKD stages G3a to G5, who were not receiving maintenance dialysis at the time of sampling. Patients were identified from laboratory records based on the availability of concurrent iPTH, total calcium, albumin, phosphate, and creatinine results during the study period. CKD stage definitions were based on KDIGO eGFR categories. [2] Patients receiving hemodialysis or peritoneal dialysis and kidney transplant recipients were excluded. Dialysis and transplant status were determined from the available laboratory and clinical records at the time of sample selection.

### Index sample selection

To avoid repeated-measures bias and within-subject correlation, we retained one index sample per patient. When more than one laboratory record was available, the index sample was defined as the sampling episode corresponding to the iPTH measurement selected for this study, and the accompanying biochemical parameters (total calcium, albumin, phosphate, and creatinine) were extracted from the same sampling episode.

### Laboratory platform and assays

All measurements were performed on the Abbott Alinity ci integrated platform, which includes the Alinity i immunoassay module and the Alinity c chemistry module. [4]

- Intact PTH (iPTH): iPTH was measured on the Alinity i immunoassay module using the manufacturer's in vitro diagnostic assay. [5]
- Inorganic phosphate: phosphate was measured on the Alinity c chemistry module using a phosphomolybdate colorimetric method. [6]
- Total calcium, albumin, and creatinine were measured on the Alinity c chemistry module using routine manufacturer-implemented methods on the Abbott platform. [4]

Laboratory reference intervals used for interpretation were those routinely applied in our laboratory during the study period.

### Corrected calcium calculation

**Corrected calcium was calculated using the following albumin-adjusted equation:**

Corrected calcium (mg/L) = measured total calcium (mg/L) + 0.8 × (40 – serum albumin [g/L]). This formula is derived from the historical albumin-adjustment approach described by Payne *et al.*, and is adapted here to the units used in our laboratory. [7] Because albumin-adjusted calcium may differ from ionized calcium and may misclassify calcium status in some clinical settings, results were interpreted with awareness of the recognized limitations of corrected calcium equations, in line with laboratory guidance documents. Calcium status in this study therefore refers to albumin-corrected total calcium rather than ionized calcium. [8]

### eGFR estimation and CKD staging

eGFR was estimated using the CKD-EPI 2021 creatinine equation. [9] CKD stages (G3a–G5) were assigned according to KDIGO eGFR categories. [2]

### Outcomes

**The primary outcomes were:**

1. stage-stratified distributions of iPTH, inorganic phosphate, and corrected calcium.
2. prevalence of mineral abnormalities (e.g., hypocalcemia and hyperphosphatemia) defined using laboratory reference intervals.

Secondary analyses explored correlations between iPTH and eGFR and between iPTH and mineral markers (phosphate and corrected calcium).

### Missing data handling

Core laboratory variables required for eligibility (iPTH, total calcium, albumin, phosphate, creatinine, and eGFR) were available for all included patients (N=150). Analyses were therefore performed on a complete-case

dataset for the variables reported in this manuscript, and no imputation was performed.

### Statistical analysis

Continuous variables were summarized primarily as median [interquartile range] because most biological variables showed non-normal distributions. Comparisons across CKD stages were performed using the Kruskal-Wallis test for continuous variables and the chi-square test or Fisher exact test for categorical variables, as appropriate. Correlations were assessed using Spearman's rank correlation coefficient. Categorical variables were expressed as counts and percentages. Statistical analyses were performed using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA). A two-sided *p* value <0.05 was considered statistically significant.

## RESULTS

### Study population and baseline characteristics

A total of 150 patients with eGFR <60 mL/min/1.73 m<sup>2</sup> were included during the study period (January 2025 to December 2025). The median age was 46.50 years (IQR 33.25–63.75), and there was a slight male predominance (81/150; 54.0%). Kidney function

was markedly impaired overall, with a median eGFR of 12.24 mL/min/1.73 m<sup>2</sup> (IQR 6.26–31.93), reflecting a cohort largely composed of advanced CKD, with G4–G5 accounting for 74.0% of included patients (Table 1).

Mineral metabolism abnormalities were frequent. Median iPTH was 269.00 pg/mL (IQR 159.25–612.25), consistent with a wide range of secondary hyperparathyroidism severity (Table 1). Median corrected calcium was 86.71 mg/L (IQR 82.85–89.63), and median phosphate was 46.00 mg/L (IQR 37.00–58.00). Using the laboratory reference intervals (corrected calcium 85–105 mg/L; phosphate 25–45 mg/L), corrected-calcium-defined hypocalcemia (<85 mg/L) was observed in 58 patients (38.7%), whereas hyperphosphatemia (>45 mg/L) was present in 79 patients (52.7%) (Table 1). Hypercalcemia (>105 mg/L) was uncommon (4/150; 2.7%), and hypophosphatemia (<25 mg/L) was rare (8/150; 5.3%).

Most patients were classified as G5 (84/150; 56.0%), followed by G4 (27/150; 18.0%), G3b (22/150; 14.7%), and G3a (17/150; 11.3%) (Table 1), highlighting the predominance of advanced non-dialysis CKD in the study population.

**Table 1: Baseline characteristics of the study population (N=150).**

Variable	Overall (N=150)
Study period	01/2025–12/2025
Number of patients	150
Age (years), median [IQR]	46.50 [33.25–63.75]
Sex, n (%) — Male	81 (54.0%)
Sex, n (%) — Female	69 (46.0%)
iPTH (pg/mL), median [IQR]	269.00 [159.25–612.25]
Corrected calcium (mg/L), median [IQR]	86.71 [82.85–89.63]
Phosphate (mg/L), median [IQR]	46.00 [37.00–58.00]
Albumin (g/L), median [IQR]	37.00 [30.00–41.00]
Creatinine (mg/L), median [IQR]	49.50 [22.00–90.75]
eGFR CKD-EPI 2021 (mL/min/1.73 m <sup>2</sup> ), median [IQR]	12.24 [6.26–31.93]
CKD stage, n (%) — G3a (45–59)	17 (11.3%)
CKD stage, n (%) — G3b (30–44)	22 (14.7%)
CKD stage, n (%) — G4 (15–29)	27 (18.0%)
CKD stage, n (%) — G5 (<15)	84 (56.0%)
Corrected calcium status (lab RI), n (%) — Hypocalcemia (<85)	58 (38.7%)
Corrected calcium status (lab RI), n (%) — Normal (85–105)	88 (58.7%)
Corrected calcium status (lab RI), n (%) — Hypercalcemia (>105)	4 (2.7%)
Phosphate status (lab RI), n (%) — Hypophosphatemia (<25)	8 (5.3%)
Phosphate status (lab RI), n (%) — Normal (25–45)	63 (42.0%)
Phosphate status (lab RI), n (%) — Hyperphosphatemia (>45)	79 (52.7%)

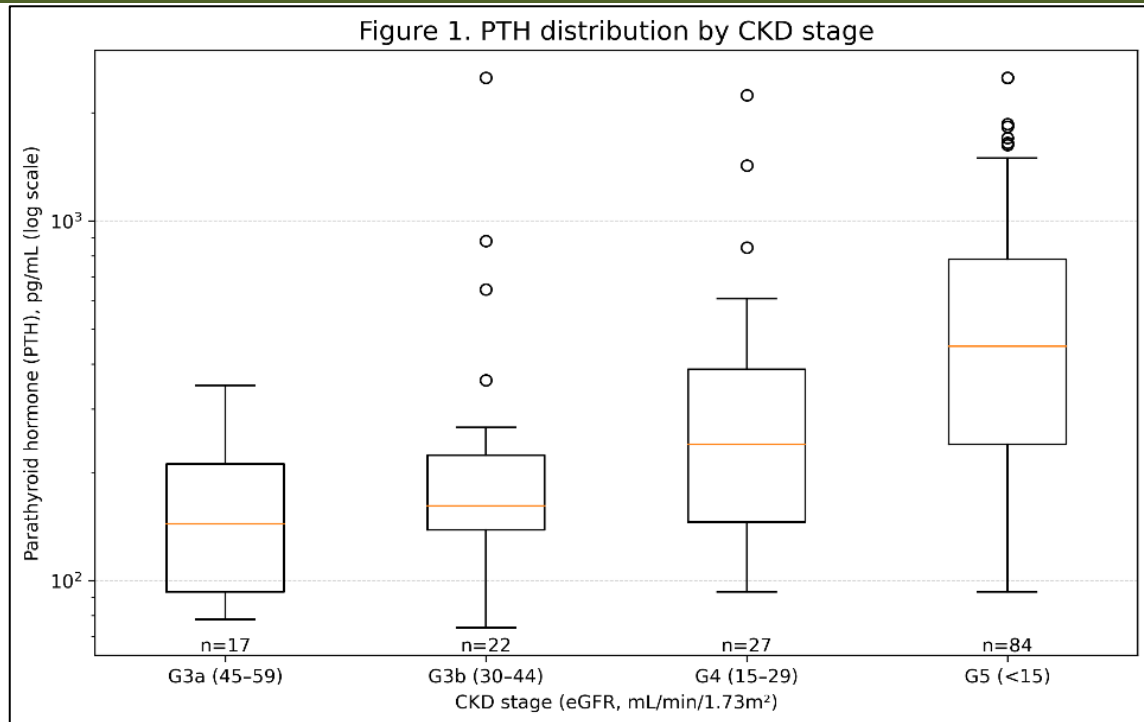
### Biochemical profile across CKD stages

A clear and statistically significant stage-dependent pattern was observed for iPTH, phosphate, and corrected calcium (Table 2; Figures 1–3).

### Parathyroid hormone (iPTH)

iPTH increased substantially with worsening kidney function (overall comparison across stages, *p* <

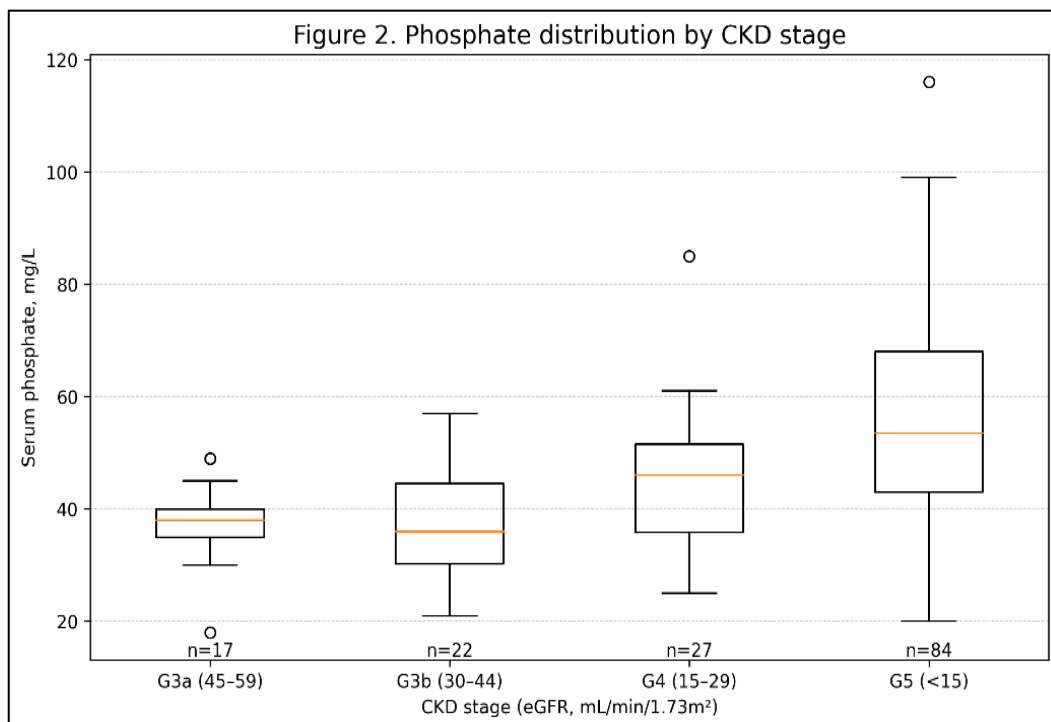
0.0001). Median iPTH rose from 144.00 pg/mL (IQR 93.00–211.00) in G3a to 161.50 pg/mL (IQR 138.25–223.50) in G3b, 239.00 pg/mL (IQR 145.50–386.50) in G4, and 448.50 pg/mL (IQR 239.50–781.50) in G5 (Table 2). The spread of iPTH values was especially wide in G5, as illustrated by the log-scale boxplot (Figure 1).



**Phosphate**

Phosphate levels also differed significantly across CKD stages ( $p < 0.0001$ ) and increased in advanced CKD. Median phosphate was 38.00 mg/L

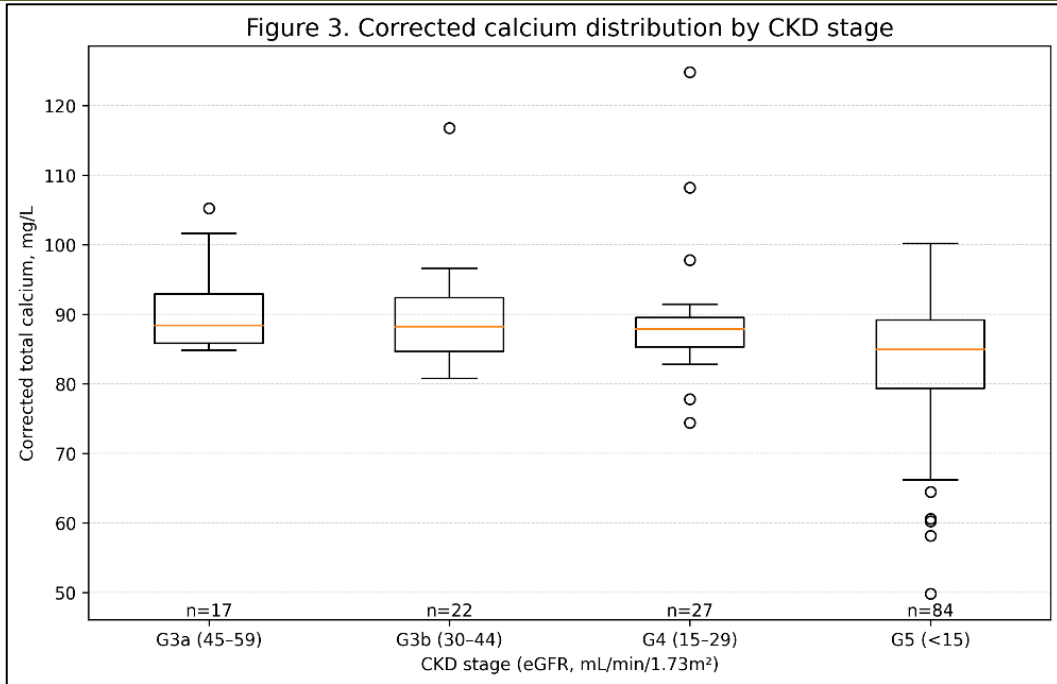
(IQR 35.00–39.91) in G3a and 36.00 mg/L (IQR 30.25–44.50) in G3b, then increased to 46.00 mg/L (IQR 35.84–51.50) in G4 and 53.50 mg/L (IQR 43.00–68.00) in G5 (Table 2; Figure 2).



**Corrected calcium**

Corrected calcium showed a modest but significant decline across stages ( $p = 0.0022$ ). Median corrected calcium remained close to 88 mg/L in G3a–G4

(G3a: 88.41 mg/L, IQR 85.80–92.89; G3b: 88.20 mg/L, IQR 84.65–92.35; G4: 87.83 mg/L, IQR 85.32–89.50) and decreased in G5 (84.92 mg/L, IQR 79.37–89.19) (Table 2; Figure 3).



**Other variables**

Albumin differed across stages ( $p = 0.0351$ ), while sex distribution (male proportion) did not differ significantly by stage ( $p = 0.1025$ ) (Table 2).

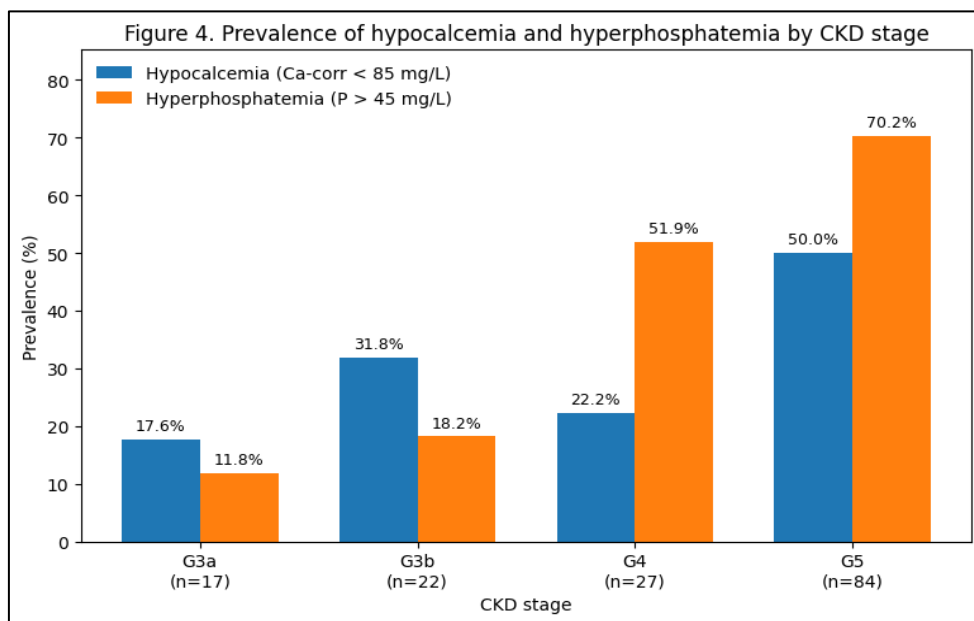
**Stage-specific prevalence of hypocalcemia and hyperphosphatemia**

Hyperphosphatemia ( $>45$  mg/L) showed a strong stage-dependent increase ( $p < 0.0001$ ). The prevalence rose from 11.8% in G3a and 18.2% in G3b to 51.9% in G4 and 70.2% in G5 (Table 2; Figure 4). Hypophosphatemia was uncommon and did not differ across stages ( $p = 0.8443$ ).

**Hypocalcemia**

Corrected-calcium-defined hypocalcemia ( $<85$  mg/L) became more frequent in advanced CKD ( $p = 0.0105$  across stages). The prevalence was 17.6% in G3a, 31.8% in G3b, 22.2% in G4, and reached 50.0% in G5 (Table 2; Figure 4). Hypercalcemia remained infrequent across all stages.

Overall, these findings show that phosphate abnormalities become highly prevalent from stage G4 onward, whereas corrected-calcium-defined hypocalcemia becomes especially frequent in stage G5, consistent with progressive CKD-MBD.



**Table 2: Comparison of demographic and biochemical markers across CKD stages (N=150)**

Variable	G3a (45–59)	G3b (30–44)	G4 (15–29)	G5 (<15)	p-value*
Age (years), median [IQR]	56.00 [48.00–75.00]	54.00 [34.75–69.00]	48.00 [33.50–66.50]	42.00 [32.75–56.25]	0.0960
Sex (Male), n (%)	8 (47.1%)	7 (31.8%)	17 (63.0%)	49 (58.3%)	0.1025
iPTH (pg/mL), median [IQR]	144.00 [93.00–211.00]	161.50 [138.25–223.50]	239.00 [145.50–386.50]	448.50 [239.50–781.50]	<0.0001
Corrected calcium (mg/L), median [IQR]	88.41 [85.80–92.89]	88.20 [84.65–92.35]	87.83 [85.32–89.50]	84.92 [79.37–89.19]	0.0022
Phosphate (mg/L), median [IQR]	38.00 [35.00–39.91]	36.00 [30.25–44.50]	46.00 [35.84–51.50]	53.50 [43.00–68.00]	<0.0001
Albumin (g/L), median [IQR]	36.00 [26.00–42.00]	40.14 [37.00–42.00]	34.00 [24.00–40.50]	36.50 [29.99–41.00]	0.0351
Creatinine (mg/L), median [IQR]	13.15 [12.00–17.00]	17.50 [16.25–20.00]	31.00 [25.50–38.00]	84.00 [61.00–113.50]	<0.0001
eGFR CKD-EPI 2021 (mL/min/1.73 m <sup>2</sup> ), median [IQR]	52.23 [46.85–55.84]	36.56 [35.01–38.33]	20.79 [18.52–25.36]	6.64 [4.39–9.02]	<0.0001
Hypocalcemia (<85 mg/L), n (%)	3 (17.6%)	7 (31.8%)	6 (22.2%)	42 (50.0%)	0.0105
Hypercalcemia (>105 mg/L), n (%)	1 (5.9%)	1 (4.5%)	2 (7.4%)	0 (0.0%)	0.1319
Hyperphosphatemia (>45 mg/L), n (%)	2 (11.8%)	4 (18.2%)	14 (51.9%)	59 (70.2%)	<0.0001
Hypophosphatemia (<25 mg/L), n (%)	1 (5.9%)	2 (9.1%)	1 (3.7%)	4 (4.8%)	0.8443

\*p-values were derived from Kruskal-Wallis tests for continuous variables and chi-square or Fisher exact tests for categorical variables, as appropriate. Abbreviations: CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; IQR, interquartile range; iPTH, intact parathyroid hormone.

### Correlation analysis

Spearman correlation analyses showed biologically plausible associations between kidney function and mineral metabolism markers (Table 3).

iPTH was inversely correlated with eGFR ( $\rho = -0.545$ ,  $p < 0.0001$ ), indicating higher iPTH levels with declining kidney function. iPTH also correlated positively with phosphate ( $\rho = 0.275$ ,  $p = 0.0007$ ) and negatively with corrected calcium ( $\rho = -0.197$ ,  $p = 0.0158$ ). Phosphate was strongly inversely correlated with eGFR ( $\rho = -0.532$ ,  $p < 0.0001$ ), whereas corrected calcium correlated positively with eGFR ( $\rho = 0.308$ ,  $p = 0.0001$ ) (Table 3).

**Table 3: Spearman correlation analysis (N=150).**

Variable pair	n	Spearman's rho	p-value
iPTH vs eGFR	150	-0.545	<0.0001
iPTH vs phosphate	150	0.275	0.0007
iPTH vs corrected calcium	150	-0.197	0.0158
Phosphate vs eGFR	150	-0.532	<0.0001
Corrected calcium vs eGFR	150	0.308	0.0001

Legend: Spearman's rho ( $\rho$ ) is reported with two-sided p-values. Abbreviations: eGFR, estimated glomerular filtration rate; iPTH, intact parathyroid hormone.

## DISCUSSION

### Overview of key findings

In this non-dialysis CKD cohort, CKD-MBD biochemical abnormalities were frequent and clearly stage-dependent. The study population was largely composed of advanced CKD, with stages G4 and G5 accounting for nearly three-quarters of cases. Within this context, iPTH rose progressively across CKD stages,

phosphate abnormalities became increasingly prominent in advanced disease, and corrected-calcium-defined hypocalcemia was especially common in G5. By contrast, hypercalcemia remained uncommon. Overall, these findings fit well within the CKD-MBD framework described in current KDIGO guidance, which recommends integrated interpretation of PTH, calcium, and phosphate across CKD stages. [1,2]

### PTH trajectory across CKD stages and heterogeneity in advanced disease

One of the main findings of this study was the progressive increase in iPTH as kidney function

declined. Median iPTH rose steadily from G3a to G5, with a highly significant stage effect. This pattern is consistent with the expected progression of SHPT in CKD and likely reflects the combined effects of phosphate retention, reduced active vitamin D synthesis, and altered calcium balance. [1,2]

Another notable finding was the wide dispersion of iPTH values in G5. This heterogeneity is biologically plausible, since PTH levels in advanced non-dialysis CKD may be influenced by several factors that were not captured in this dataset, including vitamin D status, nutritional status, inflammation, medication exposure, and treatment adherence. It also supports the KDIGO view that PTH should not be interpreted as a single static threshold in advanced CKD, but rather in its broader biochemical and clinical context. [1]

Observational studies have linked severe CKD-MBD abnormalities, including elevated iPTH, to adverse outcomes, although causal interpretation remains limited and associations may differ between dialysis and non-dialysis populations. [10-15]

#### Comparison with previous studies

Our stage-stratified patterns are broadly consistent with findings from previous non-dialysis CKD cohorts, although absolute prevalences vary according to case mix and stage distribution. In the Spanish MIPTH study, SHPT was already common in CKD stages 3–4 before overt calcium-phosphate abnormalities became prominent, which is in line with our observation that iPTH rises progressively with declining eGFR while phosphate abnormalities become more evident in advanced CKD. [16]

Comparable findings have been reported in predialysis cohorts from Bangladesh and India, where disturbances in mineral metabolism were also more pronounced in advanced CKD. [17,19] Taken together, these studies suggest that PTH abnormalities may appear relatively early, whereas hyperphosphatemia becomes especially prominent as renal impairment progresses.

Beyond descriptive biochemical findings, outcome-oriented studies also support the clinical relevance of SHPT and hyperphosphatemia in non-dialysis CKD. In the NEFRONA cohort, both SHPT and hyperphosphatemia were independently associated with CKD progression and cardiovascular events. [18] More recently, higher iPTH levels were reported as an independent predictor of ischemic heart disease in stage 3–5 non-dialysis CKD. [20] Although our study was cross-sectional and cannot address prognosis directly, these external data reinforce the importance of monitoring PTH and phosphate as kidney function declines.

#### Phosphate abnormalities: a prominent stage-related pattern from G4 onward

Phosphate disturbances emerged as one of the strongest stage-related signals in our study. Median phosphate increased substantially across CKD stages, and hyperphosphatemia rose sharply from early CKD to advanced disease, reaching more than two-thirds of patients in G5. This pattern is clinically relevant because elevated phosphate has been associated with adverse outcomes in both dialysis and non-dialysis CKD, suggesting that phosphate is not only a dialysis-related concern but a broader marker of CKD risk. [10-12,18]

From a practical perspective, the very high prevalence of hyperphosphatemia in G4–G5 supports prioritizing phosphate monitoring and control strategies as kidney function declines, in line with guideline-based integrated management of CKD-MBD. [1]

#### Corrected calcium: modest stage effect but clinically relevant hypocalcemia in G5

Compared with phosphate, corrected calcium showed a more modest stage-related gradient, but the pattern remained clinically meaningful. Median corrected calcium decreased significantly across stages, with the lowest values observed in G5. Corrected-calcium-defined hypocalcemia affected half of patients in G5, whereas hypercalcemia remained uncommon across all stages.

In advanced CKD, lower calcium values may reflect reduced calcitriol activity, phosphate retention, and treatment-related influences such as phosphate binder choice, vitamin D therapy, or calcimimetic exposure. As emphasized by KDIGO, calcium should be interpreted alongside phosphate and PTH, with attention to serial patterns rather than isolated measurements. [1]

An important methodological point is that this study relied on albumin-adjusted (corrected) calcium, derived from the Payne approach adapted to the units used in our laboratory. [7] While corrected calcium remains widely used in routine laboratory practice, professional guidance has highlighted that corrected calcium equations may misclassify calcium status when compared with ionized calcium in some clinical settings. [8] Accordingly, the hypocalcemia reported here should be interpreted as corrected-calcium-defined hypocalcemia rather than confirmed ionized hypocalcemia. [8]

#### Correlation structure supports coherent CKD-MBD clustering

The correlation analyses were biologically coherent and support the interpretation that worsening kidney function is accompanied by clustering of CKD-MBD abnormalities. iPTH correlated inversely with eGFR, phosphate also correlated inversely with eGFR, and corrected calcium correlated positively with eGFR.

In addition, iPTH correlated positively with phosphate and negatively with corrected calcium.

These findings are consistent with the expected biological model in CKD-MBD: declining kidney function favors phosphate retention, phosphate burden rises as renal excretory capacity worsens, calcium balance becomes increasingly disturbed, and PTH increases as part of the compensatory SHPT response. [1,2]

### Strengths and limitations

This study has several strengths. It provides stage-stratified data across CKD stages G3a–G5, demonstrates coherent biomarker gradients, and shows a correlation structure that supports internal biological consistency. It also contributes local laboratory-based data from a setting where CKD-MBD monitoring patterns are not widely reported.

However, several limitations should be acknowledged. This was a single-center hospital-based study, and the cohort was predominantly composed of advanced CKD, particularly G5, which limits generalizability to earlier CKD stages. Because inclusion required concurrent iPTH testing, the study population may overrepresent patients in whom CKD-MBD was already clinically suspected, introducing selection bias. Treatment data were not available, including phosphate binders, vitamin D therapy, and calcimimetics. In addition, vitamin D status, alkaline phosphatase, FGF23, and ionized calcium were not systematically available. Finally, the cross-sectional design provides only a biochemical snapshot and does not allow assessment of longitudinal trajectories, treatment response, or clinical outcomes. [8]

### CONCLUSION

In this hospital-based non-dialysis CKD cohort with eGFR <60 mL/min/1.73 m<sup>2</sup>, CKD-MBD biochemical abnormalities were frequent and showed clear stage-related gradients. Hyperphosphatemia was especially common from stage G4 onward, while corrected-calcium-defined hypocalcemia was most frequent in G5. These findings support stage-stratified monitoring and integrated interpretation of PTH, phosphate, and calcium in routine practice.

**Data availability:** The dataset is available from the corresponding author upon reasonable request, subject to institutional approval.

**Funding:** The authors received no specific funding for this work.

**Conflicts of interest:** The authors declare no competing interests.

**Ethics approval and consent:** This retrospective laboratory-based study used de-identified data obtained from routine care and was conducted with the agreement of the head of the Clinical Biochemistry Laboratory of Arrazi Hospital, CHU Mohammed VI, Marrakech, Morocco. Because the study involved retrospective analysis of fully de-identified laboratory data, with no direct patient contact or intervention, informed consent was waived in accordance with local institutional policy.

### REFERENCES

1. Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD). *Kidney Int Suppl* [2011]. 2017;7[1]:1–59. doi: 10.1016/j.kisu.2017.04.001.
2. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int Suppl*. 2013;3[1]:1–150. doi:10.1038/kisup.2012.76.
3. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet*. 2007;370[9596]:1453–1457. doi:10.1016/S0140-6736[07]61602-X.
4. Abbott. Alinity ci-series (integrated clinical chemistry and immunoassay) [Internet]. Abbott Core Laboratory. Available from: <https://www.corelaboratory.abbott/us/en/offerings/brands/alinity/alinity-ci-series.html>. Accessed February 14, 2026.
5. U.S. Food and Drug Administration. Establishment Registration & Device Listing: Alinity i Intact PTH Reagent Kit (product code CEW) [Internet]. Available from: <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfRL/rl.cfm?lid=626194&lpcd=CEW>. Accessed February 14, 2026.
6. U.S. Food and Drug Administration. Establishment Registration & Device Listing: Alinity c Phosphorus Reagent Kit (product code CEO) [Internet]. Available from: <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfRL/rl.cfm?lid=626143&lpcd=CEO>. Accessed February 14, 2026.
7. Payne RB, Little AJ, Williams RB, Milner JR. Interpretation of serum calcium in patients with abnormal serum proteins. *Br Med J*. 1973;4[5893]:643–646. doi:10.1136/bmj.4.5893.643.
8. O’Kane MJ, *et al.*, Albumin-adjusted calcium: a position statement [Internet]. Association for Clinical Biochemistry and Laboratory Medicine;

2015. Available from: <https://www.acb.org.uk/static/4ead26cf-8e26-47f1-854a894820bd3348/adjusted-calcium-position-paper-march-2015.pdf>. Accessed February 14, 2026.
9. Inker LA, Eneanya ND, Coresh J, *et al.*, New creatinine- and cystatin C-based equations to estimate GFR without race. *N Engl J Med.* 2021;385[19]:1737–1749. doi:10.1056/NEJMoa2102953.
  10. Block GA, Hulbert-Shearon TE, Levin NW, Port FK. Association of serum phosphorus and calcium × phosphate product with mortality risk in chronic hemodialysis patients: a national study. *Am J Kidney Dis.* 1998;31[4]:607–617. doi:10.1053/ajkd.1998.v31.pm9531176.
  11. Ganesh SK, Stack AG, Levin NW, Hulbert-Shearon T, Port FK. Association of elevated serum PO<sub>4</sub>, Ca × PO<sub>4</sub> product, and parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. *J Am Soc Nephrol.* 2001;12[10]:2131–2138. doi:10.1681/ASN.V12102131.
  12. Kestenbaum B, Sampson JN, Rudser KD, *et al.*, Serum phosphate levels and mortality risk among people with chronic kidney disease. *J Am Soc Nephrol.* 2005;16[2]:520–528. doi:10.1681/ASN.2004070602.
  13. Floege J, Kim J, Ireland E, *et al.*, Serum iPTH, calcium and phosphate, and the risk of mortality in a European haemodialysis population. *Nephrol Dial Transplant.* 2011;26[6]:1948–1955. doi:10.1093/ndt/gfq219.
  14. Kovesdy CP, Ahmadzadeh S, Anderson JE, Kalantar-Zadeh K. Secondary hyperparathyroidism is associated with higher mortality in men with moderate to severe chronic kidney disease. *Kidney Int.* 2008;73[11]:1296–1302. doi:10.1038/ki.2008.64.
  15. Chertow GM, Block GA, Correa-Rotter R, *et al.*, EVOLVE Trial Investigators. Effect of cinacalcet on cardiovascular disease in patients undergoing dialysis. *N Engl J Med.* 2012;367[26]:2482–2494. doi:10.1056/NEJMoa1205624.
  16. Bureo JC, Arévalo JC, Antón J, Adrados G, Jiménez Morales JL, Robles NR; en representación de los participantes en el estudio MIPTH. Prevalence of secondary hyperparathyroidism in patients with stage 3 and 4 chronic kidney disease seen in internal medicine. *Endocrinol Nutr.* 2015 Aug-Sep;62[7]:300–305. doi:10.1016/j.endonu.2015.05.006.
  17. Hai AA, Rahman MM, Anwar MR, Miah OF, Latif A, Jalil ME, Razzak MAR, Morshed AMM, Mahmud H, Dowel FA. Status of serum calcium, phosphate and intact parathyroid hormone in predialysis chronic kidney disease patients of stage-3 to stage-5 compared to KDOQI guideline. *Mymensingh Med J.* 2021 Oct;30[4]:1031–1042.
  18. Bozic M, Diaz-Tocados JM, Bermudez-Lopez M, Forné C, Martinez C, Fernandez E, Valdivielso JM. Independent effects of secondary hyperparathyroidism and hyperphosphataemia on chronic kidney disease progression and cardiovascular events: an analysis from the NEFRONA cohort. *Nephrol Dial Transplant.* 2022 Mar 25;37[4]:663–672. doi:10.1093/ndt/gfab184.
  19. Vikrant S, Parashar A. Prevalence and severity of disordered mineral metabolism in patients with chronic kidney disease: a study from a tertiary care hospital in India. *Indian J Endocrinol Metab.* 2016;20[4]:460–467. doi:10.4103/2230-8210.183457.
  20. Anumas S, Tantiyavarong P, Pattharanitima P. Parathyroid hormone levels as an independent predictor of ischemic heart disease in stage 3–5 non-dialysis chronic kidney disease: a retrospective cohort study. *J Clin Med.* 2025;14[10]:3311. doi:10.3390/jcm14103311.