Original Article www.pjkd.com.pk

The Burden of CKD–Mineral and Bone Disorder in Maintenance Hemodialysis: Insights from a Longitudinal Registry

Omer Sabir[1], Muhammad Bilal Basit[1], Noor ul Ain[2], Ateka Mansoor Ahmed[1], Muhammad Ali Nouman[1]

[1]Department of Nephrology, Fatima Memorial Hospital, Lahore, Pakistan
[2]Department of Medicine, Fatima Memorial Hospital, Lahore, Pakistan

Abstract

Background

Chronic kidney disease – mineral and bone disorder (CKD – MBD) is a frequent complication in patients receiving maintenance hemodialysis and is associated with adverse skeletal and cardiovascular outcomes. Real-world longitudinal data describing biochemical burden and guideline target attainment remain limited.

Methods

We conducted a retrospective, registry-based descriptive study of adult maintenance hemodialysis patients between January 2021 and September 2025. Laboratory measurements of calcium, phosphate, calcium – phosphate product, and intact parathyroid hormone (iPTH) were extracted from monthly registry tables. Test-level analyses included all available measurements. For patient-level categorization, the most recent available value per patient per analyte was used. Parameters were classified according to KDIGO guideline-based targets, with iPTH interpreted using an upper limit of normal (ULN) of 65 pg/mL.

Results

A total of 1,042 calcium, 1,037 phosphate, 966 calcium – phosphate product, and 141 iPTH measurements were analysed. Mean serum calcium was 8.38 ± 1.02 mg/dL, mean phosphate 5.65 ± 2.17 mg/dL, mean calcium – phosphate product 47.2 ± 18.2 mg^2/dL^2, and mean iPTH 593 ± 518 pg/mL, indicating substantial biochemical variability.

At the patient level, calcium was within target in 59.3% of patients, while phosphate was above target in 46.3%. An elevated calcium – phosphate product (>55 mg^2/dL^2) was observed in 27.0%. Among patients with iPTH measurements, 43.2% were within the KDIGO-recommended range (2 – 9×ULN), 36.5% were above target, and 20.3% were below target.

Conclusion

Maintenance hemodialysis patients in this registry demonstrated a persistent burden of CKD – MBD abnormalities with suboptimal attainment of KDIGO targets, particularly for phosphate and iPTH, highlighting the need for improved monitoring and guideline-aligned management.

Keywords: CKD, mineral and bone disorder; Hemodialysis; Calcium; Phosphate; Intact parathyroid hormone; KDIGO; Secondary hyperparathyroidism

Corresponding Author
Dr Bilal Basit,
Department of Nephrology, Fatima Memorial hospital, Lahore, Pakistan.
Email: dr.mbb842@gmail.com
DOI: 10.53778/pjkd101344
Received 5th March 2026 and accepted 30th March 2026 PJKD 2026;10(1):37-43

Introduction:

Chronic kidney disease – mineral and bone disorder (CKD – MBD) is an umbrella term encompassing a spectrum of biochemical abnormalities involving calcium, phosphate, vitamin D metabolism,

fibroblast growth factor-23 (FGF-23), and intact parathyroid hormone (iPTH) that occur as kidney function declines.[1] These disturbances typically begin to emerge during CKD stage 3 and, if left untreated, progress in severity through advanced CKD and dialysis-dependent CKD (CKD 5D).[1]

The earliest detectable abnormalities of CKD – MBD are often alterations in iPTH and calcitriol levels, which may become evident when estimated glomerular filtration rate (eGFR) falls below 60 mL/min/1.73 m^2.[2] In contrast, the more characteristic derangements in serum calcium and phosphate generally become apparent at more advanced stages, particularly when eGFR declines below 30 mL/min/1.73 m^2.[2]

Secondary hyperparathyroidism represents a central manifestation of CKD – MBD and is characterised by a recognizable biochemical profile of hyperphosphatemia, hypocalcaemia, and elevated iPTH levels.[1] These abnormalities contribute to disordered bone metabolism, increasing the risk of fractures, and are also implicated in adverse cardiovascular outcomes, including accelerated atherosclerosis and vascular calcification, leading to ischemic complications across multiple organ systems.[3,4] At the same time, therapeutic interventions for CKD – MBD—such as calcimimetics and low-calcium dialysate—are not without risk and may predispose patients to QT interval prolongation and cardiac arrhythmias.[5]

Given these competing risks, careful monitoring and tight control of calcium – phosphate metabolism and iPTH levels within recommended ranges are essential in patients with advanced CKD and those receiving maintenance hemodialysis.[1] The widespread availability of intact PTH assays has facilitated routine biochemical monitoring in clinical practice. Reflecting this, the 2017 KDIGO update on CKD – MBD placed greater emphasis on the use of iPTH measurements for categorising disease severity and provided stage-specific target ranges, including recommendations for patients with CKD 5D.[1]

Several studies have attempted to describe the burden of CKD – MBD among local dialysis populations. However, many of these reports rely on limited or cross-sectional data, which may not adequately capture the dynamic nature of mineral metabolism. Given that calcium, phosphate, and iPTH levels fluctuate over time, longitudinal data collected over extended periods may provide a more accurate and clinically meaningful representation of CKD – MBD patterns in routine dialysis practice.[6]

Methodology

Study design and setting:

This was a retrospective, descriptive study conducted using a hemodialysis registry maintained by the primary author at our center. The registry has been prospectively maintained for approximately five years and contains routinely collected clinical and laboratory data for patients receiving maintenance hemodialysis as part of standard care.

Study population:

All adult patients receiving or having received maintenance hemodialysis at our center who had at least one recorded mineral metabolism parameter during the study period were eligible for inclusion. No

exclusions were made based on dialysis vintage, frequency of dialysis, or comorbid conditions, in order to reflect real-world practice.

Study period and extracted variables:

Laboratory data recorded between January 2021 and September 2025 were included in the analysis. Although the registry spans a longer duration, only data within this predefined study window were extracted for the present study.

We extracted the following markers of mineral and bone metabolism from the registry: serum calcium, albumin-corrected calcium, serum phosphate, calcium – phosphate product, albumin-corrected calcium – phosphate product, and intact parathyroid hormone (iPTH).

When available, albumin-corrected calcium values were preferentially used; if unavailable, corresponding uncorrected calcium values were analyzed. Similarly, albumin-corrected calcium – phosphate product values were used when available; otherwise, uncorrected or calculated values were included. Intact parathyroid hormone measurements were interpreted using a laboratory upper limit of normal (ULN) of 65 pg/mL.

Data completeness varied across parameters, with more frequent measurements available for calcium and phosphate compared to iPTH, reflecting routine clinical practice. No imputation was performed for missing data, and analyses were restricted to available observations for each parameter. Accordingly, denominators differ across biochemical variables and are explicitly reported.

All available laboratory measurements were retained for test-level descriptive analyses. To address potential bias arising from repeated measurements, a separate patient-level dataset was constructed in which, for each parameter, the most recent available value per patient during the study period was used. This approach maximized data capture while minimizing bias due to unequal testing frequency and avoiding over-representation of patients undergoing more frequent laboratory monitoring.

Ethical Approval:

Ethical approval was obtained from the Institutional Review Board (IRB)FMH-16/01/2026-IRB-1856, Dated 27 Januray, 2026. Due to the retrospective nature of the study, informed consent was waived.

Definitions and categorization:

Biochemical parameters were categorized according to KDIGO guideline – based targets:

Calcium: below target (<8.4 mg/dL), within target (8.4 – 10.2 mg/dL), above target (>10.2 mg/dL)

Phosphate: below target (<3.5 mg/dL), within target (3.5 – 5.5 mg/dL), above target (>5.5 mg/dL)

Calcium – phosphate product: ≤55 or >55 mg^2/dL^2

Intact PTH: below target (<2×ULN), within target (2 – 9×ULN), above target (>9×ULN)

Statistical analysis:

Analyses were conducted using R statistical software. Data were analyzed descriptively. Continuous variables are presented as mean ± standard deviation and median with interquartile range (IQR), given the expected non-normal and right-skewed distribution of biochemical parameters, particularly iPTH. Categorical variables are presented as frequencies and percentages.

Due to the descriptive nature of the study, no inferential statistical testing was performed.

Results:

During the study period from January 2021 to September 2025, laboratory data from patients receiving or having received maintenance hemodialysis at FMH were analysed. A total of 1,042 calcium, 1,037 phosphate, 966 calcium – phosphate product, and 141 intact parathyroid hormone (iPTH) measurements were available for analysis, Table 1. Albumin-corrected calcium values were preferentially used when available; otherwise, uncorrected calcium values were analysed.

Table 1: Baseline parameters of maintenance hemodialysis patients

Parameter	Measurements (n)	Patients (n)
Calcium	1,042	177
Phosphate	1,037	174
Ca–PO4 Product	966	174
iPTH	141	74

The mean serum calcium concentration was 8.38 ± 1.02 mg/dL, with a median of 8.52 mg/dL (IQR 1.0) and a range of 2.8 – 12.8 mg/dL. Mean serum phosphate was 5.65 ± 2.17 mg/dL, with a median of 5.3 mg/dL (IQR 2.7) and a range of 1.4 – 14.2 mg/dL. The mean calcium – phosphate product was 47.2 ± 18.2 mg^2/dL^2, with a median of 45.1 mg^2/dL^2 (IQR 24.0) and values extending up to >110 mg^2/dL^2. Intact parathyroid hormone levels demonstrated substantial variability, with a mean value of 593 ± 518 pg/mL and a median of 463 pg/mL (IQR 505), reflecting marked heterogeneity in parathyroid hormone control across measurements (Table 2).

Table 2: Baseline pararmeters with median, mean and range for maintenance hemodialysis patients.

Parameter	Mean ± SD	Median (IQR)	Range	Key Note
Calcium (mg/dL)	8.38 ± 1.02	8.52 (1.0)	2.8–12.8	Mostly normal-low
Phosphate (mg/dL)	5.65 ± 2.17	5.3 (2.7)	1.4–14.2	Tendency to high
Ca–PO4 (mg^2/dL^2)	47.2 ± 18.2	45.1 (24.0)	>110	Some markedly high
iPTH (pg/mL)	593 ± 518	463 (505)	-	Wide variability

For patient-level analysis, the most recent available laboratory value for each parameter was used. Calcium values were available for 177 patients, phosphate for 174 patients, calcium – phosphate product for 174 patients, and iPTH for 74 patients.

Using KDIGO-recommended targets, serum calcium was within target range in 59.3% (105/177) of patients, below target in 37.9% (67/177), and above target in 2.8% (5/177). Serum phosphate was within target in 42.4% (75/177) of patients, while 46.3% (82/177) had values above target and 11.3% (20/177)

were below target. An elevated calcium – phosphate product (>55 mg^2/dL^2) was present in 27.0% (47/174) of patients, whereas 73.0% (127/174) had values ≤55 mg^2/dL^2. Among patients with available iPTH measurements, 43.2% (32/74) were within the KDIGO-recommended range of 2 – 9 times the upper limit of normal (ULN), 36.5% (27/74) had values above target (>9×ULN), and 20.3% (15/74) had values below target (<2×ULN) (Table 3).

Table 3: Laboratory values related to mineral bone disease among maintenance hemodialysis patients that were within the target range.

Parameter	Within Target	Above Target	Below Target
Calcium	105 (59.3%)	5 (2.8%)	67 (37.9%)
Phosphate	75 (42.4%)	82 (46.3%)	20 (11.3%)
Ca X PO4	127 (73.0%)	47 (27.0%)	-
iPTH	32 (43.2%)	27 (36.5%)	15 (20.3%)

Discussion:

Disturbances in the calcium – phosphate – parathyroid hormone axis remain a central challenge in the management of chronic kidney disease, particularly in patients receiving maintenance hemodialysis. These abnormalities, broadly categorized into high bone turnover and low bone turnover states, exert substantial downstream effects on skeletal integrity and cardiovascular health.[7,8] Effective control of mineral metabolism is therefore a cornerstone of long-term CKD care.[7]

Despite the clinical importance of CKD – MBD, systematic and longitudinal characterization of mineral metabolism patterns in routine dialysis practice—particularly in resource-limited settings—remains limited.[9] In this context, our registry-based analysis provides a real-world picture of the burden an extended time period.

At the full cohort level, our findings demonstrate a substantial burden of hyperphosphatemia and marked variability in intact parathyroid hormone (iPTH) levels, reflecting the dynamic and often unstable nature of mineral metabolism in hemodialysis patients.[9] The wide dispersion of iPTH values underscores the biological heterogeneity of secondary hyperparathyroidism and highlights the complexity of achieving sustained biochemical control in this population.[10]

When assessed at the patient level using the most recent available measurements, only approximately half of patients achieved KDIGO-recommended targets for iPTH, with a predominance of elevated iPTH values, consistent with a high bone turnover phenotype.[7] Low bone turnover states were observed less frequently. This pattern aligns with the typical trajectory of secondary hyperparathyroidism in advanced CKD, where chronic phosphate retention, impaired vitamin D activation, and prolonged parathyroid stimulation drive progressive hormonal dysregulation. [8]

Phosphate control also emerged as a major area of concern, with nearly half of patients demonstrating above-target serum phosphate levels. Given the established links between hyperphosphatemia, vascular calcification, and adverse cardiovascular outcomes, this finding carries important clinical implications.[11] Similarly, more than one-quarter of patients exhibited an elevated calcium – phosphate product, suggesting ongoing risk for extra skeletal calcification. [12]

In contrast, serum calcium levels were within target range in the majority of patients, indicating comparatively better control of calcium homeostasis. However, this apparent stability must be interpreted cautiously, as aggressive therapeutic strategies—including calcium-based phosphate binders, calcimimetics, and dialysate calcium manipulation—may influence serum calcium levels while masking ongoing disturbances in phosphate balance and parathyroid activity.[13]

Taken together, our findings suggest that current CKD – MBD management in routine dialysis practice may fall short of achieving recommended guideline targets, particularly with respect to phosphate and iPTH control. This underscores the need for more structured treatment algorithms, improved patient adherence to phosphate-lowering strategies, and regular biochemical surveillance. It also highlights the potential value of individualized treatment approaches that account for evolving turnover states over time.

Our study benefits from the use of longitudinal registry data spanning multiple years, allowing us to capture temporal variability and maximize data completeness. By combining test-level and patient-level analyses, we provide complementary insights into both the overall biochemical burden and clinically interpretable target attainment. Nevertheless, several limitations merit acknowledgment. The retrospective nature of the analysis and reliance on routinely collected laboratory data introduce the possibility of missing or irregular measurements. Additionally, iPTH testing was performed less frequently than calcium or phosphate measurement, which may limit the generalizability of parathyroid hormone – related findings. Finally, the descriptive design precludes causal inference regarding treatment effects or clinical outcomes.

Conclusion:

In conclusion, this registry-based analysis reveals a persistent burden of CKD – MBD biochemical abnormalities in maintenance hemodialysis patients, with suboptimal attainment of KDIGO targets, particularly for phosphate and iPTH. These findings reinforce the importance of sustained monitoring, guideline-informed therapy, and locally adapted quality improvement strategies aimed at optimizing mineral metabolism and mitigating long-term skeletal and cardiovascular risk.

Conflict of Interest: None
Funding Source: None

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. 2017;7(1):1-59.

2. Moe S, Drüeke T, Cunningham J, Goodman W, Martin K, Olgaard K, et al. Definition, evaluation, and classification of renal osteodystrophy: a position statement from KDIGO. Kidney Int. 2006;69(11):1945-53.
3. Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. J Am Soc Nephrol. 2004;15(8):2208-18.
4. London GM, Guérin AP, Marchais SJ, Métivier F, Pannier B, Adda H. Arterial media calcification in end-stage renal disease: impact on cardiovascular function and mortality. Nephrol Dial Transplant. 2003;18(9):1731-40.
5. Charytan DM, Patrick AR, Liu J, Setoguchi S, Brookhart MA, Winkelmayer WC. Cinacalcet use and cardiovascular events among hemodialysis patients. Circulation. 2012;126(23):2685-94.
6. Tentori F, Blayney MJ, Albert JM, Gillespie BW, Kerr PG, Bommer J, et al. Mortality risk for dialysis patients with different levels of serum calcium, phosphorus, and PTH. Am J Kidney Dis. 2008;52(3):519-30
7. Kidney Disease: Improving Global Outcomes (KDIGO) CKD-MBD Update Work Group. KDIGO 2017 clinical practice guideline update for diagnosis, evaluation, prevention, and treatment of CKD-MBD. Kidney Int Suppl. 2017;7(1):1-59.
8. Cunningham J, Locatelli F, Rodriguez M. Secondary hyperparathyroidism: pathogenesis, disease progression, and therapeutic options. Clin J Am Soc Nephrol. 2011;6(4):913-21.
9. Floege J, Kim J, Ireland E, Chazot C, Drueke T, de Francisco A, et al. Serum iPTH, calcium and phosphate and mortality risk in dialysis patients: results from the DOPPS. Nephrol Dial Transplant. 2011;26(6):1948-55.
10. Tentori F, Wang M, Bieber BA, Karaboyas A, Li Y, Jacobson SH, et al. Recent changes in therapeutic approaches and association with outcomes among dialysis patients with secondary hyperparathyroidism. Clin J Am Soc Nephrol. 2015;10(1):98-109.
11. Block GA, Hulbert-Shearon TE, Levin NW, Port FK. Association of serum phosphorus and calcium-phosphate product with mortality risk in chronic hemodialysis patients. Am J Kidney Dis. 1998;31(4):607-17.
12. Goodman WG, London G, Amann K, Block GA, Giachelli C, Hruska KA, et al. Vascular calcification in CKD. Am J Kidney Dis. 2004;43(3):572-79.
13. Chertow GM, Block GA, Correa-Rotter R, Drueke TB, Floege J, Goodman WG, et al. Effect of cinacalcet on cardiovascular disease in patients undergoing dialysis. N Engl J Med. 2012;367(26):2482-94.