

**BONE MINERAL METABOLISM AND PARATHYROID HORMONE LEVELS IN CHRONIC KIDNEY DISEASE PATIENTS ON HEMODIALYSIS: A CROSS-SECTIONAL EVALUATION WITH BIOCHEMICAL PARAMETERS**

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ABSTRACT **Background:** Chronic kidney disease (CKD) is frequently complicated by disturbances in bone and mineral metabolism, collectively termed CKD-mineral and bone disorder (CKD-MBD). Declining renal function leads to phosphate retention, reduced vitamin D activation and hypocalcemia, driving persistent parathyroid hormone (PTH) secretion and secondary hyperparathyroidism. Patients on maintenance hemodialysis are particularly vulnerable to these derangements. This study was undertaken to assess bone mineral metabolism and intact parathyroid hormone (iPTH) levels in CKD patients on hemodialysis and to correlate them with biochemical parameters and disease stage. **Methods:** This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, JMF's ACPM Medical College, Dhule, over a period of two years. Ninety-seven patients with CKD undergoing maintenance hemodialysis who fulfilled the inclusion criteria were enrolled after informed consent. Venous blood samples were collected before a scheduled dialysis session and analyzed for hemoglobin, urea, creatinine, serum calcium, serum phosphorus and iPTH; the calcium-phosphorus ($\text{Ca} \times \text{P}$) product was calculated. Patients were staged by estimated glomerular filtration rate (eGFR). Data were analyzed using SPSS version 20, with ANOVA for continuous variables and chi-square for categorical variables; $p < 0.05$ was considered significant. **Results:** The mean age of participants was 50.78 ± 14.88 years, with a male predominance (62.89%). The majority (62.89%) had Stage 5 CKD. Mean hemoglobin was 8.12 ± 1.93 g/dL, serum calcium 7.91 ± 1.14 mg/dL, serum phosphorus 6.88 ± 1.67 mg/dL, $\text{Ca} \times \text{P}$ product 54.62 ± 12.48 mg^2/dL^2 , and iPTH 286.74 ± 118.36 pg/mL. Hypocalcemia was present in 64.95% and hyperphosphatemia in 75.26% of patients; 45.36% had iPTH levels exceeding 488 pg/mL. Serum calcium showed a significant inverse association with iPTH ($p = 0.009$), while serum phosphorus and the $\text{Ca} \times \text{P}$ product showed significant positive associations with iPTH ($p = 0.004$ and $p = 0.01$, respectively). Mean serum calcium declined and mean serum phosphorus, $\text{Ca} \times \text{P}$ product and iPTH rose progressively across advancing CKD stages (all $p < 0.0001$). **Conclusion:** CKD-MBD, characterized by hypocalcemia, hyperphosphatemia and secondary hyperparathyroidism, is highly prevalent among patients on maintenance hemodialysis and worsens progressively with advancing CKD stage. Routine monitoring of calcium, phosphorus and iPTH is essential for early diagnosis and timely therapeutic intervention to reduce the risk of renal osteodystrophy, vascular calcification and cardiovascular complications.

KEYWORDS :**INTRODUCTION**

Chronic kidney disease (CKD) is a major global health problem, with a substantial and rising worldwide burden of morbidity and mortality (Hill et al., 2016). It is defined as persistent abnormalities of kidney structure or function, including a glomerular filtration rate below 60 mL/min, for at least three months; substantial renal damage may precede overt biochemical abnormalities, underscoring the importance of early detection (National Kidney Foundation K/DOQI, 2002).

As renal function deteriorates, impaired phosphate excretion, reduced vitamin D activation and consequent hypocalcemia drive persistent parathyroid hormone (PTH) secretion, causing secondary hyperparathyroidism. These derangements — collectively termed CKD-mineral and bone disorder (CKD-MBD) — contribute to renal osteodystrophy, vascular calcification and cardiovascular morbidity, and are incompletely corrected by dialysis alone (KDIGO CKD-MBD Work Group, 2009; Moe et al., 2007). Evaluation of intact PTH (iPTH) alongside serum calcium, phosphorus and the calcium-phosphorus ($\text{Ca} \times \text{P}$) product is therefore essential to gauge CKD-MBD severity and guide treatment (Kritmetapak and Pongchaiyakul, 2019). This study assessed bone mineral metabolism and iPTH levels in CKD patients on hemodialysis and their association with biochemical parameters and disease stage.

MATERIALS AND METHODS**Design, Setting and Sample Size:**

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, JMF's ACPM Medical College, Dhule, over two years. Sample size was calculated as $n = Z^2 P(1-P)/d^2$ ($Z = 1.96$ for 95% confidence, expected prevalence $P = 17\%$, margin of error $d = 7.5\%$), yielding a minimum of 97 patients, all of whom were enrolled after informed consent.

Inclusion Criteria:

- Adults aged >18 and <85 years with CKD undergoing maintenance hemodialysis, willing to give informed consent.

Exclusion Criteria:

- Acute kidney injury on CKD; genitourinary malignancy; other primary kidney disease; pre-existing bone mineral metabolism disorder.

Methodology and Statistical Analysis:

With Institutional Ethics Committee approval, venous blood was collected before a scheduled dialysis session and analyzed for serum calcium, phosphorus and iPTH; the $\text{Ca} \times \text{P}$ product was calculated (calcium $\times$ phosphorus, mg/dL). Reference ranges: calcium 8.5–10.5 mg/dL, phosphorus 2.5–4.5 mg/dL, iPTH 10–65 pg/mL, $\text{Ca} \times \text{P}$ product <55 mg^2/dL^2 . CKD stage was assigned by eGFR (KDIGO criteria). Data were analyzed in SPSS v20; continuous variables (mean $\pm$ SD) were compared by one-way ANOVA and categorical variables by chi-square test; $p < 0.05$ was significant.

RESULTS

Ninety-seven CKD patients on maintenance hemodialysis were studied (mean age 50.78 ± 14.88 years; 62.89% male, 37.11% female). Most patients (43.30%) had been on hemodialysis for 2–5 years and 38.14% for under 2 years.

Table 1: Age-wise Distribution of Study Participants (n = 97)

Age Group (years)	No. of Cases	Percentage
18–30	10	10.31%
31–45	24	24.74%
46–60	38	39.18%
61–75	20	20.62%
76–84	5	5.15%
Total	97	100.00%

Table 2: Mean Biochemical Parameters among Study Participants

Parameter	Mean $\pm$ SD
Hemoglobin (g/dL)	8.12 $\pm$ 1.93
Urea (mg/dL)	198.36 $\pm$ 79.54
Creatinine (mg/dL)	11.47 $\pm$ 5.23
Serum Calcium (mg/dL)	7.91 $\pm$ 1.14
Serum Phosphorus (mg/dL)	6.88 $\pm$ 1.67
Ca $\times$ P Product (mg ² /dL ²)	54.62 $\pm$ 12.48
iPTH (pg/mL)	286.74 $\pm$ 118.36

The profile confirmed anemia (Hb 8.12 $\pm$ 1.93 g/dL) and severe renal impairment (urea 198.36 $\pm$ 79.54; creatinine 11.47 $\pm$ 5.23 mg/dL). Hypocalcemia (<8.4 mg/dL) was present in 64.95% and hyperphosphatemia in 75.26% of patients; an elevated Ca $\times$ P product ($\geq$ 55 mg²/dL²) was seen in 46.39%.

Table 3: Distribution of Patients According to Serum iPTH Levels

iPTH (pg/mL)	No. of Cases	Percentage
<122	5	5.15%
122–488	48	49.48%
>488	44	45.36%
Total	97	100.00%

Table 4: Association of Serum Calcium, Phosphorus and Ca $\times$ P Product with iPTH Category

iPTH Category	Mean Calcium (mg/dL)	Mean Phosphorus (mg/dL)	Mean Ca $\times$ P (mg ² /dL ²)
<122 pg/mL	8.42 $\pm$ 0.98	5.92 $\pm$ 1.21	48.62 $\pm$ 8.11
122–488 pg/mL	7.88 $\pm$ 1.10	6.71 $\pm$ 1.45	53.18 $\pm$ 10.44
>488 pg/mL	7.22 $\pm$ 1.25	7.62 $\pm$ 1.58	58.91 $\pm$ 11.26

Serum calcium showed a significant inverse association with iPTH ($p = 0.009$); serum phosphorus and Ca $\times$ P product showed significant positive associations with iPTH ($p = 0.004$ and $p = 0.01$).

Table 5: Distribution of Patients by CKD Stage (eGFR-based) and Corresponding Biochemical Parameters

CKD Stage	n (%)	Calcium (mg/dL)	Phosphorus (mg/dL)	Ca $\times$ P (mg ² /dL ²)	iPTH (pg/mL)
Stage 2	3 (3.09%)	8.82 $\pm$ 0.46	4.08 $\pm$ 1.05	35.96 $\pm$ 8.42	92.18 $\pm$ 26.44
Stage 3	5 (5.15%)	8.31 $\pm$ 0.53	4.92 $\pm$ 1.41	—	146.52 $\pm$ 41.08
Stage 4	28 (28.87%)	7.94 $\pm$ 0.61	5.78 $\pm$ 1.66	—	221.34 $\pm$ 66.12
Stage 5	61 (62.89%)	7.35 $\pm$ 0.88	7.18 $\pm$ 2.19	52.76 $\pm$ 15.94	328.41 $\pm$ 91.74

Calcium declined ($F = 7.48$, $p < 0.0001$) while phosphorus ($F = 6.02$, $p < 0.0001$) and iPTH ($F = 21.32$, $p < 0.0001$) rose progressively with advancing stage. No patients presented in Stage 1, reflecting late presentation typical of a dialysis-dependent cohort.

DISCUSSION

CKD-MBD arises from impaired phosphate excretion, reduced vitamin D activation and consequent hypocalcemia, which together drive persistent PTH secretion (Moe et al., 2007; Zaimi and Grapsa, 2024). The present mean iPTH of 286.74 $\pm$ 118.36 pg/mL, with 45.36% exceeding 488 pg/mL, confirms a high burden of secondary hyperparathyroidism, consistent with George and Bhat (2025). The predominance of middle-aged males (mean age 50.78 $\pm$ 14.88 years; 62.89% male) mirrors Reddy et al. (2014) and Chaturvedy et al. (2023), likely reflecting a higher burden of hypertension, diabetes and smoking among men.

The inverse calcium–iPTH relationship parallels the negative correlation reported by Chaturvedy et al. (2023), reflecting compensatory PTH rise triggered by hypocalcemia, while the positive phosphorus–iPTH association agrees with Rodriguez et al. (2016), confirming phosphate retention as a key driver of PTH secretion. Slinin et al. (2005) similarly linked disturbed calcium-phosphorus homeostasis and elevated PTH to cardiovascular risk in hemodialysis patients. The progressive rise in Ca $\times$ P product with iPTH corroborates Dosogi et al. (2022), reinforcing the role of phosphorus control in calcification risk.

The stepwise deterioration in calcium, phosphorus and iPTH across CKD stages agrees with Rastogi et al. (2021), who reported significantly higher iPTH in Stage 5 CKD patients with calcium below 8.5 mg/dL. The Stage 5 predominance in this cohort (62.89%) reflects

enrolment restricted to patients already on maintenance hemodialysis, consistent with the substantial global burden of advanced CKD described by Hill et al. (2016). Collectively, these findings reinforce the pathophysiology of CKD-MBD and support early, regular monitoring of calcium, phosphorus and iPTH to permit timely intervention and reduce renal osteodystrophy, vascular calcification and cardiovascular risk.

LIMITATIONS:

Single-center design and a sample of 97 limit generalizability; clinical outcomes (fractures, cardiovascular events, mortality) and other bone turnover markers (ALP, osteocalcin, FGF-23) were not assessed.

CONCLUSION

CKD-mineral and bone disorder is a common, progressive complication of advanced CKD on maintenance hemodialysis. Hypocalcemia, hyperphosphatemia, an elevated Ca $\times$ P product and secondary hyperparathyroidism were highly prevalent and worsened significantly with advancing CKD stage ($p < 0.0001$ for calcium, phosphorus and iPTH); calcium correlated inversely, and phosphorus and Ca $\times$ P product correlated positively, with iPTH. These findings support early identification and regular monitoring of calcium, phosphorus and iPTH, with timely correction of mineral imbalance, to reduce bone disease, vascular calcification and cardiovascular risk in this population.

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