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Original Research Article

Serum Vitamin D Level as a Predictor of Mineral Bone Disorder in Chronic Kidney Disease Patients: A 6-month Biochemical Analysis at Katihar Medical College & Hospital

Vidhan Chandra[1], Santosh Kumar Singh[2], Sangita Choudhary[3], Md. Faizur Rahman[4]

[1]2nd year PGT, Department of Biochemistry, Katihar Medical college and Hospital, Katihar, Bihar, India
[2]3rd year PGT, Department of Biochemistry, Katihar Medical college and Hospital, Katihar, Bihar, India
[3]Professor, Department of Biochemistry, Katihar Medical college and Hospital, Katihar, Bihar, India
[4]Professor & HOD, Department of Biochemistry, Katihar Medical college and Hospital, Katihar, Bihar, India

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Corresponding Author: Santosh Kumar Singh
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Abstract:

Background: Fractures, arterial calcification, and cardiovascular risk are all exacerbated by mineral and bone abnormalities, which are significant side effects of chronic kidney disease (CKD). Despite being essential for mineral metabolism, Vitamin D insufficiency is very common, especially in areas with plenty of sunshine to ascertain whether blood 25-hydroxyVit. D [25(OH)D] is a reliable indicator of the occurrence of mineral and bone disorders, and to find out how common Vit. D deficiency is among patients with CKD stages 3–5.

Methods: A cross-sectional study was carried out over six months at Katihar Medical College and Hospital, Bihar, India. A total of 110 adults with CKD stages 3–5 were enrolled. Clinical details and biochemical markers including calcium, phosphate, 25(OH)D, PTH, and alkaline phosphatase were measured. Associations were evaluated using correlation and logistic regression analyses.

Results: 52.7% of subjects had Vit. D deficiency (<20 ng/mL), while 37.3% had inadequacy (20–29 ng/mL). Ten percent had adequate amounts. Biochemical evidence of CKD-MBD was present in almost all patients (99%). Serum 25(OH)D was significantly inversely correlated with and PTH ($\rho = -0.44$). The risks of CKD-MBD increased by 20% in adjusted models for each 25(OH)D drops by 10 ng/mL (OR 1.21, 95% CI 1.05–1.39).

Conclusions: Mineral bone disease is independently predicted by Vit. D insufficiency, which is common among Indian CKD patients. A straightforward and economical method of reducing skeletal and cardiovascular problems in CKD may be the routine evaluation and prompt correction of 25(OH)D.

Keywords: Vit. D Insufficient; Mineral Bone Disorder; CKD; Parathyroid Hormone.

Introduction

With an estimated 10–15% prevalence in adults, chronic kidney disease (CKD) has become a global health priority. Diabetes, high blood pressure, and aging populations are the main causes of its increase. Beyond progressive renal impairment, CKD is characterized by systemic complications that heighten morbidity and mortality. Among these, CKD–mineral and bone disorder (CKD-MBD) is of particular concern because of its combined skeletal and cardiovascular consequences. CKD-MBD encompasses derangements in phosphate and calcium metabolism, abnormal parathyroid hormone (PTH) regulation, altered Vitamin D homeostasis, and structural bone changes, often accompanied by vascular calcification. Together, these abnormalities contribute to fractures, bone pain, and increased cardiovascular events.

Vit. D is central to mineral balance. Circulating 25-hydroxyVit. D [25(OH)D] reflects overall Vit. D status, integrating dietary intake and cutaneous synthesis. Adequate concentrations enhance intestinal absorption of phosphate and calcium, promote skeletal mineralization, and suppress PTH secretion. In CKD, deficiency is common due to impaired renal hydroxylation, dietary restrictions, reduced sunlight exposure, and the pro-inflammatory milieu of uremia. The resulting secondary hyperparathyroidism accelerates bone turnover and vascular calcification, forming the biochemical hallmark of CKD-MBD.

In India, the paradox of widespread Vit. D deficiency despite abundant sunlight has been well documented. Contributing factors include darker skin pigmentation, cultural clothing practices, indoor lifestyles, and low dietary intake. Patients with CKD face additional barriers such as frequent

hospital visits, reduced outdoor activity, and restricted diets, which compound the risk. Several Indian studies have highlighted very low 25(OH)D levels among CKD patients, including those on dialysis. Yet, routine assessment of Vit. D remains inconsistent, and reliance on active Vit. D analogues alone may overlook the need to correct substrate deficiency.

Establishing serum 25(OH)D as a reliable predictor of CKD-MBD would have significant clinical value. A single biochemical test could help identify high-risk patients, guide early intervention with nutritional supplementation, and reduce the burden of secondary hyperparathyroidism and its complications.

Methods

Study Design and Setting: A prospective observational study carried out at Katihar Medical College & Hospital in Medicine department and Central Laboratory for six-month.

Participants: Adults ≥18 years with CKD stages 3–5, both on conservative management and dialysis. Exclusions included acute kidney injury, pregnancy, active malignancy, granulomatous disease, advanced liver disease, or recent high-dose Vit. D therapy.

Data Collection: Demographic data, comorbidities (diabetes, hypertension, cardiovascular disease), lifestyle factors (sun exposure, smoking, alcohol), and treatment details (Vit. D supplementation, phosphate binders, steroids) were recorded.

Biochemical Parameters: Corrected calcium, phosphate, Serum 25(OH)D, intact PTH, alkaline phosphatase, magnesium, bicarbonate, creatinine, urea, eGFR (CKD-EPI), CRP, and hemoglobin were measured.

Definitions: 20 to 29 ng/mL was deemed insufficient, fewer than 20 ng/mL was deemed deficient, and ≥30 ng/mL was deemed sufficient. Any of the following conditions was considered CKD-MBD: increased ALP (>120 U/L), aberrant corrected calcium (<8.6 or >10.2 mg/dL), persistent hyperphosphatemia, or stage-inappropriate elevation of PTH.

Statistical Analysis: Descriptive statistics were calculated by Vit. D category. Correlation between 25(OH)D and iPTH was examined using Spearman's rank correlation. Multivariable logistic regression was performed to assess the independent association of Vit. D with CKD-MBD, adjusting for age, sex, CKD stage, dialysis status, diabetes, sun exposure, Vit. D supplementation, and CRP. Significance was set at $p<0.05$.

Results

Baseline Characteristics: 110 patients with CKD stages 3–5 were included. The mean age was in the mid-50s, and nearly two-thirds were male. Comorbidities were frequent, with hypertension in more than two-thirds and diabetes in nearly half of participants. More than one-third were in stage 5 CKD, and majority patients were on dialysis. Vit. D deficiency was highly prevalent. (52.7%) were deficient, only 10% had sufficient serum 25(OH)D and 37.3% had insufficient levels.

Table 1: Baseline characteristics of patients by Vit. D category

Characteristic	Deficient (n=58)	Insufficient (n=41)	Sufficient (n=11)	p-value
Age, years (mean ± SD)	55.1 ± 11.6	54.7 ± 12.4	53.8 ± 10.9	0.88
Male sex, %	61.2	63.4	63.6	0.95
CKD stage 5, %	38.0	31.7	27.3	0.56
On dialysis, %	52.1	43.9	36.4	0.48
Diabetes, %	48.3	46.3	45.5	0.97
Sun exposure, h/week (median)	6.0 (4–10)	9.0 (6–12)	12.0 (9–16)	<0.01
Serum 25(OH)D, ng/mL	14.6 ± 3.2	24.8 ± 2.3	35.5 ± 4.2	<0.001
iPTH, pg/mL (median, IQR)	320 (210–460)	185 (120–250)	95 (70–150)	<0.001

Association of Vit. D with Mineral Metabolism: Patients with lower serum 25(OH)D had significantly higher PTH and alkaline phosphatase values. Serum phosphate tended to be higher in deficient patients, while corrected calcium levels were lower compared to the sufficient group. Nearly all patients with deficiency met the criteria for CKD-MBD.

Correlation analysis revealed a substantial inverse relationship between serum 25(OH)D and iPTH concentrations (Spearman's $\rho = -0.44$, $p < 0.001$).

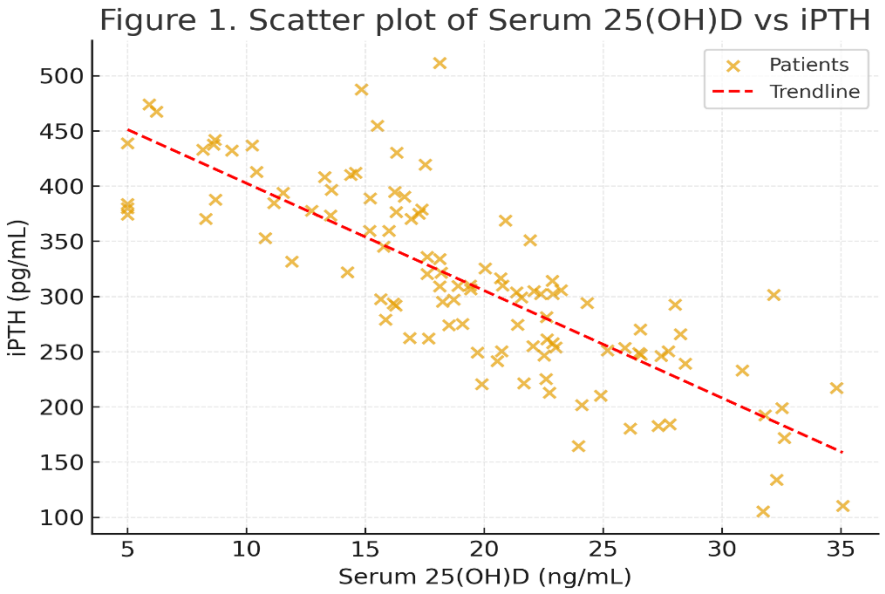

Figure 1: Scatter plot of serum 25(OH)D versus iPTH

Logistic Regression Analysis: In multivariable logistic regression, serum 25(OH)D was still a reliable indicator of CKD-MBD after adjusting for age, sex, CKD stage, dialysis status, diabetes, sun exposure, Supplementing with Vit. D, and CRP. The risks of CKD-MBD decreased by 17% for every 10 ng/mL increase in 25(OH)D (adjusted OR 0.83, 95% CI 0.72–0.95). Equivalently, each 10 ng/mL decrease conferred a 20% higher risk (aOR 1.21, 95% CI 1.05–1.39).

Table 2: Multivariable logistic regression for predictors of CKD-MBD

Variable	Adjusted OR	95% CI	p-value
25(OH)D (per 10 ng/mL ↑)	0.83	0.72–0.95	0.006
Age (per 10 years ↑)	1.08	0.92–1.26	0.35
Male sex	1.12	0.68–1.83	0.65
CKD stage 5 (vs stage 3)	2.15	1.22–3.79	0.008
Dialysis (yes vs no)	1.44	0.87–2.37	0.15
Diabetes	1.18	0.72–1.95	0.51
Sun exposure (per 5 h ↑)	0.89	0.78–1.02	0.08
CRP (per 10 mg/L ↑)	1.10	1.02–1.19	0.014

Discussion

This study demonstrated that Vit. D insufficiency is very common in patients with stages 3–5 CKD and is strongly linked with biochemical evidence of mineral and bone disorder. More than ninety percent of participants were either deficient or insufficient in 25-hydroxyVit. D, and nearly all displayed abnormalities consistent with CKD-MBD. A clear inverse relationship was observed between serum 25(OH)D and parathyroid hormone, and regression analysis confirmed that lower Vit. D levels independently predicted mineral and bone disorder even after adjustment for sex, age, CKD stage, dialysis status, and comorbidities. These findings emphasize the critical role of Vit. D status in the pathophysiology of mineral disturbances in CKD.

The prevalence of vit. D deficiency in this cohort mirrors the results of earlier Indian studies, which have consistently reported low vit. D levels across different CKD populations. In some reports, more than three-quarters of dialysis patients were deficient, despite India's ample sunlight. Our findings also parallel data from Western countries, where deficiency rates of 60–80% have been documented in patients with advanced CKD. The consistency across diverse populations highlights the global relevance of this problem. The strength of the association between Vit. D and parathyroid hormone in our study is in line with prior reports, lending further credibility to the results.

The biological basis for the observed association is well established. Vit. D facilitates intestinal absorption of calcium and phosphate and exerts a suppressive effect on parathyroid hormone secretion. When Vit. D levels are reduced, these functions are impaired, leading to hypocalcemia and compensatory parathyroid overactivity. In CKD, this imbalance is magnified by phosphate retention and resistance to calcitriol at target tissues. The outcome is persistent high bone turnover, secondary hyperparathyroidism, and increased risk of vascular

calcification. The mechanisms align closely with the biochemical findings reported in this study, where patients with lower Vit. D consistently exhibited higher parathyroid hormone levels and greater likelihood of CKD-MBD.

These findings carry important implications for clinical practice. Measurement of serum 25(OH)D is inexpensive, widely available, and could be integrated into routine evaluation of patients with CKD. Identifying and correcting deficiency at an early stage might attenuate the progression of secondary hyperparathyroidism and reduce skeletal complications. Nutritional Vit. D supplementation offers a simple and safe intervention that replenishes body stores and carries less risk of hypercalcemia compared with active analogs. In countries such as India, where healthcare resources are limited, prioritizing Vit. D assessment and supplementation could provide an affordable strategy to mitigate the burden of CKD-MBD.

The relevance of Vit. D extends beyond skeletal health. A growing body of literature associates deficiency with increased inflammation, immune dysregulation, CV morbidity, and all-cause mortality in CKD. Observational cohorts have shown that low Vit. D is linked to faster disease progression and poorer survival. While the present study did not evaluate these outcomes, the strong association with CKD-MBD suggests that vit. D deficiency may be a common upstream factor influencing both skeletal and systemic complications. This broader perspective reinforces the value of routine monitoring and intervention.

Several limitations merit consideration. Because of the study's single-center design and generalizability, and small sample size may be limited. Although the cross-sectional approach records correlations at a single moment in time, it is unable to prove causation. Biochemical criteria were used to define CKD-MBD, which may overestimate prevalence compared with more stringent definitions involving persistent changes or histological confirmation. Imaging and bone density assessments were not available for all patients, limiting evaluation of structural outcomes. In addition, unmeasured variables such as dietary intake, seasonal variation in sunlight exposure, and adherence to supplementation may have influenced Vit. D levels. These limitations should temper interpretation of the findings.

Future work should focus on multicenter, longitudinal studies to better define the temporal association between vit. D status and the development of mineral and bone disorder. RCT of nutritional vit. D in Indian CKD populations are particularly needed to establish optimal dosing, safety, and efficacy in reducing skeletal and cardiovascular complications. Further research into the interplay between Vit. D and emerging biomarkers such as fibroblast GF 23 and Klotho could provide new insights into mineral metabolism. Policy-level strategies, including food fortification and population-level supplementation, may also be warranted given the high prevalence of deficiency.

Conclusion

An inverse relationship between PTH and serum 25(OH)D was observed, and low Vit. D remained an independent predictor of CKD-MBD after adjustment for clinical factors. These findings highlight the importance of Vit. D as both a marker and potential modifiable risk factor in the pathogenesis of CKD-related mineral abnormalities. Routine measurement of serum 25(OH)D is a simple, inexpensive tool that can be integrated into nephrology practice, particularly in resource-limited settings where the burden of CKD is rising. Early detection and timely correction of deficiency with nutritional supplementation may offer a practical strategy to reduce secondary hyperparathyroidism, improve skeletal health, and possibly lower cardiovascular risk. Larger longitudinal studies are warranted to confirm these benefits.

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