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A Cross Sectional Study to Estimate the Prevalence of Mineral Bone Disease in Stage 3-5 Chronic Kidney Disease Patients

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Abstract

Background: Chronic Kidney Disease (CKD) affects 13.4% of adults worldwide, leading to Mineral Bone Disease (MBD), a common and severe complications in patients with advanced CKD (KDIGO stage 3-5), contributing significantly to morbidity and mortality.

Objective: To estimate the prevalence of MBD in stage 3-5 CKD patients and explore associations with clinical and biochemical parameters.

Methodology: A cross-sectional study was conducted in 103 stage 3-5 CKD patients, assessing biochemical parameters and radiological examinations.

Results: Prevalence of MBD in CKD among study population was 66.02% (n=68). Significant associations existed between hyperphosphatemia, hypocalcemia, hyperparathyroidism were the most common abnormalities. Factors such as advanced CKD, age and co morbidities were significantly associated with MBD.

Conclusion: This study emphasizes the high prevalence of CKD – MBD among advanced CKD patients and need for early screening and management.

Keywords: Chronic Kidney Disease (CKD), Mineral Bone Disease (MBD), iPTH, Vitamin D, Calcium, Phosphate, Cardiovascular Complications.

Introduction

Chronic Kidney Disease (CKD) has gained a global health concern, affecting 13.4% adults worldwide impacting the overall well-being and adversely affecting

the life expectancy due to the declining kidney function.[1,2] Conditions such as polycystic kidney disease, glomerulonephritis, diabetes mellitus, hypertension can result in CKD which leads to the progressive loss of nephrons and a decline in renal function.[3] The severity of which can be measured by eGFR with the higher stages being difficult to manage as well as susceptible to specific complications and elevated mortality risks.[4,5]

Chronic Kidney Disease (CKD) is associated with various complications, including fluid and electrolyte imbalances, cardiovascular disease, anemia, and mineral bone disease (MBD), characterized by abnormal bone structure, mineral metabolism, with altered serum levels of iPTH, vitamin D3, calcium, and phosphate.[6,7,8,9] Notably, all phases of CKD are associated with MBD which deteriorates parallelly to the declining renal function.[10] Chronic Kidney Disease-Mineral Bone Disorder (CKD-MBD) is a complex condition characterized by vascular and valvular calcifications leading to cardiovascular disease, bone abnormalities, fractures and disturbances in mineral metabolism, significantly increasing mortality and morbidity risks in CKD patients. Hallmarked by hypocalcemia, hyperphosphatemia, secondary hyperparathyroidism, and abnormal bone turnover, CKD-MBD arises from the progressive impairment of calcium and phosphate regulation, leading to elevated parathyroid hormone levels and associated bone and cardiovascular complications.[8,11,12] The primary reason being altered regulation of Calcium, phosphate, PTH and vitamin D characterized by an increased PTH secretion, due to declining calcium reabsorption and phosphate retention. Furthermore, the decreased synthesis of active vitamin D (Calcitriol) leads to alterations in soft and skeletal tissues, accelerating the development of CKD-MBD.[8,9]

The deterioration of kidney function impairs phosphate excretion, leading to hyperphosphatemia, a hallmark of Mineral Bone Disease (MBD) in CKD. To counterbalance, the parathyroid glands secrete excess parathyroid hormone (PTH), triggering increased bone resorption, structural abnormalities, and mineral loss. Concurrently, decreased calcitriol synthesis reduces calcium absorption, exacerbating secondary hyperparathyroidism and contributing to hypocalcemia, thereby perpetuating the complex pathophysiology of MBD in CKD.[8,9]

Although prevalent in all phases of CKD, MBD may show variation in terms of incidence and prevalence as well as severity based on the subjects under study. Generically, by the time a patient reaches stage 5 CKD, they will have some form of MBD. Owing to the severity of the condition and associated risks of morbidity and mortality, early detection and treatment are essential in enhancing the patient outcomes.[10,13–18] This cross-sectional study aimed to estimate the prevalence of MBD in stage 3-5 CKD patients, and explore the factors associated with its occurrence providing crucial insights into the severity and scope of this complication so as to provide targeted interventions, enabling healthcare professionals to optimize MBD management and improve outcomes and quality of life for CKD patients.

Methodology

This cross-sectional cohort study was conducted on patients attending Medicine and Nephrology OPD and inpatient wards at Bharati Hospital and Research Centre, Pune, from June 2022 to June 2024 on 103 patients with stage 3-5 Chronic Kidney Disease (CKD) confirmed based on eGFR using KDIGO classification, sample size was calculated using a formula based on a 49.6%

prevalence of hypocalcemia from a previous study.[19] meeting the following inclusion and exclusion criteria:

Inclusion Criteria: Patients with stage 3-5 CKD, eGFR < 60 mL/min, and biochemical, ultrasonographic, or histological evidence of CKD.

Exclusion Criteria: Patients with liver disease, rickets, osteomalacia, renal transplant recipient, or individuals taking non-steroidal anti-inflammatory drugs or anti-epileptics and medications affecting bone metabolism like steroids and bisphosphonates.

Methodology

- Patient demographics and medical history were collected using a questionnaire.
- Venous blood (8-10 ml) was collected for biochemical analysis, including serum albumin, creatinine, Vitamin D3, iPTH (parathyroid hormone), magnesium, calcium, phosphorus, and alkaline phosphatase (ALP) were measured for assessment.
- Radiological assessments included 2D echocardiography for valvular and vascular calcifications, ultrasonography abdomen pelvis, and X-ray abdomen erect for aortic calcifications.
- The prevalence of MBD and its co relations with demographic, clinical and laboratory parameters were analyzed.

Statistical Analysis

Statistical analysis involved descriptive statistics to summarize demographic and clinical characteristics of CKD patients. Inferential statistics, specifically independent t-tests and Mann-Whitney U tests, examined differences in biochemical parameters between patients with and without Mineral Bone Disease (MBD). Associations between MBD status and demographic/clinical variables were also investigated. Statistical significance was set at $p < 0.05$. Data analysis was facilitated using SPSS, yielding results reported with effect sizes, confidence intervals, and p-values.

Results

Keeping in mind, 49.6% prevalence of hypocalcemia in stage 3 – 5 CKD (one of our predictor variables) in previous study by Shankar P et al 2017.[19]

The formula used to calculate sample size for my study is as follows:

n = z2pq/d2 here, 4- z2

n = 4 x 49.6 x 50.9 / 9.8 x 9.8

p - prevalence n= 103, q – (100-P), d – 20% of P

Hence, sample size is taken as 103, with a margin of 5% and 95% confidence interval.

A total of 103 patients were included in present study to evaluate the prevalence of MBD in these patients which had a male: female distribution of 75:28 (72.82%: 27.18%) with mean age of 52.19% . While the majority of participants had presence of co-morbidities (n=89, 86.41%) and biochemical abnormalities (n=68, 66.02%), relatively fewer patients were on dialysis (n=45, 43.69%). Also, from among the study participants, 68 (66.02%) had MBD while 35 (33.98%) did not have MBD.

Gender	Frequency	Percent
Male	75	72.82
Female	28	27.18
Total	103	100.00

It was observed that majority of patients did not consume any form of supplements. From among those taking some form of supplements, maximum contribution was by calcium supplements (n=40) followed by vit. D (n=29), phosphate binders (n=27), active vit. D (n=12) and calcimimetics (n=4) (Figure 1).

The descriptive statistics for several biochemical parameters evaluated have been tabulated in Table 2. The correlation between presence or absence of MBD based on serum levels of different minerals and albumin showed that while there was no significant association between levels of albumin, phosphorous, magnesium and MBD, the serum calcium levels were significantly lower in those with MBD (7.22) as compared to those without (8.37) as indicated by a p value <0.001 (Table 3, Figure 2).

For the other serum parameters (creatinine, urea, vit. D, iPTH, ALP) evaluated similarly, only iPTH showed a statistically significant association (p<0.001) with MBD cases having a mean value of 452.65 compared to their negative counterparts with a mean of 126 units (Table 4, Figure 3).

Radiological examinations revealed a high prevalence of chronic kidney disease features in all patients (100%) via ultrasound, while valvular calcifications were observed in 13.59% (n=14) of patients via 2D-echo. In contrast, vascular calcifications were detected in only 1.94% of patients (2/103) through X-ray of the abdomen erect, however, the majority of the patients (97.09%) did not undergo this examination. Notably, 86.41% of patients showed no valvular calcifications, suggesting a potential area for targeted interventions to mitigate cardiovascular complications in this CKD population.

Discussion

The present study was conducted on stage 3-5 CKD patients to evaluate the prevalence of MBD in such cases and also identify association, if any between MBD and clinical/ biochemical parameters. The study participant comprised of almost 3/4th males (72.82%) with the rest being females (27.18%) similar to the findings of Vikrant and Parashar who also observed that men are more likely to be affected by CKD compared to women.[10]

The study population's demographics showed a wide age range (13-83 years) with a mean age of 52.19 ± 15.46 years. It was observed that 86.41% had comorbidities and 43.69% undergoing hemodialysis. These findings highlight the complexity of CKD care, emphasizing the need for individualized approaches to address multifaceted health needs, particularly in patients with comorbid conditions.

Evaluation of the various biochemical parameters revealed that statistically significant difference existed between the serum levels of Calcium and iPTH in patients with and without MBD. While the mean values for these parameters were 7.61 mg/dL and 439.48 units respectively, the serum calcium levels were significantly lower for patients with MBD (7.22 vs 8.37) and for iPTH, the values were significantly higher for MBD cases (452.65 vs 126).

Calcium supplementation is vital in managing Chronic Kidney Disease (CKD), particularly when combined with Mineral Bone Disease (MBD), which is common in advanced CKD stages.[20] CKD patients often struggle to maintain optimal serum calcium and phosphate levels, leading to hypocalcemia and secondary hyperparathyroidism, necessitating the intake of calcium supplements so as to prevent bone demineralization and

fracture risk.[20,21] However, careful control is necessary to avoid hypercalcemia and vascular calcification, especially when combined with calcimimetics or vitamin D analogs. Regular monitoring of serum calcium, phosphate, and PTH levels is crucial to customize dosage and ensure safe and effective use of calcium supplements in CKD patients.[22]

In the present study, 38.83% (n=40) of the patients in were taking calcium supplements, 26.21% (n=27) were taking phosphate binders, 28.16% (n= 29) were taking vitamin D supplements, and 11.65% (n = 12) were taking active vitamin D. The findings reveal various strategies for managing mineral imbalances in chronic kidney disease (CKD) patients, highlighting a notable gap in treatment adherence, as a substantial proportion of patients are not receiving necessary supplements. In the Chuang et al. study, phosphate binders and vitamin D analogues were prescribed to 84.9% and 41.9% patients on peritoneal dialysis, respectively, indicating a significant increase in their use. In contrast, only 26.21% of participants in the current study used phosphate binders, and 28.16% used vitamin D supplements. Chuang et al. also found that the patient survival rate after a year was 88.4%, and that the mortality rates were significantly affected by meeting the KDIGO targets for serum phosphorus.[18]

In the study conducted by Abdu et al. none of the patients had normal vitamin D3 levels, with a mean of 43.79 ± 21 ng/ml.[17] While vitamin D deficiency is prevalent in CKD patients, affecting bone health and disease outcomes. Our study underscores the importance of monitoring and managing vitamin D levels to prevent/treat MBD, as evidenced by the fact that several patients in the present study had intake of supplements.

Radiographic findings highlighted the underutilization of diagnostic tests and significant vascular calcifications in CKD patients. X-ray abdomen was performed in only 2.91% (3/103) of patients as we tried to reduce unnecessary radiation exposure, revealing vascular calcifications in 1.94% (2/103). Abdominal and pelvic ultrasounds showed universal CKD features in all participants (100%), while 2D-echo detected valvular calcifications in 13.59% (14/103). Ghosh et al. reported a low incidence of renal osteodystrophy and normal X-ray findings in most CKD patients.[14] Echocardiography revealed no stage-dependent differences in valvular calcifications, highlighting a need for further research into MBD-related cardiovascular risks.

Our study's prevalence of MBD (66.02%) is comparable to Chuang et al.'s findings (52.3-86.0%) wherein the prevalence of CKD-MBD varied after the onset of peritoneal dialysis.[18] Also, Abdu et al. have reported a prevalence of CKD-MBD of 58% among hemodialysis patients.[17] However, it contrasts with study conducted by George et al. which found a lower prevalence of low turnover bone disease (44.4%).[15] These variations highlight the diversity of MBD manifestations and the impact of patient demographics, management modalities, and CKD stages.

Biochemical abnormalities, hallmark of MBD, were present in 66.02% of our patients, consistent with Vikrant and Parashar's report of high rates of secondary hyperparathyroidism (82.7%) and hyperphosphatemia (55.4%).[10] Significant disruptions in calcium and phosphate metabolism were observed, with lower serum calcium and higher phosphorus levels in MBD patients. Our findings corroborate Ghosh et al.'s report of widespread vitamin D deficiency and hyperparathyroidism in CKD stages 4 and 5D.[14]

Similarly, in a study conducted by Valson et al. the authors have reported vitamin D deficiency in 74.7% of patients and hyperparathyroidism in 89.3%.[13] Choudhary et al.'s study also reported significant biochemical abnormalities, including hypocalcemia (27.8%) and hyperphosphatemia (48.3%).[14]

The low adoption rate of calcimimetics (3.9%) in our study highlights a significant treatment gap for secondary hyperparathyroidism. Regular monitoring of biochemical markers is crucial to modify treatment plans and prevent severe skeletal and cardiovascular complications.

In conclusion, our study underscores the high prevalence and complexity of MBD in CKD patients, emphasizing the need for comprehensive management strategies, including vitamin D supplementation, phosphate binders, and calcimimetics.

Conclusion

This study highlights the high prevalence and complex management needs of Mineral Bone Disease (MBD) in Chronic Kidney Disease (CKD) patients. Early intervention, tailored treatment, and regular monitoring of biochemical markers are crucial for improving bone health and reducing cardiovascular complications, emphasizing the need for ongoing research and clinical vigilance.

Limitations of the Study

While this study provides valuable insights into the prevalence and biochemical profile of mineral bone disease among patients with advanced chronic kidney disease, certain limitations should be noted. As it was a cross-sectional study, the findings reflect associations observed at a single point in time and do not establish causality. The study was conducted at a single tertiary care center with a modest sample size of 103 participants; however, the detailed biochemical and radiological evaluation of each patient adds strength to the reliability of the data. Advanced diagnostic modalities such as DEXA scans, bone biopsies, or bone turnover markers were not included, which could have offered a more in-depth assessment of bone status. Some potential confounders, including dietary factors, vitamin D supplementation, duration of CKD, and use of medications like phosphate binders or calcimimetics, were not fully explored, which may have influenced the results to some extent. Additionally, biochemical parameters were assessed at a single time point, and serial measurements might have provided a more dynamic picture of mineral metabolism. Despite these limitations, the study contributes important baseline information on CKD–MBD in the local population and underscores the need for early screening and preventive strategies in this high-risk group.

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Legend Tables and Figures

Table 1: Status of study participants w.r.t. presence or absence of Co-morbidities, Dialysis, Biochemical abnormalities, and MBD

		Frequency	Percent
Comorbidities	Yes	89	86.41
	No	14	13.59
On dialysis	Yes	45	43.69
	No	58	56.31
Biochemical abnormalities	Yes	68	66.02
	No	35	33.98
MBD	Yes	68	66.02
	No	35	33.98

Figure 1: Usage of Supplements in Study Participants

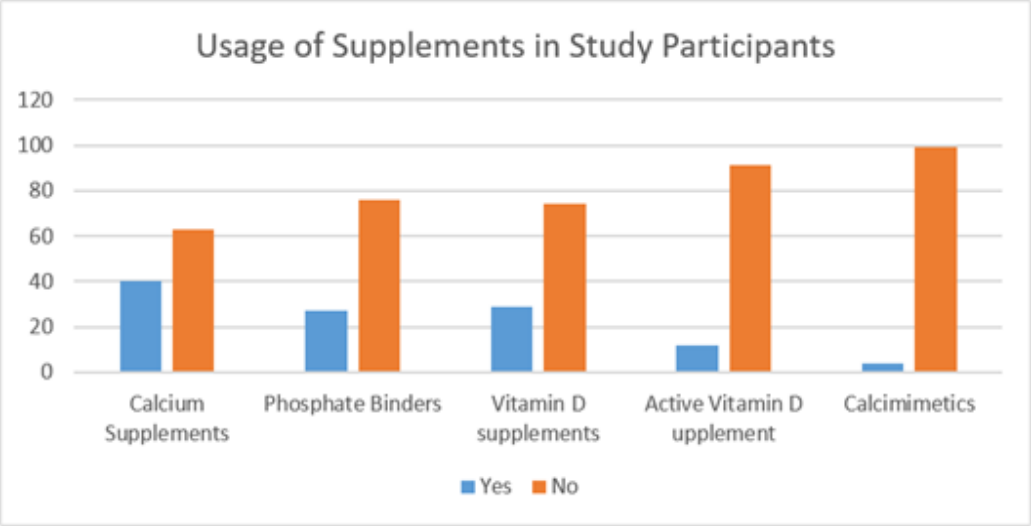

Table 2: Descriptive Statistics of Biochemical Parameters in Study Participants

	Minimum	Maximum	Mean	SD
Sr. Creatinine	1.99	22.37	8.76	5.02
Sr. Urea	20.00	510.00	123.02	71.89

Sr. Phosphorus	1.60	13.10	5.12	2.16
Sr. Calcium	3.00	11.92	7.61	1.39
Sr. Albumin	1.40	4.70	3.17	0.57
Sr. Magnesium	1.05	2.90	2.05	0.34
iPTH	16.00	1335.00	439.48	332.93
Sr. Vitamin D	4.00	71.80	14.73	10.32
Sr. Alkaline phosphatase	43.00	659.00	113.66	80.33

Table 3: Association of Biochemical Parameters with Mineral Bone Disease (Independent t-test)

Independent t test	MINERAL BONE DISEASE	Mean	SD	p-value
Sr. Phosphorus	No	4.20	1.26	0.002
	Yes	5.59	2.37	
Sr. Calcium	No	8.37	1.12	<0.001
	Yes	7.22	1.36	
Sr. Albumin	No	3.19	0.54	0.88
	Yes	3.17	0.18	
Sr. Magnesium	No	2.15	0.34	0.03
	Yes	2.00	0.33	

Figure 2: Association of Biochemical Parameters with Mineral Bone Disease (Independent t-test)

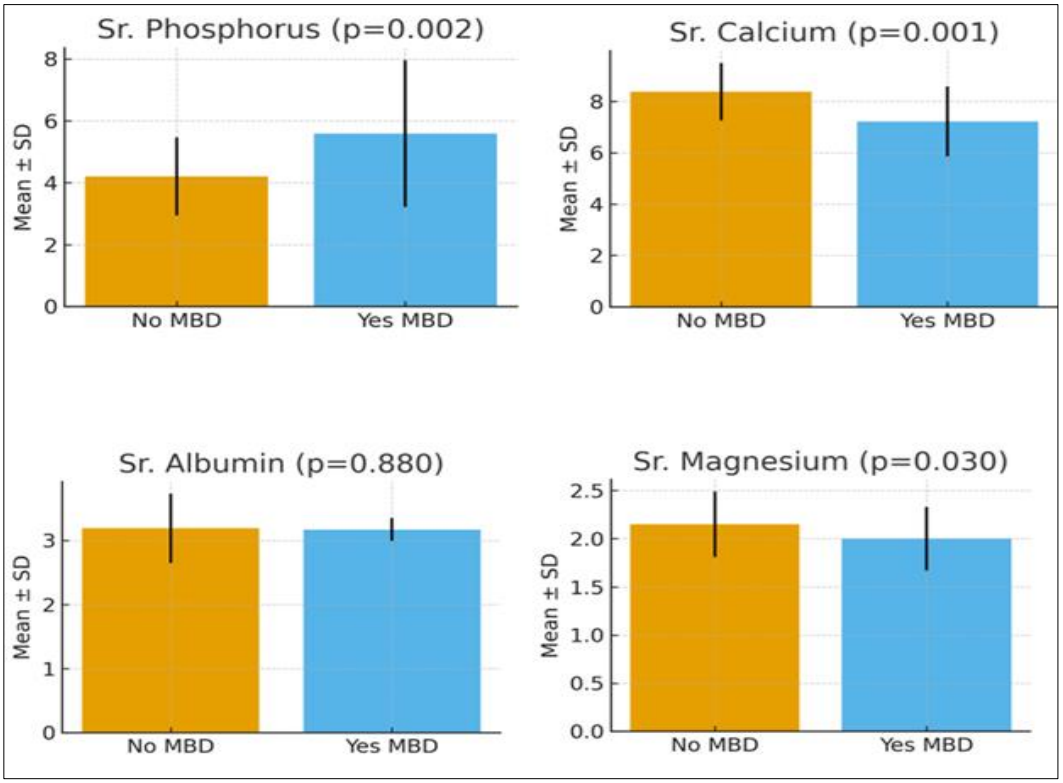

Table 4: Biochemical Parameters Assessed by Mann-Whitney U Test for Mineral Bone Disease

Mann Whitney UTest	Mineral Bone Disease	Median (IQR)	p- value
Sr. Creatinine	No	6.50 (4.43-7.67)	0.007
	Yes	7.95 (5.99-12.31)	
Sr. Urea	No	108 (71-127)	0.043
	Yes	127 (69.25-157)	
iPTH	No	126 (83.40-185.10)	<0.001
	Yes	452.65 (367.20-725.60)	
Sr. Vitamin D	No	14.70 (10.60-21.40)	0.046
	Yes	12 (8.23-17.33)	
Sr. Alkalinephosphatase	No	87 (60-136)	0.55
	Yes	90 (77-137)	

Figure 3: Biochemical Parameters Assessed by Mann-Whitney U Test for Mineral Bone Disease