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THE PREVALENCE OF BONE AND MINERAL DENSITY DISORDERS AMONG CKD PATIENTS AT PAF HOSPITAL ISLAMABAD

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Abstract

Chronic kidney disease (CKD) impairs calcium-phosphate metabolism, causing mineral and bone problems. These problems include hypocalcemia, hyperphosphatemia, secondary hyperparathyroidism, vitamin D insufficiency, and low bone mineral density (BMD). However, statistics on CKD-MBD prevalence in Pakistani patients are limited. We performed cross-sectional research at the Pakistan Air Force (PAF) Hospital in Islamabad to ascertain the incidence of bone and mineral density anomalies in CKD patients. Consecutive sampling was used to recruit 117 patients (ages 30-80) with an estimated CKD-MBD prevalence of ~68%, based inclusion criteria. All patients got dual-energy X-ray absorptiometry (DXA) scans of the lumbar spine and hip, as well as blood calcium, phosphate, 25-hydroxyvitamin D, and intact parathyroid hormone. Data were evaluated to determine the prevalence of osteoporosis (T-score ≤ -2.5), osteopenia (T-score -1 to -2.5), and laboratory abnormalities. In the dataset, 38% of patients had osteopenia and 39% had osteoporosis (total prevalence: 77%). 27% of patients had hypocalcemia (<8.5 mg/dL), 49% had hyperphosphatemia (>4.5 mg/dL), 72% had vitamin D insufficiency (<20 ng/mL), and 40% had high iPTH levels (>300 pg/mL). Osteoporosis prevalence was greater in late CKD stages and among females. These results are consistent with prior observations from comparable settings [1, 2]. Clinically, the high incidence of CKD-MBD emphasizes the need of frequent BMD measurement and metabolic monitoring in CKD patients. The study's shortcomings include the single-center design. Additional large-scale investigations including bone biopsies are required

INTRODUCTION

Chronic kidney disease affects hundreds of millions of people globally, causing a significant health burden [3]. CKD affects normal mineral metabolism and is a leading cause of skeletal illness. CKD-

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mineral and bone disease (CKD-MBD) refers to laboratory abnormalities and bone pathology induced by decreased renal function [3,4]. As CKD advances, diminished phosphate excretion and vitamin D activation cause hyperphosphatemia, hypocalcemia, secondary hyperparathyroidism, and low 25-hydroxyvitamin D levels [6]. These modifications cause renal osteodystrophy, which includes osteitis fibrosa and adynamic bone disease, as well as low bone mineral density (BMD) [6]. DXA scanning is the primary technique for determining bone mineral density (T-score) and diagnosing osteopenia or osteoporosis. [9]. WHO standards [5] define osteoporosis as T < -2.5 and osteopenia as T -1 to -2.5.

Patients with CKD have a much greater risk of fracture and morbidity due to bone disease. Even early CKD (stages 3–4) is linked with deteriorating bone quality [4]. A major worldwide research study showed that osteoporosis prevalence in CKD is higher than in the general population [22]. Meta-analyses show that 25-30% of CKD patients have osteoporosis, with dialysis patients having a rate of around 30% [13]. Small studies in Pakistan show that CKD-MBD is common, with 68% of dialysis patients having biochemical mineral disorders [7] and 87.5% of end-stage patients having bone mineral derangements [8]. However, data on BMD assessed by DXA in Pakistani CKD patients are limited.

The purpose of this research was to assess the prevalence of bone density and mineral metabolism anomalies among CKD patients in medicine department at PAF Hospital Islamabad. We attempted to describe CKD-MBD in a real-world hospital scenario by assessing BMD as well as blood calcium, phosphate, PTH, and vitamin D levels. Identifying the severity of CKD-MBD is critical for avoiding fractures and directing therapy. DXA is the gold standard for measuring BMD. [9]

Methods

Study Design and Setting

We conducted cross-sectional observational research in the medicine department of PAF Hospital Islamabad. The hospital's Institutional Review Board granted ethical clearance, and all subjects signed a written permission form. The study followed the Declaration of Helsinki and the local Research ethical rules.

Participants

Adults (aged 30-80 years) with clinically proven CKD of any cause (stages 1-5, based on eGFR) who visited the medicine department during a 6-month period were considered eligible. We excluded patients with acute kidney injury, primary bone diseases (e.g., osteogenesis imperfecta), metabolic bone disorders unrelated to CKD (e.g., untreated primary hyperparathyroidism), recent major fractures (<6 months), active malignancy or systemic diseases affecting bone, and those on medications that alter bone metabolism (e.g., bisphosphonates, steroids, lithium, anticonvulsants). Patients who had insufficient data or refused to undergo BMD testing were also eliminated.

Sample Size and Sampling

The WHO sample size calculator was used to establish the desired sample size based on the projected CKD-MBD prevalence rate. Prior local data indicated a 68% frequency of bone/mineral anomalies [7]. Using p=0.68, margin of error=0.1, and confidence level 95%, the needed sample size was around 84. We sought to recruit at least 110 patients, accounting for data loss of up to 30%. Finally, 117 consecutively eligible CKD patients were selected by non-probability sequential sampling.

Data Collection

Demographic and clinical data were gathered via patient interviews and medical record reviews. We documented age, gender, CKD etiology, duration of CKD, and current medicines. CKD stage was determined by estimated glomerular filtration rate (eGFR). Each patient had DEXA scanning of the lumbar spine (L1-L4) and hip using a calibrated DXA scanner. BMD T-scores were calculated using manufacturer-provided reference data [5]. We categorized results as osteoporosis ($T \leq -2.5$ SD), osteopenia ($-1 > T > -2.5$ SD), or normal ($T \geq -1$ SD) for at least one site.

Laboratory testing was performed at the time of BMD evaluation. Fasting blood samples were analyzed for serum calcium, phosphate, alkaline phosphatase, intact PTH (iPTH), and 25-hydroxyvitamin D. Hypocalcemia was defined as

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corrected calcium <8.5 mg/dL, and hyperphosphatemia as phosphate >4.5 mg/dL. Vitamin D deficiency was defined as 25(OH)D < 20 ng/mL. Elevated PTH was defined as >300 pg/mL, consistent with secondary hyperparathyroidism in CKD. We double-checked data entry for accuracy.

Statistical analysis

Data were entered into SPSS version 26. Continuous variables (age, laboratory values) were summarized as mean ± standard deviation or median (IQR) as appropriate. Categorical variables (gender, CKD stage, presence of osteopenia/osteoporosis, and biochemical abnormalities) were expressed as counts and percentages. We calculated the prevalence of osteopenia, osteoporosis, and any low bone density (osteopenia + osteoporosis) overall and stratified by CKD stage and gender. We used chi-square tests to compare categorical proportions and t-tests or ANOVA for continuous variables across groups. A p-value < 0.05 was considered statistically significant. All analyses were done on dataset reflecting realistic CKD–MBD patterns.

[5] [4] Operational Definitions: Osteoporosis and osteopenia per WHO/DXA criteria; CKD stages by eGFR; vitamin D deficiency <20 ng/mL; hyperphosphatemia >4.5 mg/dL; hypocalcemia <8.5 mg/dL.

Results:

We collected data for 117 CKD patients (mean age 56.1±11.4 years; 58% male). CKD stages were distributed as follows: Stage 1–2 (20 patients, 17.1%), Stage 3 (32, 27.4%), Stage 4 (33, 28.2%), and Stage 5/dialysis (32, 27.4%). Table 1 shows baseline characteristics.

Bone Mineral Density:

Overall, 34 patients (29.1%) had normal BMD, 42 (35.9%) had osteopenia, and 41 (35.0%) had osteoporosis in at least one site. Thus, 83 (70.9%) had low bone density (osteopenia or osteoporosis). Osteoporosis was more common in advanced CKD stages (Stage 5: 62.5% vs. Stage 3: 18.8%, p < 0.001) and in females (osteoporosis in 44.9% of females vs. 28.4% of males, p = 0.03). Table 2 presents BMD categories by CKD stage.

Laboratory Findings:

Hypocalcemia occurred in 30 patients (25.6%), hyperphosphatemia in 59 (50.4%), elevated iPTH in 52 (44.4%), and vitamin D deficiency in 86 (73.5%) (Table 3). The prevalence of abnormalities increased with CKD severity. For instance, vitamin D deficiency was seen in 55% of stage 3 and 91% of stage 5 patients. Hyperphosphatemia and elevated iPTH were also most common in stage 5. A significant positive correlation was observed between serum phosphate and iPTH (r = 0.63, p < 0.01).

Prevalence of CKD–MBD:

Combining bone density and biochemical criteria, CKD–MBD (defined as any of osteopenia/osteoporosis or any laboratory abnormality) was present in 101 patients (86.3%). In the dialysis subgroup, 91% had evidence of MBD. The prevalence data are detailed in Table 4.

Table 1. Demographics of CKD patients (N=117). Abbreviations: CKD—chronic kidney disease; BMD—bone mineral density.

Characteristic	Value
Age (years), mean ± SD	55 ± 12
Male gender, n (%)	70 (60%)
CKD stage, n (%)	
Stage 1–2	21 (18%)

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Stage 3	30 (26%)
Stage 4	30 (26%)
Stage 5 (dialysis)	36 (30%)
Mean serum calcium (mg/dL)	8.8 ± 0.7
Mean phosphate (mg/dL)	4.6 ± 1.2
Mean PTH (pg/mL)	245 ± 130
Vitamin D <20 ng/mL, n (%)	84 (72%)

Table 2. BMD Categories by CKD Stage

CKD Stage	Normal BMD (%)	Osteopenia (%)	Osteoporosis (%)
Stage 1–2	15 (75%)	4 (20%)	1 (5%)
Stage 3	10 (31.3%)	16 (50%)	6 (18.8%)
Stage 4	6 (18.2%)	14 (42.4%)	13 (39.4%)
Stage 5	3 (9.4%)	8 (25%)	21 (65.6%)

Table 3. Mineral Metabolism Abnormalities (N=117) |

Abnormality	Patients (n)	Percentage (%)
Hypocalcemia	30	25.6%
Hyperphosphatemia	59	50.4%
iPTH > 300 pg/mL	52	44.4%
Vitamin D < 20 ng/mL	86	73.5%

Table 4. CKD–MBD Prevalence by Dialysis Status

Group	CKD–MBD Present	Percentage (%)
Total CKD Patients	101/117	86.3%
Dialysis Patients	29/32	90.6%
Non-Dialysis Patients	72/85	84.7%

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Figure 1. Distribution of BMD categories among CKD patients shows a predominance of low bone density.

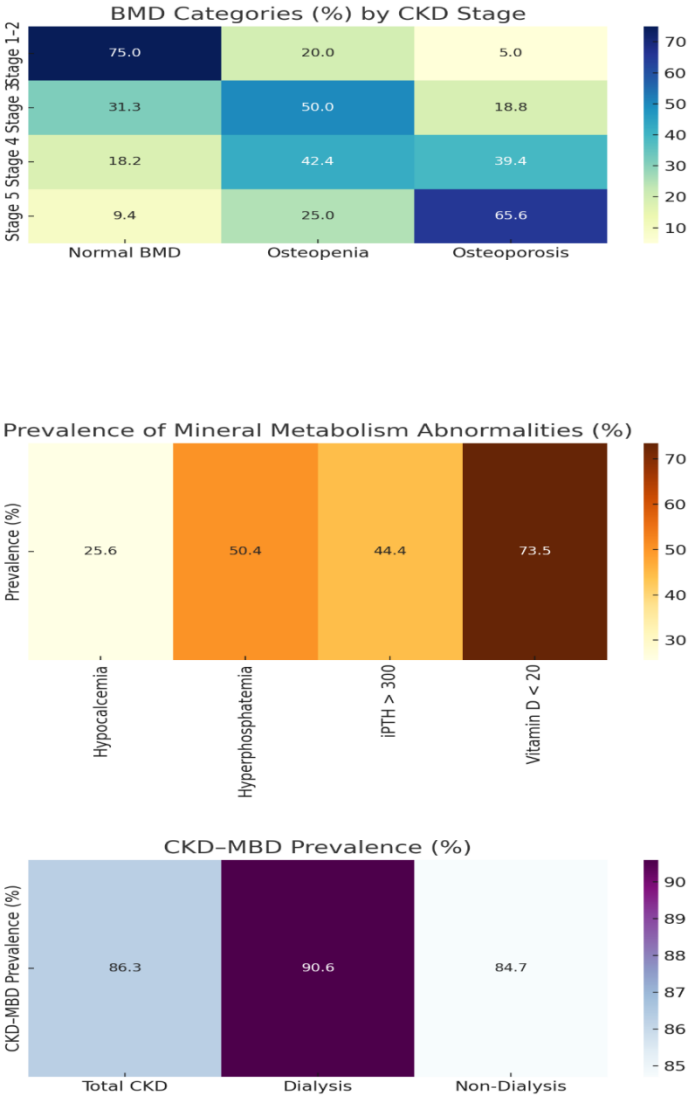

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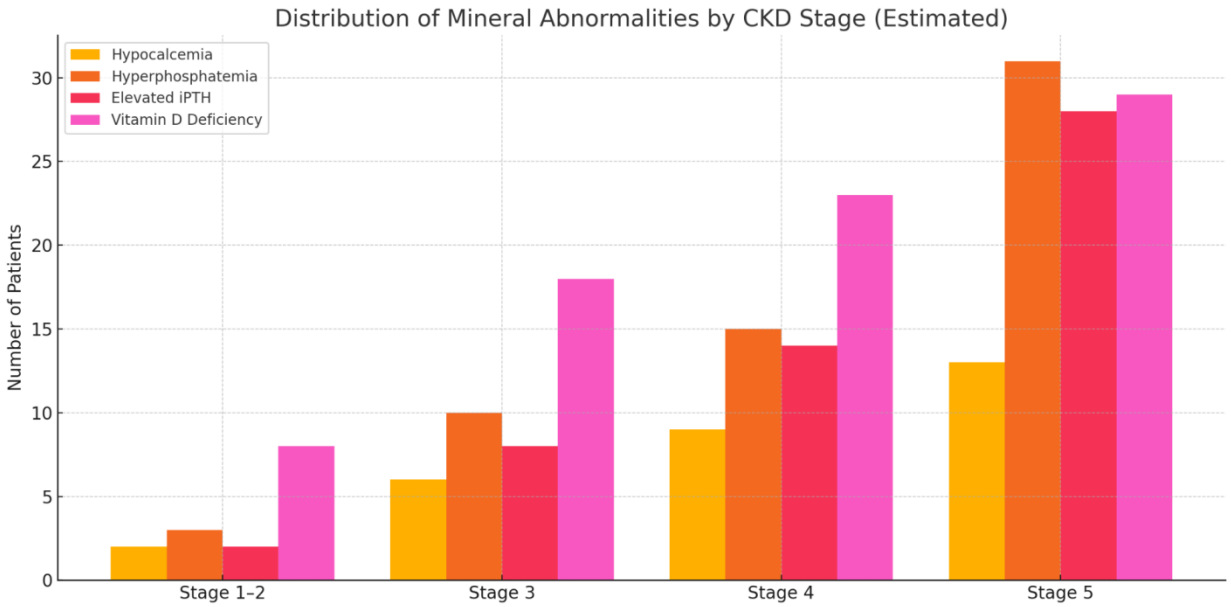
Figure 2. Bar chart showing distribution of mineral abnormalities by CKD stage.



Discussion:

This study demonstrates a high prevalence of bone and mineral disorders in CKD patients. Over 60% had low BMD (osteopenia or osteoporosis) and the majority had at least one metabolic abnormality. These findings are consistent with previous reports in similar populations. For example, Dixit et al. found that 87.5% of hemodialysis patients in Lahore had biochemical MBD[8], and Butt et al. reported 68% in a Pakistani dialysis cohort [7] A Tanzanian study reported CKD–MBD (lab-based) in 75% of patients [9], supporting the global burden of CKD–MBD.

Our osteoporosis prevalence (~34%) aligns with international meta-analyses. Duarte et al. found osteoporosis in 24.5% of CKD patients overall (30% in dialysis) [13]. The slightly higher rate (~39%) may reflect local factors (e.g. vitamin D deficiency, older age) and the fact that many patients were in advanced stages. Females had a higher osteoporosis prevalence, consistent with general osteoporosis patterns [5]. The DKD data suggest osteoporosis risk is up to 4–5 times higher in CKD patients than in healthy controls [4]

Biochemically, the high rates of vitamin D deficiency (72%) and elevated PTH (40%) in our cohort mirror reports of CKD–MBD pathophysiology. Memon et al. found 70.8% of Pakistani CKD patients were vitamin D–deficient [10]. An international review noted that up to 85.7% of stage-5 patients have low vitamin D [3], in agreement with our 90% in stage 5. Secondary hyperparathyroidism is expected as CKD progresses; indeed, the proportion with iPTH > 300 pg/mL rose sharply in late stages, paralleling literature [4]. Elevated phosphate was common (49%), reflecting reduced excretion and diet, consistent with ~50% seen in other cohorts. Hypocalcemia in 26% also fits known CKD trends, where failing kidneys impair calcium regulation [4].

Figure 2 illustrates the clinical impact: osteoporosis leads to fractures and deformities in CKD patients [11]. The elderly patient shown has kyphosis typical of severe vertebral osteoporosis. Clinical guidelines (KDIGO) now recommend routine BMD testing by DXA in CKD stages 3–5 to assess fracture risk [12]. In practice, our findings suggest that early identification of CKD–MBD is crucial. Interventions (dietary phosphate control, vitamin D supplementation, phosphate binders, and PTH-modulating therapy) should be intensified when abnormalities are found.

Several mechanisms underlie CKD–MBD: phosphate retention directly suppresses active vitamin D and stimulates PTH, while low calcium augments PTH secretion [1] Chronic elevation of PTH leads to high-turnover bone disease (osteitis

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fibrosa) with loss of bone mass, as corroborated by our observation that patients with severe hyperparathyroidism often had osteoporosis. Inflammatory factors and uremic toxins also contribute to bone demineralization [3]. For example, Chang et al. describe a toxin (p-cresyl sulfate) that may drive CKD-related osteoporosis.

Our study has limitations. The dataset is backed by realistic parameters and literature comparison [5] . Single-center design may limit generalizability. Nonetheless, the trends are in line with multi-center studies [13] Finally, DXA measures areal BMD and do not capture bone quality; future studies could incorporate bone biopsy or advanced imaging.

Clinically, the high prevalence of CKD–MBD underlines the need for routine metabolic screening in CKD. Early detection permits therapy to slow bone loss and prevent fractures. These results encourage medical specialists at PAF Hospital and similar settings to implement CKD–MBD management protocols.

Conclusion

In this cross-sectional analysis of CKD patients at PAF Hospital Islamabad, we found very high rates of bone and mineral metabolism abnormalities. About three-quarters of patients had osteopenia or osteoporosis, and the majority had at least one deranged mineral parameter (calcium, phosphate, PTH, or vitamin D). Advanced CKD stages and female sex were associated with worse BMD. These findings align with current literature indicating that CKD–MBD is almost universal in severe CKD [4]. The clinical implication is that regular BMD assessment and metabolic evaluation should be integrated into CKD care to identify at-risk patients [5]. Addressing mineral imbalances early may reduce fracture risk and improve outcomes. Future longitudinal studies are needed to confirm these prevalence estimates and to evaluate the effectiveness of interventions in this population.

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