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Evaluation of Bone Disease in Chronic Kidney Disease and Post-Transplant Patients Using Dual X-Ray Absorptiometry and Bone Biomarkers – A Single-Center Study

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ABSTRACT

Background: Chronic kidney disease-mineral and bone disorder (CKD-MBD) is a common complication in CKD patients and often persists after renal transplantation. Dual-energy X-ray absorptiometry (DXA) assesses bone mineral density (BMD), while bone biomarkers provide insight into bone turnover. This study evaluates bone disease in CKD and post-transplant patients using DXA and bone biomarkers to assess changes in BMD and metabolic bone parameters post-transplantation.

Methods: This observational study included CKD patients and post-transplant recipients. BMD was measured at the spine, femur, and radius using DXA. Bone turnover markers, including parathyroid hormone (PTH), vitamin D3, calcium, phosphorus, and osteocalcin, were analyzed. Comparative statistical analysis was performed to assess differences between CKD and post-transplant groups.

Results: Mean BMD at the femur and radius did not differ significantly between CKD and post-transplant patients ($p > 0.05$). Osteopenia was the most prevalent bone abnormality in both groups. No significant correlation was found between calcium, phosphorus, and PTH levels, but PTH and osteocalcin were significantly associated ($p = 0.02$). A significant relationship was also found between BMD at the radius and T-score at the spine ($p = 0.008$).

Conclusion: Bone disease persists in CKD and post-transplant patients, highlighting the need for long-term monitoring and tailored interventions.

Keywords: Chronic kidney disease, renal transplantation, bone mineral density, DXA scan, bone biomarkers, parathyroid hormone, vitamin D3, osteocalcin

INTRODUCTION

Chronic kidney disease (CKD) includes a spectrum of bone disorders collectively known as CKD-mineral and bone disorder (CKD-MBD), which contributes to increased fracture risk and cardiovascular morbidity. CKD-MBD results from disturbances in calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolism, resulting in abnormalities affecting bone turnover, volume and mineralisation.[1] Renal transplantation (RT) is

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considered the optimal management for end-stage renal disease (ESRD), yet post-transplant bone disease remains a major problem due to persistent metabolic abnormalities, glucocorticoid exposure, and immunosuppressive therapy.[2] Bone mineral density (BMD) assessment using dual-energy X-ray absorptiometry (DXA) is the widely utilized modality to evaluate bone health in CKD and post-transplant patients. However, DXA alone does not provide a complete picture of bone quality, necessitating the use of bone biomarkers such as PTH, osteocalcin, and vitamin D3 for a comprehensive evaluation.[3] Several studies have highlighted that post-transplantation bone disease manifests as osteoporosis and osteopenia, often due to pre-existing bone loss exacerbated by immunosuppressive agents such as glucocorticoids and calcineurin inhibitors.[4] Despite improvements in transplantation outcomes, the management of post-transplant bone disease remains a challenge, with conflicting reports on whether BMD significantly improves after renal transplantation.[5] Given the increasing longevity of transplant recipients, understanding bone health changes in CKD and post-transplant populations is crucial. This study aims to study bone disease using DXA and bone biomarkers in CKD and post-transplant patients to assess the impact of renal transplantation on bone metabolism.

MATERIALS AND METHODS

This single-center observational study was conducted at a tertiary care hospital. CKD Patients with stages 3-5 and those who had undergone renal transplantation at least six months prior were enrolled. The study was approved by the institutional ethics committee, and informed consent was obtained from all participants.

Eligibility Criteria: This study included adult patients over 18 years of age diagnosed with CKD stages 3 to 5. Patients who had undergone renal transplantation at least six months prior were also eligible. Additionally, participants were required to be willing to undergo a DXA scan and biochemical evaluation.

Patients with primary bone disorders or metabolic bone disease unrelated to CKD were excluded. Those receiving bisphosphonates or other bone-modifying agents were also not eligible. Furthermore, individuals with uncontrolled diabetes, malignancies, or other significant comorbidities affecting bone metabolism were excluded from the study.

BMD was measured using DXA scan at the lumbar spine, femur, and distal radius. The results were expressed as BMD values (g/cm²) and T-scores. Based on WHO criteria, osteoporosis was defined as a T-score $\leq$ -2.5, and osteopenia as a T-score between -1.0 and -2.5. Bone biomarkers, including serum calcium, phosphorus, PTH, 25-hydroxyvitamin D3, and osteocalcin, were measured using chemiluminescent immunoassays. Correlations between biochemical parameters and BMD were analyzed using regression models. Statistical analysis was performed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation (SD). Student's t-test and Mann-Whitney U test were used for group comparisons. Pearson's correlation coefficient was used to evaluate associations between variables. A p-value less than 0.05 was considered statistically significant.

RESULTS

A total of 100 participants were included in the study, with 50 patients in the CKD group and 50 in the post-transplant group. Bone Mineral Density (BMD), T-score, and Z-score values were obtained using DXA scans at three skeletal sites: the femur, lumbar spine, and radius. The results are presented in the following tables.

Table 1: Bone Mineral Density (BMD) Comparison

Site	CKD Group (n=50)	Post-Transplant Group (n=50)	p-value
Femur (g/cm²)	0.82 ± 0.09 (0.46 - 0.99)	0.82 ± 0.10 (0.46 - 0.99)	0.87
Radius (g/cm²)	0.46 ± 0.11 (0.24 - 0.69)	0.49 ± 0.10 (0.24 - 0.69)	0.31

Table 2: T-score Comparison

Site	CKD Group (n=50)	Post-Transplant Group (n=50)	p-value
Lumbar Spine	-1.06 ± 1.62 (2.9 - -3.9)	-1.71 ± 1.04 (0.6 - -3.8)	0.07
Femur Neck	-1.27 ± 1.04 (1.6 - -3.9)	-1.15 ± 1.32 (1.1 - -3.9)	0.65
Radius	-1.96 ± 1.55 (2.2 - -6.2)	-2.13 ± 1.87 (2.6 - -6.2)	0.65

T-scores at the lumbar spine, femur neck, and radius were compared between groups. Although a trend toward lower T-scores in post-transplant patients was noted, the differences were not statistically significant. (Table 2)

Table 3: Z-score Comparison

Site	CKD Group (n=50)	Post-Transplant Group (n=50)	p-value
Lumbar Spine	-0.41 ± 1.68 (4.19 - -3.4)	-0.87 ± 1.45 (1.7 - -2.7)	0.22
Femur Neck	-0.69 ± 1.04 (2.3 - -3.0)	-0.70 ± 1.24 (2.3 - -3.2)	0.97
Radius	-1.89 ± 1.35 (0.4 - -5.3)	-2.22 ± 1.44 (0.4 - -5.3)	0.30

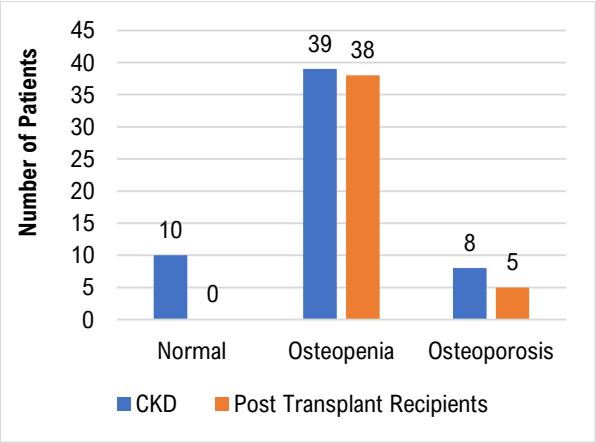

Fig 1: T score at spine in CKD & post-transplant cases

The mean BMD values at the femur and radius were similar between CKD and post-transplant patients, with no significant difference observed ($p > 0.05$). (Table 1)

Z-score values were evaluated to assess deviations from age- and sex-matched normative data. No significant difference in Z-scores was observed between CKD and post-transplant groups at any of the skeletal sites. (Table 3)

Osteopenia was the most common bone abnormality in both groups, affecting 39 CKD patients and 38 post-transplant patients. (Fig 1)

The analysis of bone metabolism and mineral homeostasis in CKD and post-transplant patients revealed several key findings. There was no significant relationship between serum calcium and phosphorus levels ($p > 0.05$), suggesting that phosphate regulation mechanisms, though altered in CKD, do not drastically shift post-transplant. Similarly, the relationship between serum calcium and PTH levels ($p = 0.63$) and serum Vitamin D3 and PTH levels ($p = 0.11$) remained statistically insignificant, indicating persistent dysregulation of parathyroid function despite transplantation. (Table 5)

Additionally, the correlation between serum Vitamin D3 and calcium levels ($p = 0.11$) was insignificant, reflecting the complex interplay between vitamin D metabolism and calcium homeostasis in renal patients. However, a statistically significant association ($p = 0.02$) was observed between PTH and osteocalcin levels, indicating increased bone turnover linked to parathyroid activity. This suggests that secondary hyperparathyroidism in CKD continues to influence bone remodelling, even after transplantation. Furthermore, the association between BMD at the radius and the T-score at the spine was significant ($p = 0.008$), indicating that reduced bone mineral density at peripheral sites corresponds with decreased bone strength at weight-bearing sites. (Table 5) This highlights the importance of monitoring systemic bone loss in both CKD and post-transplant patients, as osteoporosis risk remains high despite renal replacement therapy.

Table 5: Relationship Between Biochemical Parameters and BMD in CKD and Post-Transplant Groups

Parameter Comparison	CKD Group (Mean ± SD)	Post-Transplant Group (Mean ± SD)	p-value
Serum Calcium (mg/dL) vs. Phosphorus (mg/dL)	8.6 ± 1.1 vs. 4.5 ± 0.9	8.8 ± 1.2 vs. 4.4 ± 0.8	0.07
Serum Calcium (mg/dL) vs. PTH (pg/mL)	8.6 ± 1.1 vs. 135 ± 40	8.8 ± 1.2 vs. 128 ± 35	0.63
Serum Vit D3 (ng/mL) vs. PTH (pg/mL)	22.3 ± 5.4 vs. 135 ± 40	24.1 ± 6.1 vs. 128 ± 35	0.11
Serum Vit D3 (ng/mL) vs. Calcium (mg/dL)	22.3 ± 5.4 vs. 8.6 ± 1.1	24.1 ± 6.1 vs. 8.8 ± 1.2	0.11
Serum PTH (pg/mL) vs. Osteocalcin (ng/mL)	135 ± 40 vs. 28.6 ± 5.2	128 ± 35 vs. 32.2 ± 6.1	0.02*
BMD at Radius (g/cm²) vs. T-score at Spine	0.46 ± 0.11 vs. -1.06 ± 1.62	0.49 ± 0.10 vs. -1.71 ± 1.04	0.008*

*Statistically significant association.

DISCUSSION

This study evaluated bone mineral density (BMD) using Dual X-ray Absorptiometry (DXA) in patients with chronic kidney disease (CKD) and post-renal transplant recipient patients. The results were compared to multiple contemporary studies, emphasizing the role of DXA in detecting osteoporosis and osteopenia in this patient population.

In this study, there was no significant difference in BMD values at the femur, radius, or spine between CKD and post-transplant patients. This finding is consistent with previous studies that have reported persistent bone mineral deficits even after successful renal transplantation.[1,2] A study by Sharma et al. found that BMD remained low in transplant recipients despite normalization of renal function, suggesting that factors beyond kidney function, such as persistent hyperparathyroidism or immunosuppressive therapy, contribute to ongoing bone loss.[3]

DXA scan data from our study indicate that osteopenia was the most prevalent bone disorder in both CKD and post-transplant patients, particularly at the femur and spine. These findings are similiar with those of Iyer et al., who reported high rates of osteopenia (79%) and osteoporosis (21%) in renal transplant recipients.[4] In contrast, other studies, such as that by Gonzalez et al., noted a greater prevalence of osteoporosis post-transplantation due to cumulative steroid exposure.[5] A major point of comparison is the regional variation in BMD changes. In our study, the mean BMD at the radius in CKD cases was 0.46 g/cm², while in post-transplant patients, it was 0.49 g/cm². Although not statistically significant ($p=0.31$), this trend aligns with findings by Malluche et al., who suggested that cortical bone loss is more pronounced in CKD patients, whereas post-transplant bone disease primarily affects trabecular sites such as the spine.[6] However, our study did not demonstrate a significant worsening of trabecular BMD

post-transplant, which differs from the findings of Kwon et al., who documented a marked decline in spine BMD following transplantation.[7]

This study found no significant correlation between serum calcium and phosphorus levels, which is in agreement with the results of a large cohort study by Nickolas et al. showing that biochemical markers alone are insufficient in predicting BMD loss in CKD patients.[8] However, we observed a significant relationship between PTH and osteocalcin levels (p=0.02), reinforcing the notion that secondary hyperparathyroidism is a key player in CKD-associated bone disease, as previously stated by Jørgensen et al.[9]

Regarding the effect of immunosuppressive therapy, several studies have identified glucocorticoid-induced osteoporosis as a major complication post-transplantation. For example, a study by Silva et al. found that patients receiving high doses of corticosteroids post-transplant exhibited a significant reduction in DXA scan values at the spine and femoral neck within the first six months.[10] However, this study did not find a significant difference in BMD values between CKD and post-transplant groups, likely due to differences in corticosteroid tapering protocols and newer immunosuppressive regimens that minimize bone loss.

Overall, these findings highlight the importance of DXA scans in monitoring bone health in both CKD and post-transplant patients. While transplantation may stabilize renal function, it does not completely reverse bone mineral deficits, necessitating long-term monitoring and intervention strategies such as vitamin D supplementation, bisphosphonates, and reduction in steroid exposure where feasible.

CONCLUSION

Our study demonstrates that BMD, as assessed by DXA, remains largely unchanged after renal transplantation, with osteopenia being the most prevalent bone disorder in both CKD and post-transplant patients. Despite improvements in renal function, transplant recipients remain at risk for ongoing bone loss, likely due to persistent hyperparathyroidism and immunosuppressive therapy. These findings underscore the importance of routine DXA screening and early interventions to mitigate fracture risk in this population. Further longitudinal studies with larger sample sizes are required for better understanding of the long-term effects of renal transplantation on bone health.

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Author Contribution: GV contributed to the study conception and design. **NR** was responsible for data collection, data analysis and interpretation. **AK** played a key role in manuscript preparation.

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