

Original Article

Evaluation of the fibroblast growth factor-23 and bone minerals in renal transplant recipients: A follow-up study

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Received: 21 October 2024

Accepted: 24 November 2025

Epub Ahead of Print: 14 January 2026

Published:

DOI

10.25259/JLP_275_2024

Quick Response Code:



ABSTRACT

Objectives: The objective of the study is to assess fibroblast growth factor-23 (FGF23) and its association with bone minerals in renal transplant recipients.

Materials and Methods: A prospective longitudinal study comprising 32 end-stage renal disease cases awaiting renal transplant was followed up for three months post-transplant.

Statistical analysis: Data were analyzed using appropriate tests such as the Friedman test, Pearson correlation with Statistical Package for the Social Sciences 20, and GraphPad Prism 7. $P < 0.05$ was considered statistically significant.

Results: A significant difference was noted in fibroblast growth factor 23 (FGF23) levels between pre and post-transplantation samples, with an increasing trend until 1 month, and a decreasing 1 by 3 months post-transplantation. Parathyroid hormone (PTH) levels reached normal ranges immediately post-transplant, and further decreased to lower normal ranges by three months. Vitamin D levels increased in the 1st week and stabilized thereafter. Glomerular filtration rate (GFR) increased post-transplant immediately and stabilized in the following months. Calcium levels improved by 1 month post-transplant and stabilized thereafter. Phosphorus decreased in the immediate post-transplant period, and then a slight increase was noted. GFR has shown a negative correlation ($r = -0.24$, $P < 0.01$) with FGF23 when pre and post-transplant samples were considered together. Phosphorus showed a positive correlation ($r = 0.3$, $P < 0.001$) with FGF23. Pre-transplant ($r = 0.36$, $P < 0.044$) and 3 months post-transplant ($r = 0.44$, $P = 0.025$), FGF23 levels showed a positive correlation with phosphorus. FGF23 levels increase significantly post-transplantation within three months of stable graft function. An increase in phosphorus levels within the normal range was noted, with a significant positive correlation with FGF23.

Conclusions: Greater information about aberrant FGF23 and its effects on bone minerals is necessary to understand whether efforts to restore it after transplant are desirable.

Keywords: Bone mineral density, Fibroblast growth factor 23, Parathormone, Phosphaturia, Renal transplantation

INTRODUCTION

Chronic kidney disease (CKD) and end-stage renal disease (ESRD) represent significant health problems in developed and developing countries. In India, over 15,000 patients initiate maintenance hemodialysis, and approximately 3000 patients are on continuous ambulatory peritoneal dialysis annually, while 3500 patients receive kidney transplants.^[1]

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ESRD, defined as a glomerular filtration rate (GFR) of less than 15 ml/min/1.73 sq.m., is the major indication for kidney transplantation.

Bone disease is a major cause of morbidity in kidney transplant recipients following transplantation. It presents with a significantly higher risk of fractures because of changes in bone quality and density, alterations in mineral metabolism, thus increasing healthcare costs and morbidity. Bone changes can be attributed to a variety of causes, such as immunosuppression and alterations in the PTH-Vitamin D-fibroblast growth factor 23 (FGF23) axis, as well as changes in mineral metabolism.

Osteocytes mainly produce FGF23. It has a key role to play in the bone-kidney axis with a strong control on calcium and phosphate metabolism. It downregulates sodium-phosphate co-transporters in renal proximal tubules, resulting in phosphaturia. It also decreases Vitamin D levels by inhibition of 25(OH)D-1 α -hydroxylase, and upregulation of the catabolic 25(OH)D-24-hydroxylase pathway. It also suppresses PTH synthesis. Thus, it evaluates the variations in mineral metabolism before and after renal transplantation. Information regarding chronic kidney disease and bone mineral disorders in the population who undergo renal transplantation in India is scarce. This study aims to describe such bone mineral anomalies in renal transplant recipients.

MATERIALS AND METHODS

This is a prospective longitudinal study comprising 32 cases of ESRD, conducted in the department of Biochemistry in collaboration with the department of Nephrology at Nizam's Institute of Medical Sciences (NIMS), Hyderabad, India. A total of 64 subjects (32 cases who underwent renal transplantation and 32 age and gender-matched controls) were included in the study. All the transplants were live donor types, with the majority of them receiving the graft from their parents or siblings. Chronic glomerulonephritis, diabetes, and hypertension were the most common causes leading to ESRD in our group of patients. Immunosuppression was induced and maintained by tacrolimus, mycomofetil sodium, and solumedrol. The recipients were followed up for 3 months after the transplant.

Patients with a history of any neoplasms who did not provide informed consent were excluded. This was approved by the Institutional Ethical Committee (EC/NIMS/2133/2018), and written informed consent was obtained. A volume of 5 mL of blood was drawn at pre-transplant, 1st week, 1st month, 3rd month post-transplant. Part of the serum was aliquoted and stored at -40°C for subsequent serum FGF23 measurement by sandwich enzyme-linked immunosorbent assay (KRISHGEN BIOSYSTEMS, CA, USA). Serum PTH, Vitamin D-total (ADVIA CENTAUR), calcium, phosphorus,

urea, and creatinine (COBAS C501) were analyzed using dedicated reagents. Statistical analysis was done by Microsoft Excel, Statistical Package for the Social Sciences 20, and Graphpad Prism 7. Data are presented as mean \pm SD and median with inter-quartile ranges. The Mann-Whitney U test, Freidman's test, and Spearman correlation analysis were applied as needed. $P < 0.05$ is considered statistically significant.

RESULTS

Among 32 cases, 7 were female, and 25 were male. 32 age and gender-matched controls were evaluated.

Significantly low levels were noted in GFR, calcium, and phosphorus in cases as compared to controls. Statistically higher values were noted in levels of urea, creatinine, and FGF23 in cases when compared to controls [Table 1].

A significant difference was noted in GFR, PTH, phosphorus, calcium, and FGF23 levels when pre-transplant, 1st week, 1st month, 3rd month samples were compared using Freidman's test [Table 2].

A significant difference was also noted in FGF23 levels when pre-transplant, 1st week, 1st month, 3rd month samples were compared, $P < 0.0024$, with an increasing trend till 1st month after post-transplant and decreasing by 3rd month [Figure 1]. FGF23 has shown an increasing trend until 1 month post-transplant and then declined later [Figure 2].

PTH levels have come down to normal ranges immediately after transplant and further decreased to lower normal ranges

Table 1: Baseline characteristics of controls and cases.

Variable	Controls (n=32)	Cases (n=32) Pre-transplant	P-value
Age (years)	30.1 \pm 6.3	29.9 \pm 7.5	0.99
Urea (mg/dL)	16.5 (11.25-20.75)	54 (41.7-74)	<0.001*
Creatinine (mg/dL)	0.8 (0.6-1.0)	5.7 (4.7-6.7)	<0.001*
GFR (mL/min) [†]	123.5 (101.8-136.5)	12.15 (8.9-15.9)	<0.001*
FGF23 (pg/mL) [‡]	376 (312-476.5)	372 (296.8-743.3)	0.0276*
Vitamin-D (ng/mL)	33.5 (27.2-46.5)	31 (20-54)	0.554
PTH (pg/mL) [§]	39.5 (25.5-54)	163 (110.5-252)	<0.001*
Calcium (mg/dL)	9.7 \pm 0.84	8.4 \pm 0.94	<0.001*
Phosphorus (mg/dL)	4.0 \pm 0.63	4.16 \pm 1.4	0.691

[†]GFR: Glomerular filtration rate, [‡]FGF23: Fibroblast growth factor-23,

[§]PTH: Parathormone. * $P < 0.05$ is taken as statistically significant for the Freidman test, and Pearson correlation.

Table 2: Summary of variables before renal transplantation, 1st week, 1 month, and 3rd month.

Biomarker	Controls	Pre-transplant Median (IQR)	1st week Median (IQR)	1st month Median (IQR)	3rd month Median (IQR)	P-value
GFR [†] (mL/min)	123.5 (101.8-136.5)	12.1 (8.9-15.9)	78 (64-113.3)	76 (55.5-106.5)	70.8 (55.5-106.5)	<0.001*
Vitamin-D (ng/mL)	33.5 (27.2-46.5)	31 (20-54)	48 (29.7-66.7)	47 (34-65)	41.5 (34.5-57.2)	0.1605
PTH [‡] (pg/mL)	39.5 (25.5-54)	163 (110.5-252)	65.5 (37.5-117.5)	41.5 (13.5-72.7)	19 (10.5-34.7)	<0.001*
Phosphorus (mg/dL)	4 (3.7-4.3)	4.1 (3.1-4.9)	3.1 (2.1-4.0)	3.4 (2.7-3.7)	3.5 (3.1-3.8)	0.0023
Calcium (mg/dL)	9.6 (9-10.1)	8.7 (7.7-9.1)	8.4 (8.1-8.7)	9.4 (9.1-9.7)	9.8 (9.3-10.1)	<0.001*
FGF23 [§] (pg/mL)	376.5 (312.8-476.5)	372 (296.8-743.3)	442 (382.8-1150)	502.5 (403.8-905)	477 (419.5-766)	0.0024*

[†]GFR: Glomerular filtration rate, [‡]PTH: Parathormone, [§]FGF23: Fibroblast growth factor-23, IQR: interquartile range. **p*<0.05 is taken as significant for the Freidman test, and Pearson correlation

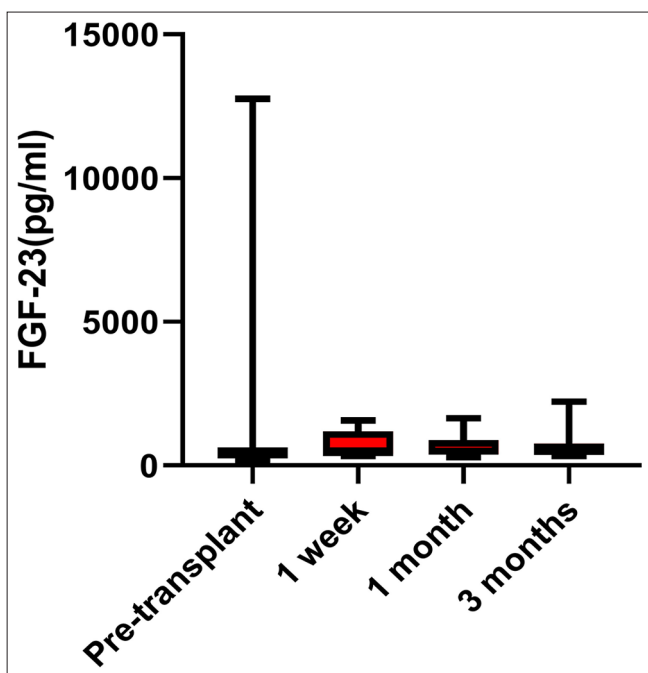


Figure 1: Comparison of fibroblast growth factor 23 (FGF-23) levels of cases before renal transplantation, 1 week, 1 month, and 3 months post-transplant. A significant difference was noted in FGF23 levels when pre-transplant, 1 week, 1 month, and 3 months post-transplant samples were compared, *P* < 0.0024.

by 3rd month. Vitamin D levels have shown an increase in the 1 week and stabilized thereafter. GFR increased immediately post-transplant and stabilized in the following months [Figure 3].

Calcium levels improved by 1st month post-operatively and stabilized thereafter, and phosphorus decreased in the immediate post-operative period. A slight increase was noted after the first week, which was significant [Figure 4].

GFR has shown a significant negative correlation (*r* = -0.24, *P* < 0.01), i.e., as FGF23 increased over follow-up, GFR decreased when pre and post-transplant samples were

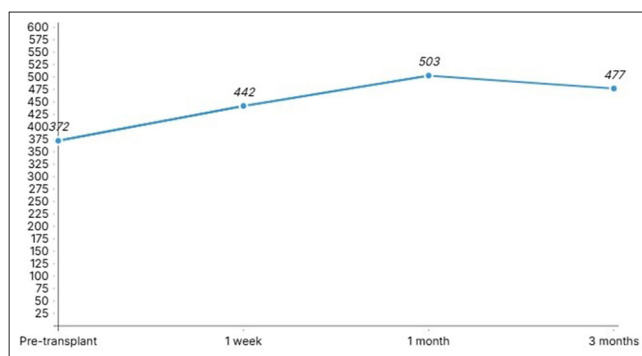


Figure 2: Pre-transplant to 3 months post-transplant trend of fibroblast growth factor 23 (FGF-23). FGF23 has shown an increasing trend until 1 month post-transplant and then declined later.

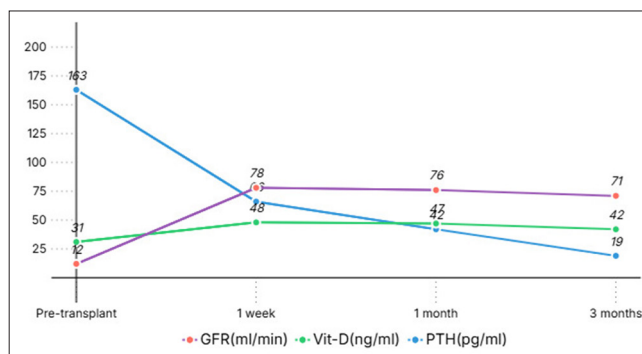


Figure 3: Pre-transplant to 3 months trend of parathyroid hormone (PTH), Vitamin D (Vit-D), Glomerular filtration rate (GFR), PTH levels and GFR have come to normal ranges immediately after transplant; PTH further decreased to lower normal ranges by 3 months while GFR stabilized. Vit-D levels have shown an increase in 1 week and have been maintained in normal ranges thereafter.

correlated together. Similarly, phosphorus has shown a significant positive correlation (*r* = 0.3, *P* < 0.001), i.e., as FGF23 increased during follow-up, phosphorus levels increased too [Table 3].

DISCUSSION

Bone abnormalities following transplantation are significant complications in the majority of patients, although the illness course varies by case. Immunosuppression, impaired kidney function, and other potentially morbid factors can appear after transplantation.^[2] Several significant alterations in bone mineral physiology occur after transplantation. However, it is crucial to understand that the relationship between changes in mineral metabolism and graft/patient outcomes is associative and not necessarily adverse.

FGF23, a phosphaturic hormone, provides a different outlook on understanding the etiology of post-transplant bone disease. Hyperparathyroidism in CKD, when treated with high-dose Vitamin D (a potent regulator of FGF23 gene transcription) analogs, may lead to further elevation of FGF23 levels. Finally, increased levels can persist even after renal replacement by renal transplantation in some cases.^[3]

FGF23 has a crucial role to play in secondary hyperparathyroidism (SHPT) of patients with CKD.^[4-7] Residual FGF23 activity after transplantation may lead to

early post-transplant hypophosphatemia, which could in turn contribute to the pathogenesis of post-transplant bone disorders.^[8,9] Similar to previous observations made in patients with CKD, FGF23 values were elevated in most transplant recipients of our study group (72%).^[5,8,10] A significant inverse correlation ($r = -0.2, P < 0.01$) was noted between GFR and FGF23 levels in this group, which could be attributed to poorer kidney function. The median FGF23 (372 pg/mL) levels in our cases were above the normal range. However, they were lower than those of patients with similar eGFR who did not undergo transplant.^[11] To a similar observation made by Wolf *et al.*^[12], 28% of our cases had decreased FGF23 [Table 4]. Reports on the early post-transplant period (3-12 months) summarize a marked decrease in FGF23 levels, with mean values very close to normal at the end of follow-up.^[7-13] However, we found high levels of FGF23 in the early post-transplant interval.

The previous chronic phosphate retention could have stimulated aberrant FGF23 production in our recipients. Several mouse models explain that the association between bone turnover and serum FGF23 is variable and dependent on a variety of exogenous and endogenous factors.^[14] In addition to renal function, factors such as increased cumulative doses of corticosteroids, PTH, serum phosphate levels, and decreased serum calcitriol were associated with excess FGF23.^[15] Finally, previous bone abnormalities and immunosuppressive drugs in the post-operative period should also be considered, as they can affect bone turnover.

Phosphorus levels increased in 32% of instances, with a significant positive connection with FGF23 [Table 4]. Isakova *et al.*^[6] found that the elevation of serum phosphate, even within normal ranges, can stimulate FGF23 production. Various workers have also reported similar observations in patients with CKD who did not undergo transplantation.^[14-16]

Controversy still exists over whether the phosphorus losses after transplantation persist in the long term. According to a study, possible explanations for FGF23's apparent lack of action on phosphate reabsorption include a lack of

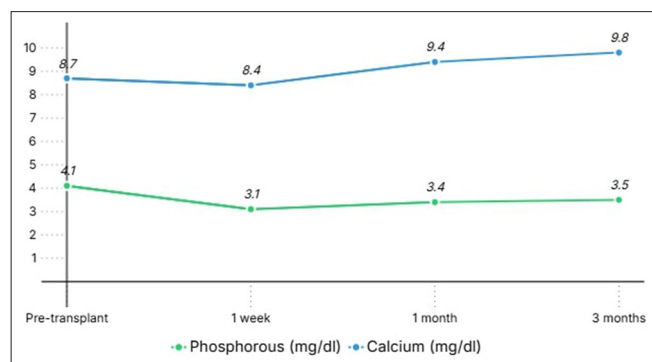


Figure 4: Pre-transplant to 3 months post-transplant trend of calcium and phosphorus. Calcium levels improved by 1 month post-operatively and were in normal ranges thereafter. Phosphorus decreased in the immediate post-operative period; however, statistically significant elevation of phosphorus levels was noted after 1 month.

Table 3: Correlation of FGF23 with other variables in all cases combined.

Variable	Spearman correlation (ρ)	P-value
GFR (mL/min)†	-0.24	0.01*
Calcium (mg/dL)	-0.05	0.597
Phosphorus (mg/dL)	0.3	0.001*
Vitamin-D (ng/mL)	-0.09	0.325
PTH (pg/mL)‡	0.15	0.103

†GFR: Glomerular filtration rate, ‡PTH: Parathormone, FGF23: Fibroblast growth factor-23. Spearman correlation (ρ) analysis was done as the data is non-parametric. * $P < 0.05$ is taken as significant

Table 4: Percentage of cases showing an increase or decrease of various analytes when compared between the pre-transplant and post-transplant period.

Variable	Increased (%)	Decreased (%)
FGF23 [†]	72	28
PTH [‡]	0	100
Vit-D	50	50
Calcium	50	50
Phosphorous	32	68

[†]FGF23: Fibroblast growth factor-23, [‡]PTH: Parathormone

or resistance to Klotho (the single-pass transmembrane protein required for FGF23-mediated receptor activation) or interference by immunosuppressive agents with Klotho.^[17]

Parathormone

We noticed that PTH levels were very high in the pre-transplant period (SHPT in CKD), which corrected to normal ranges immediately during the post-operative period [Table 4]. These levels went on to further decrease to lower normal ranges. Median PTH levels in the pre-transplant period, 1st week, 1st month, 3rd month post-transplant period were 163, 66, 42, and 19 pg/mL, respectively. CKD patients before transplantation with an elevated PTH and a certain degree of parathyroid gland hyperplasia could explain this pronounced effect of the hormone. Hence, our data support the theory of PTH having a synergistic effect with other hormones to induce renal phosphate excretion in patients with CKD.

Calcium

Hyperphosphatemia and hypocalcemia are inevitable with a reduction in the GFR. In the current observation, pre-transplant period and 1st week, 1st month, 3rd month post-transplant, levels of serum calcium were 8.7, 8.4, 9.4, and 9.8 mg/dL, respectively, which indicates an increasing trend. The increased levels of calcium (50% of cases), however, were still within the normal range [Table 4]. Hypercalcemia, usually of a mild degree and without clinical consequences, is one of the most frequent findings in transplant recipients. Massari observed that transplant patients developed hypercalcemia within the first three months of transplantation. This was self-limited in every case, and it resolved spontaneously before the first year after transplantation.^[18] Similarly, our results have shown a gradual increase in calcium levels until three months of post-transplant observation. Further follow-up might have revealed any abnormalities in calcium levels.

Vitamin D

The kidneys activate Vitamin D through the enzyme 1α -hydroxylase. Vitamin D insufficiency causes SHPT, which is consistent with our observations. Low Vitamin D levels reduce the expression and activity of Vitamin D receptors in parathyroid cells and increase PTH production.^[19] A prospective study from Iran showed that low Vitamin D levels after 3 months of transplantation were independently associated with a lower GFR and a higher risk for interstitial fibrosis at 12 months.^[20]

There was no correlation between FGF23 and vitamin D levels. This has also been reported by Economidou *et al.*^[21] Vitamin D, an immune modulator, has a role in preventing allograft rejection post-transplantation.^[22-24] Despite a good

graft function after renal transplantation, patients continue to have important biochemical changes. It leads to deterioration of bone quality, particularly hyperparathyroidism and Vitamin D deficiency.

Stavroulopoulos *et al.*^[25] observed that Vitamin-D deficiency in correlation with SHPT was very common in all of their renal transplant recipients. Vitamin D deficiency prevalence has been reported to be quite high at the time of renal transplantation.^[26] GFR and Vitamin-D deficiency/insufficiency dictate PTH levels in renal transplant patients. An optimal correction of hyperparathyroidism and Vitamin D deficiency following transplantation has been desirable.

Vitamin-D levels in the pre-transplant period and 1st week, 1st month, 3rd month post-transplant (31, 48, 47, and 42 ng/dL, respectively) showed an increasing trend (50% of cases) post-transplantation in our study [Table 4]. We also noticed after transplantation, an increasing trend of Vitamin-D levels along with eGFR in our cases, as opposed to observations made by Keyzer *et al.*^[27], where no such pattern was seen. Three months after transplantation, there was an increase in levels of Vitamin D along with a simultaneous decrease in PTH levels in recipients. Various studies on *in vitro* cell cultures indicate that the intracellular activity of Vitamin D might be required to achieve desirable immune responses.^[28,29] Our data reveal a high prevalence of Vitamin D deficiency or insufficiency in patients with CKD, and it persisted even after kidney transplantation in 50% of patients and was strongly associated with SHPT. In our current observations, patients with Vitamin D deficiency had a trend of decreasing eGFR, which could have a deleterious effect on graft outcome.

Our short follow-up design limits us in noticing long-term changes in FGF23, calcitriol, PTH, and bone mineral levels. Successful transplantation corrects most of the mineral and bone variables involved in the development of CKD-associated bone mineral disorders, yet some abnormalities persist. A report by Wolf *et al.*^[12] says that high FGF23 levels are associated with an increased risk of mortality and allograft loss. It has been hypothesized that high FGF23 levels in transplant recipients could reflect the load of pre-transplant vascular calcification, indicating the likelihood of subsequent mortality.^[16] Therefore, factors that stimulate the production of high FGF23 levels should be modified. Measures aimed at controlled dosing of corticosteroids, use of phosphate binders (to regulate phosphate levels), and reducing PTH could help lower FGF23 levels, thereby having a plausibly positive impact on long-term graft survival. Limited data exists on bone histology in transplant recipients, and imaging techniques do not predict fracture risk. According to the Kidney Disease Improving Global Outcomes working group, all patients with CKD should be assessed for mineral, bone, and vascular complications of CKD-mineral and bone

disorder (MBD), and more research is needed to improve skeletal health in this population.

Our study provides a perspective on these conditions in the Indian population. This warrants further long-term study for a better understanding of CKD-MBD and renal transplant-associated bone physiology.

Limitations

A short follow-up term and a small sample size are the major limitations of this study.

CONCLUSIONS

FGF23 levels increase significantly even after successful renal transplantation within three months of stable graft function. Indeed, the biochemical profile of an increase in phosphorus levels within the normal range was noted with a significant positive correlation with FGF23. Vitamin D levels achieved a normal range within a week of transplant and are maintained. Serum calcium levels show an increasing trend within the normal range without any implications. High PTH levels because of SHPT were seen in the pre-transplant period, which normalized after successful transplantation. No significant correlations of FGF23 with calcium, Vitamin D, and PTH are noticed. The ease of estimating FGF23 is useful in the assessment of hypophosphatemia and related mineral abnormalities. Critical analysis of FGF23 and its biological effects at normal and abnormal levels on bone minerals is necessary to understand whether it is desirable to normalize FGF23 levels following successful renal transplants.

Author contributions: PJ, SSBK, NM, SN: Concept and design, data acquisition, data analysis and interpretation, drafting the article, reviewing, final approval of the manuscript, accountable. SBR, RD: data acquisition, reviewing, final approval of the manuscript, accountable.

Ethical approval: The research/study was approved by the Institutional Review Board at Nizam's Institute of Medical Sciences, approval number EC/NIMS/2133/2018, dated 1st March, 2018.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript, and no images were manipulated using AI.

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How to cite this article: Japa P, Kompella S, Mohammed N, Raju S, Neelam S, Devraj R. Evaluation of the fibroblast growth factor-23 and bone minerals in renal transplant recipients: A follow-up study. *J Lab Physicians*. doi: 10.25259/JLP_275_2024