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Association of mineral metabolism biomarkers in patients with chronic kidney disease and renal transplant recipients: A single-centre prospective study

Gaurav Vohra
Doctorate of Medicine, Associate Professor, Surgeon Captain
Institute of Naval Medicine
400704, WW26+FHF Kegaon, Navi Mumbai, India
https://orcid.org/0000-0001-9608-7312

Nagamahendran Rajendran*
Diplomate of National Board, Assistant Professor, Wing Commander
Air Force Hospital
345001, WV5V+7X8, Dhauwa, Kishan Ghat, Rajasthan, India
https://orcid.org/0000-0002-9854-7236

Anju Kumari
Master of Surgery, Consultant Ophthalmologist
Laxmi Eye Hospital
400704, Mulla Hamid Rd., Navi Mumbai, India
https://orcid.org/0009-0006-7447-0277

Abstract. Holistic assessment of mineral bone disorder parameters, including serum calcium, phosphorus, parathyroid hormone, and 1,25-dihydroxyvitamin D, can predict renal outcomes in kidney transplant recipients, though results have varied. This study aimed to evaluate the biochemical parameters of mineral bone disorder in patients with chronic kidney disease and renal transplant recipients. A total of 78 patients with chronic kidney disease, 24 post-renal transplant recipients, and 28 control cases were included, with a mean age of 48.24 years. The predominant cause of chronic kidney disease was chronic glomerulonephritis, followed by diabetes and hypertension. Serum levels of calcium, phosphorus, parathormone, and vitamin D3 were assessed. Patients with chronic kidney disease exhibited a mean serum calcium level of 8.37 mg/dL and phosphorus level of 4.25 mg/dL, while post-transplant patients had mean levels of 8.17 and 4.15 mg/dL, respectively. A significant reduction in serum parathyroid hormone levels was observed post-transplant (mean 8.13 U/dL) compared to patients with chronic kidney disease (mean 24.39 U/dL). No significant changes were noted in vitamin D3 levels after transplantation. Regression analysis revealed an insignificant relationship between serum calcium and phosphorus levels and parathyroid hormone levels. This study highlights the biochemical imbalances associated with chronic kidney disease and the impact of renal transplantation on parathyroid hormone levels, emphasising the need for regular monitoring and management of mineral bone disorder parameters in these patient populations

Keywords: biomolecular indicators; mineral bone disorder; hormonal imbalance in kidney disease; 1,25-dihydroxyvitamin D

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*Corresponding author

Introduction

Chronic kidney disease (CKD) is a multifactorial disease complex and one of the most prevalent conditions, as noted by L. Hu *et al.* [1]. CKD is characterised by abnormal kidney function and a progressive decline in glomerular filtration rate (GFR), leading to systemic complications. These complications include anaemia, fatigue, weight and muscle loss, and disturbances in calcium, phosphorus, and hormone levels, particularly calcitriol and parathyroid hormone (PTH). According to S. Fernández-Villabrille *et al.* [2], bone is a metabolically active tissue undergoing continuous remodelling, orchestrated by the dynamic interplay between osteoblast and osteoclast cells. These cellular processes are modulated by a complex interaction of biochemical and mechanical factors, which are instrumental in assessing bone remodelling. CKD is associated with the development of mineral bone disorder (MBD), osteoporosis, and fragility fractures, as noted by CY Hsu *et al.* [3]. Early in CKD (GFR < 60 mL/min/1.73 m^2), there is an increase in serum phosphorus and a decrease in 1,25-dihydroxyvitamin D3 (1,25$(OH)_2$D), resulting in hypocalcaemia and elevated PTH levels. According to T. Liyanage *et al.* [4], this compensatory mechanism aims to increase phosphates excretion and normalise serum calcium levels. Secondary hyperparathyroidism in CKD primarily results from reduced 1,25$(OH)_2$D or hyperphosphataemia.

According to U.G. Hasparyk *et al.* [5], renal transplantation can rectify many metabolic disturbances by normalising phosphates excretion and restoring renal calcitriol production. This process results in reductions in plasma phosphates, PTH, and alkaline phosphates levels, as well as the mobilisation of soft tissue calcifications. The main biochemical abnormalities of mineral metabolism in kidney transplantation (KTx) include hypophosphataemia, hyperparathyroidism (HPTH), vitamin D insufficiency or deficiency, and hypercalcaemia. However, certain abnormalities, such as hyperparathyroidism and osteopenia, may persist following transplantation. Renal osteodystrophy, a common complication of CKD, manifests in various forms, including osteitis fibrosa cystica (OFC), adynamic bone disease, osteomalacia, and mixed osteodystrophy, with OFC and mixed osteodystrophy being the most prevalent [6]. Hyperphosphataemia is also common and inadequately managed in CKD stage 5 [7].

Existing studies have shown variability in the assessment of mineral metabolism parameters, including serum calcium, phosphorus, parathyroid hormone (PTH), and vitamin D levels, with differing results depending on study populations, stages of CKD, and post-transplant conditions. Some studies suggest significant post-transplant improvements in mineral metabolism, particularly PTH levels, while others indicate persistent abnormalities despite graft function. This inconsistency emphasises the need for further research to establish a clearer understanding of the pathophysiological changes and clinical implications of mineral and bone disorders in both CKD patients and renal allograft recipients. This study, conducted at a tertiary care centre, aimed to investigate the association of mineral metabolism biomarkers in patients with CKD and renal transplant recipients, utilising these noninvasive markers, and to compare the findings with global literature.

Materials and Methods

This prospective study was conducted over a 24-month period (June 2021 to June 2023) at a large tertiary care hospital in a cosmopolitan city in India. Approval was obtained from the Hospital Ethics Committee (Approval No. INM/06/07), and informed consent was secured from all participants prior to their inclusion in the study. A total of 130 participants were enrolled, consisting of 78 patients with chronic kidney disease (CKD), 24 renal transplant recipients, and 28 controls. Patients with CKD were aged 22 to 70 years, with a mean age of 46 years, and a male-to-female sex ratio of 2:1 (Table 1). Demographic characteristics, including age, sex, and relevant medical history such as underlying CKD etiology, disease duration, and comorbidities (e.g., hypertension and diabetes mellitus), were collected at baseline through structured patient interviews and a review of medical records. This information was verified against hospital records and entered into a secure database for analysis.

Table 1. Sex distribution of CKD, renal transplant, and control cases

	Females	Males	Total
CKD	25	53	78
Post-renal transplant	9	15	24
Controls	10	18	28

Source: compiled by the authors

Patients aged 20 to 80 years with CKD stages 3 to 5, including those on maintenance haemodialysis (HD), and post-renal transplant recipients with stable graft function for at least 6 months were included in the study. Exclusion criteria comprised patients with acute kidney injury (AKI), recent infections, or those undergoing treatment with medications affecting mineral metabolism, such as bisphosphonates or calcimimetics. The sample size of 130 participants was determined through power analysis using a significance level (α) of 0.05 and a power of 80%, ensuring the detection of meaningful differences in mineral metabolism parameters between study groups.

Laboratory parameters related to mineral metabolism were analysed, including serum calcium, phosphorus,

1,25-dihydroxycholecalciferol (Vitamin D3), and intact parathyroid hormone (iPTH) levels. Serum calcium and phosphorus were measured using an automated colorimetric method, 1,25-dihydroxycholecalciferol levels were assessed using radioimmunoassay, and intact PTH levels were quantified using a two-site chemiluminescent immunoassay (CLIA). All analyses were performed using SPSS software (version 22.0). Data were tested for normality using the Shapiro-Wilk test. Normally distributed continuous variables were compared using independent t-tests, while non-normally distributed data were analysed using the Mann-Whitney U-test. Categorical variables were compared using the chi-square test. Regression analysis was performed to explore the relationships among serum calcium, phosphorus, Vitamin D3, and PTH levels, with dependent and independent variables clearly defined. Correlation analyses included Pearson's correlation for normally distributed data and Spearman's correlation for non-normally distributed data. Statistical significance was set at $p < 0.05$.

Results

Renal transplant patients were aged 20 to 65 years, with a mean age of 42.5 years, and 62.5% were male, resulting in a sex ratio of 1.5:1. The mean age of all patients studied was 48.24 years, with a standard deviation of 14.13 years. For CKD patients, the mean age was 51.59 years (SD 12.82), and for transplant patients, it was 37.33 years (SD 12.82) (Table 2; Fig. 1).

Table 2. Age distribution of CKD, renal transplant, and control cases

Age (Years)	CKD	Renal transplant	Controls	Total
20-29	06	06	02	14
30-39	09	10	15	34
40-49	15	04	08	27
50-59	19	02	03	24
60-69	28	02	00	30
70-80	01	00	00	01
Grand total	78	24	28	130

Source: compiled by the authors

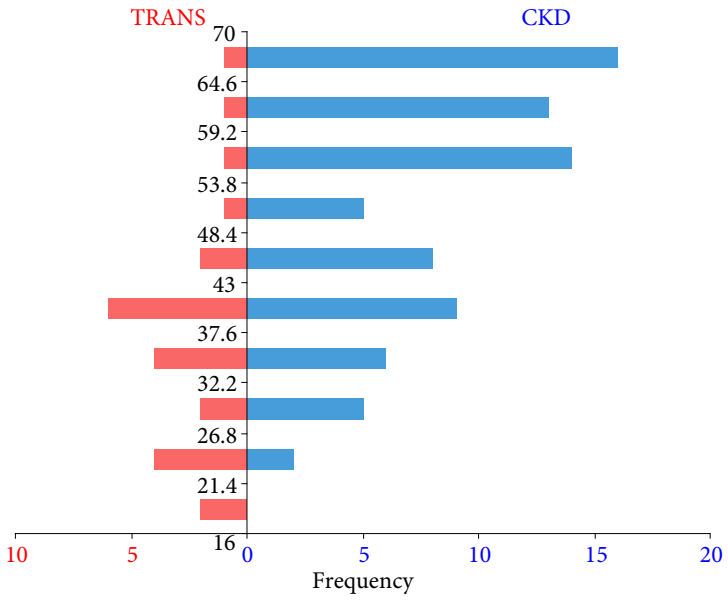

Figure 1. Frequency of CKD and renal transplant patients

Source: compiled by the authors

Chronic glomerulonephritis was the leading cause of CKD, accounting for 43.5% of cases, followed by diabetes mellitus, hypertension, and other diseases. The average duration of CKD from diagnosis was 2.5 years, ranging from 1 month to 5 years. Comorbidities in CKD patients included diabetes mellitus, hypertension, ischaemic heart disease, and chronic viral hepatitis caused by Hepatitis B and C viruses (Fig. 2).

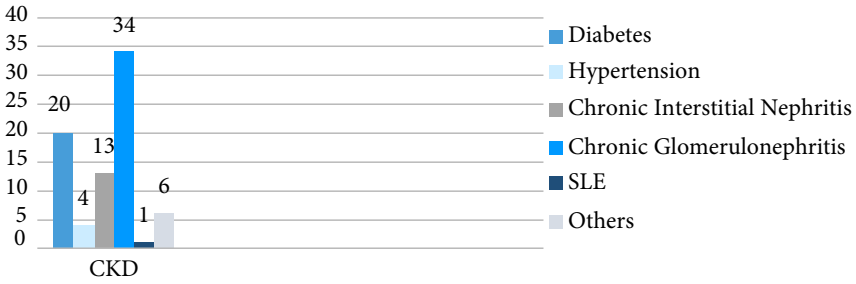

Figure 2. Leading causes of CKD

Source: compiled by the authors

Various investigations were conducted in CKD and post-renal transplant patients, including serum calcium, phosphates, parathormone, and vitamin D3 levels. The mean serum calcium level in CKD cases was 8.37 mg/dL (SD 0.92), ranging from 1.3 to 9.4 mg/dL, while post-transplant cases had a mean serum calcium level of 8.17 mg/dL (SD 1.09), ranging from 3.6 to 9.0 mg/dL (Fig. 3), which depicts minimal reduction in mean calcium levels post transplantation. For serum phosphorus, CKD cases had a mean level of 4.25 mg/dL (SD 0.47), with a range of 3.4 to 5.6 mg/dL, compared to post-transplant cases, which had a mean of 4.15 mg/dL (SD 0.51), ranging from 3.4 to 5.8 mg/dL (Fig. 4). The mean serum parathormone level in CKD cases was 24.39 U/dLL (SD 41.8), ranging from 1.6 to 260 U/dL, whereas post-transplant cases had a mean level of 8.13 U/dLL (SD 7.13), with a range of 2 to 24 U/dLL. A significant reduction in serum parathormone levels after transplantation was observed (p = 0.001, CI 0.05) (Fig. 5).

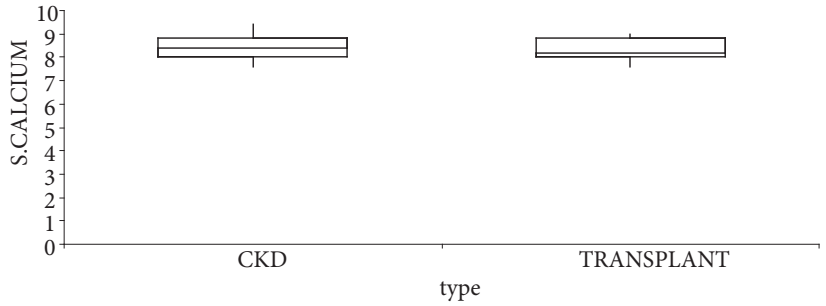

Figure 3. Serum calcium levels

Source: compiled by the authors

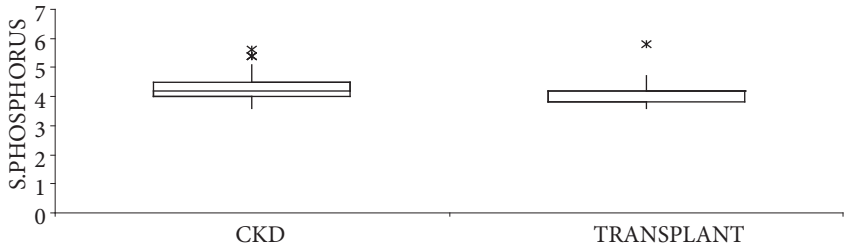

Figure 4. Serum phosphorus levels

Source: compiled by the authors

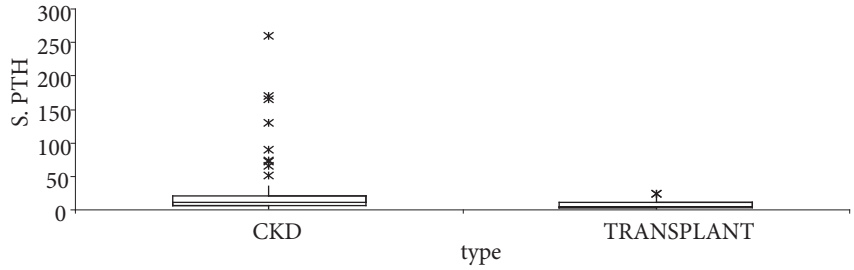

Figure 5. Serum parathormone levels

Source: compiled by the authors

Regression analysis was conducted to examine the relationships between various variables. The relationship between serum calcium and phosphorus was found to be insignificant ($p > 0.05$), as was the relationship between serum calcium and PTH levels ($p = 0.63$). Similarly, the associations between serum vitamin D3 and PTH levels ($p = 0.11$) and between serum vitamin D3 and calcium levels ($p = 0.11$) were also insignificant. The comparison between CKD and post-transplant patients revealed a significant decline in parathyroid hormone (PTH) levels in the post-transplant group (mean PTH: 24.39 U/dLL in CKD vs. 8.13 U/dLL in post-transplant; $p = 0.001$), suggesting improved mineral metabolism after transplantation. There were no significant differences in serum calcium and phosphorus levels between the two groups, although the post-transplant patients exhibited more stable control of these parameters.

The observed reduction in PTH post-transplant is clinically relevant, as it indicates effective reversal of secondary hyperparathyroidism and better bone-mineral regulation. This finding has important implications for managing bone health in CKD patients, suggesting that post-transplant care should focus on maintaining this improvement to reduce the risk of renal osteodystrophy and associated fractures. These results enhance understanding of mineral metabolism in CKD and posttransplant patients and support the need for tailored interventions to address disruption of mineral and bone metabolism in CKD.

Discussion

This study included a cohort of 78 patients with CKD, 24 post-renal transplant patients, and 28 control cases. The analysis of various biochemical parameters related to MBD revealed significant insights, particularly in the context of serum calcium, phosphates, vitamin D3, and PTH levels. Each of these parameters plays a crucial role in the management of CKD and the outcomes following renal transplantation. In this study, the mean serum calcium level among CKD patients was 8.37 mg/dL (SD 0.92), while for post-transplant patients, it was 8.17 mg/dL (SD 1.09). Both levels are below the normal range, indicating potential disturbances in calcium metabolism. In contrast, A.K. Yadav *et al.* [8] reported a mean calcium level of 9.5 ± 0.6 mg/dL in their cohort, which was higher than the levels observed in this study. The differences could be attributed to variations in dietary intake, geographic factors, or differences in the patient population. The study by A.K. Yadav *et al.* [8] also demonstrated significant associations between vitamin D deficiency and calcium levels, reinforcing the notion that vitamin D status is a critical factor influencing calcium homeostasis in CKD patients.

The mean serum phosphorus levels in this study were 4.25 mg/dL (SD 0.47) for CKD patients and 4.15 mg/dL (SD 0.51) for post-transplant patients, both of which are above the normal range. This is consistent with the findings of C.P. Kovesdy *et al.* [9], who reported an association between elevated serum phosphorus levels and anaemia in kidney transplant recipients. In their analysis, every 1 standard deviation increase in phosphorus was linked to a significant decrease in haemoglobin levels. This correlation highlights the importance of managing phosphates levels to prevent complications such as anaemia, a common issue in CKD patients. Furthermore, the findings of Y. Doi *et al.* [10] indicate that high phosphates levels, along with parathormone levels predict poor renal outcomes in kidney transplant patients. Their study underscores the need for careful monitoring of phosphorus levels in this cohort, as elevated phosphorus could lead to increased cardiovascular risk and graft dysfunction. A. Rastogi *et al.* [11] suggest an integrated approach to phosphorus control in CKD patients on dialysis by incorporating measurements of calcium, phosphorus, and PTH along with correlations between dietary adjustments and CKD-MBD drugs, which facilitate improved patient management. A study by M. Conley *et al.* [12] emphasises the role of dietary phosphates intake in renal disease patients. The study concluded that levels of total dietary phosphates intake showed no statistically significant relationship with biochemical markers of bone and mineral metabolism or intermediate cardiovascular markers. This finding mandates further extensive research into this group of patients.

This study reported the mean serum vitamin D3 levels of CKD patients as 45.21 U/dLL (SD 36.74), with a range of 1.8 to 149 U/dLL, while post-transplant patients had a mean vitamin D3 level of 30.14 U/dLL (SD 33.06), ranging from 1.2 to 122 U/dLL. Notably, the comparison of serum vitamin D3 levels between CKD and post-transplant patients showed no significant change after transplantation ($p = 0.06$, CI 0.05). This finding suggests that, despite successful transplantation, vitamin D3 levels may not improve significantly, which contrasts with expectations of enhanced vitamin D metabolism following renal transplantation. In the study by C.S. Kim *et al.* [13], vitamin D deficiency was prevalent among CKD patients, with 76.7% of stage 1 CKD patients exhibiting deficiency. This indicates that vitamin D status may remain a concern even after transplantation, potentially due to ongoing metabolic alterations or insufficient dietary intake. Additionally, the observations of A.K. Yadav *et al.* [8] reinforce the significance of vitamin D in maintaining calcium and phosphorus balance. Their findings revealed that lower vitamin D levels were associated with reduced serum calcium levels, which could explain the suboptimal vitamin D3 levels in both CKD and post-transplant patients in this study. Furthermore, the impact of vitamin D on bone health and its correlation with PTH levels in this study suggest that interventions aimed at correcting vitamin D deficiency could improve PTH levels and overall mineral metabolism in CKD patients. Regular assessment and supplementation of vitamin D may, therefore, be necessary to manage MBD in this population effectively.

The mean serum PTH level in this study for CKD patients was 24.39 U/dLL (SD 41.8), which decreased significantly to 8.13 U/dLL (SD 7.13) post-transplant ($p = 0.001$). This finding is consistent with Y. Doi *et al.* [10], who found

that high iPTH levels are associated with poor renal outcomes. In their study, they reported that elevated PTH levels can indicate underlying metabolic disturbances that may impact graft survival. In comparison, H. Komaba *et al.* [14] reported a mean iPTH level of 58.4 ± 32.9 ng/mL, significantly higher than the levels observed in this CKD cohort. This discrepancy may suggest that the patients in this study might have been at an earlier stage of MBD or that the treatment and dietary management of CKD patients differed between the studies. It also emphasises the importance of regular monitoring and management of PTH levels to prevent complications associated with secondary hyperparathyroidism. A study conducted in Japan recommends parathyroid interventions, such as parathyroidectomy and percutaneous ethanol injection therapy, for mineral disorders that are not managed by pharmacological means.

F. Li *et al.* [15] supported the correlation between iPTH levels and anaemia severity, suggesting that interventions targeting PTH levels may improve haemoglobin levels. In the findings of the current study, the significant reduction in PTH levels post-transplant could potentially lead to similar improvements in anaemia, highlighting the interconnectedness of these parameters. J. Malyzko *et al.* [16] discussed the challenges in assessing calcium and phosphates levels in kidney transplant recipients, indicating a potential gap in monitoring bone metabolism parameters post-transplant. This underscores the importance of regular evaluation of PTH and other mineral metabolism markers, as highlighted in this study, to prevent complications such as secondary hyperparathyroidism.

M. Fusaro *et al.* [17] surveyed 106 Italian nephrologists, providing valuable insights into the clinical practices surrounding the management of CKD and secondary hyperparathyroidism (sHPT). Their findings present a stark contrast to those of the current study. In this research, which involved CKD and post-renal transplant patients, significant variations were observed in calcium, phosphorus, and PTH levels, as well as in the relationships among these parameters. Notably, the current study reported a mean serum calcium level of 8.37 mg/dL in CKD patients, which is relatively lower than the normal range, indicating potential hypocalcaemia. This contrasts with the findings from the Italian nephrologists' survey, which highlighted high accessibility to ionised calcium and PTH measurements, suggesting that nephrologists in Italy have the resources to closely monitor these critical parameters.

K. Nakai *et al.* [18] emphasised the role of persistent hyperparathyroidism concerning graft function. The findings of this study, particularly the significant reduction in PTH levels posttransplant, support the view that effective management of PTH levels is crucial for optimising graft function and improving patient outcomes. While biochemical markers are essential in monitoring bone health, bone histomorphometry remains the gold standard for assessing bone turnover. However, it is rarely performed in routine clinical practice. Bone turnover markers (BTMs) could serve as a much-needed, non-invasive diagnostic tool to bridge the therapeutic gap for patients with advanced CKD and bone fragility [19]. Future research comparing the efficacy of bone histomorphometry and BTMs will likely yield useful insights. Other indicators of bone turnover, such as dual-energy X-ray absorptiometry (DEXA), have not been found to be reliable markers in this context [20].

This study demonstrated that abnormalities in mineral metabolism, particularly elevated PTH levels, in CKD patients significantly improve following renal transplantation. The stabilisation of calcium and phosphorus levels post-transplant highlights the effectiveness of restored kidney function in mitigating CKD-MBD. These findings underscore the importance of monitoring and managing mineral imbalances to enhance long-term bone health outcomes in both CKD and post-transplant patients.

Conclusions

This study contributes to the understanding of MBD parameters in CKD and post-renal transplant patients. It highlights significant differences in key markers of mineral metabolism between these groups. In CKD patients, the mean serum PTH level was markedly elevated at 24.39 U/dLL (SD 41.8), while post-transplant patients exhibited a significantly lower mean PTH level of 8.13 U/dLL (SD 7.13), indicating a statistically significant reduction ($p = 0.001$). Similarly, the mean serum calcium levels were slightly higher in CKD patients (8.37 mg/dL, SD 0.92) compared to post-transplant patients (8.17 mg/dL, SD 1.09), although this difference was not statistically significant. Serum phosphorus levels followed a similar trend, with CKD patients recording a mean of 4.25 mg/dL (SD 0.47), while post-transplant patients had a mean of 4.15 mg/dL (SD 0.51). Despite the minor differences in calcium and phosphorus levels between the groups, the most notable improvement post-transplant was the significant decline in PTH levels, reflecting enhanced control of bone-mineral metabolism. These results underscore the critical impact of renal transplantation on normalising mineral metabolism, particularly the reversal of secondary hyperparathyroidism, which plays a key role in managing CKDMBD and reducing the risk of complications such as renal osteodystrophy. Regular assessment of vitamin D status should also be integrated into management plans, given its pivotal role in calcium and phosphorus homeostasis. As understanding of the complex interactions among these parameters improves, it will be essential to implement targeted interventions aimed at optimising mineral metabolism in CKD and transplant populations to enhance patient outcomes and quality of life. Future studies should focus on longitudinal data to evaluate the long-term effects of managing these parameters on clinical outcomes.

Acknowledgements

None.

Conflict of Interest

None.

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Асоціація біомаркерів мінерального обміну у пацієнтів з хронічною хворобою нирок та реципієнтів ниркового трансплантата: одноцентрове проспективне дослідження

Гаурав Вохра

Доктор медичних наук, доцент, капітан-хірург
Інститут військово-морської медицини
400704, WW26+FHF Кегаон, м. Наві Мумбаї, Індія
https://orcid.org/0000-0001-9608-7312

Нагамахендран Раджендран

Дипломант Національної ради, доцент, командир крила
Госпіталь військово-повітряних сил
400704, WV5V+7X8, Дхаува, Кішан Гхат, м. Раджастан, Індія
https://orcid.org/0000-0002-9854-7236

Анджу Кумарі

Магістр хірургії, консультант-офтальмолог
Офтальмологічна лікарня Лаксмі
Мулла Хамід Роуд, м. Наві Мумбаї, Індія
https://orcid.org/0009-0006-7447-0277

Анотація. Цілісна оцінка параметрів мінеральних порушень кісткової тканини, включаючи сироватковий кальцій, фосфор, паратгормон і 1,25-дигідроксивітамін D, може прогнозувати ниркові наслідки у реципієнтів ниркового трансплантата, хоча результати варіюють. Метою цього дослідження було оцінити біохімічні параметри мінеральних порушень кісткової тканини у пацієнтів з хронічною хворобою нирок та реципієнтів ниркового трансплантата. Всього було включено 78 пацієнтів з хронічною хворобою нирок, 24 реципієнти після трансплантації нирки та 28 осіб контрольної групи, середній вік яких становив 48,24. Переважною причиною хронічної хвороби нирок був хронічний гломерулонефрит, за яким слідували діабет і гіпертонія. Було оцінено рівні кальцію, фосфору, паратгормону та вітаміну D3 у сироватці крові. У пацієнтів з хронічними захворюваннями нирок середній рівень кальцію в сироватці крові становив 8,37 мг/дл, а фосфору – 4,25 мг/дл, тоді як у пацієнтів після трансплантації середній рівень становив 8,17 і 4,15 мг/дл відповідно. Значне зниження рівня паратгормону в сироватці крові спостерігалося у пацієнтів після трансплантації (в середньому 8,13 Од/дл) порівняно з пацієнтами з хронічною хворобою нирок (в середньому 24,39 Од/дл). Після трансплантації не було відмічено суттєвих змін у рівні вітаміну D3. Регресійний аналіз виявив незначний зв'язок між рівнями кальцію і фосфору в сироватці крові та рівнем паратгормону. Це дослідження висвітлює біохімічний дисбаланс, пов'язаний з хронічними захворюваннями нирок, та вплив трансплантації нирки на рівень паратгормону, підкреслюючи необхідність регулярного моніторингу та управління параметрами мінеральних порушень кісткової тканини у цих популяціях пацієнтів

Ключові слова: біомолекулярні показники; мінеральні порушення кісткової тканини; гормональний дисбаланс при захворюваннях нирок; 1,25-дигідроксивітамін D