Volume 4, Issue 5, 2026

Frontier in Medical & Health Research

ISSN: (e) 3007-1607 (p) 3007-1593

EVALUATION OF VITAMIN D, CALCIUM AND RFT'S IN CHILDREN SUFFERING FROM NEPHROTIC SYNDROME

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DOI: http://doi.org/10.5281/zenodo.20367077

Keywords
NS (VDBP), Vitamin D binding protein, 25 Hydroxy vitamin D, PCR

Article History
Received: 22 March 2026
Accepted: 01 May 2026
Published: 21 May 2026

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Abstract

Background: *Nephrotic syndrome is one of the most prevalent chronic kidney diseases among children and is marked by the symptoms of massive proteinuria, hypoalbuminemia, hyper lipidaemia and edema, which have significant metabolic repercussions for vitamin D, calcium, and renal health. Vitamin D deficiency can further exacerbate immunologic dysregulation, make infections more likely, and cause worse proteinuria, thus complicating the course of the disease. Although there is a need for evaluating vitamin D, calcium, and renal function parameters due to their significance for clinical outcomes in nephrotic syndrome, there is an insufficient focus on their assessment in children with nephrotic syndrome.*

Objective: *To evaluate the serum levels of vitamin D, calcium and Rfts in children suffering with nephrotic syndrome.*

Methodology: *It was Retrospective Observational study to evaluate serum vitamin D and calcium levels in children suffering with nephrotic syndrome. The study conducted at Children Hospital and collaborating paediatric nephrology units Total duration of study will be 4 months. Sample size = 91 (Sample size was calculated using the Cochran's formula).*

Results: *A total of 91 children with nephrotic syndrome were included in this study. The average age of the participants was11.08 years of age with an SDof4.20, weight was11.08kg with SD of 4.20, and height was 84.10 cm with SD of 16.62, indicating that there was some variation in development among the participants. In terms of gender, the distribution is almost equal with 47 (51.6) being males and 44 (48.4) being females. Overall, the demographic data show a balanced population in terms of gender and heterogeneous distribution of age, weight, and height, providing a solid baseline for evaluating laboratory parameters in these children*

Conclusion: *The present study concludes that children with nephrotic syndrome exhibit significant abnormalities in vitamin D, calcium, and renal function parameters. Serum vitamin D is correlated weakly with serum calcium indicating that the status of vitamin D alone is not a predictor of calcium levels in this population.*

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INTRODUCTION

Nephrotic syndrome (NS) stands as the most prevalent chronic kidney disease affecting children while it serves as a primary source of worldwide health issues. The disease presents itself through four main symptoms which include extreme protein loss from urine and low blood albumin levels and high blood lipid levels and body swelling (1). In addition to these classical features, NS is associated with significant derangement in calcium, vitamin D metabolism, which may result in substantial skeletal and growth abnormalities in children.

Proteinuria is the primary pathological feature of NS and the main driver of associated metabolic disturbances. Among the urinary protein losses are albumin and small-molecular-weight proteins like vitamin D-binding protein albumin and small-molecular-weight proteins like vitamin D-binding protein (VDBP), the major transporter of 25-hydroxyvitamin D [25(OH)D] in circulation (2). Because nearly 85–90% of vitamin D metabolites are bound to VDBP and the remainder to albumin, urinary loss leads to reduced circulating levels of vitamin D (3). Consequently, serum 25(OH) D levels fall, resulting in reduced intestinal calcium absorption, hypocalcaemia, compensatory hyperparathyroidism, skeletal demilitarization, and bone deformities. Reduced sunlight exposure, poor dietary intake, and chronic inflammation further worsen vitamin D deficiency in these children (4).

Consequently, serum 25(OH) D levels fall, resulting in reduced intestinal calcium absorption, hypocalcaemia, compensatory hyperparathyroidism, skeletal demilitarization, and bone deformities. Reduced sunlight exposure, poor dietary intake, and chronic inflammation further worsen vitamin D deficiency in these children. Corticosteroids the mainstay of therapy in-steroid-sensitive NS further aggravate this metabolic derangement. Glucocorticoids impair intestinal calcium absorption, increase renal calcium excretion, and suppress osteoblastic activity, thereby enhancing bone resorption and lowering bone mineral density (BMD). Repeated or prolonged steroid therapy in frequently relapsing and steroid-dependent NS accelerates bone demineralization and increases the risk of rickets, osteomalacia, and fractures (5). Children with NS may demonstrate biochemical abnormalities of bone metabolism even before significant loss of kidney function occurs.6,7 Mehta and Nanda reported that children with active NS had significantly lower serum calcium and higher parathyroid hormone (PTH) levels than those in remission, indicating secondary hyperparathyroidism. Chronic hypocalcaemia stimulates PTH secretion, which helps maintain serum calcium but at the cost of increased bone resorption, perpetuating bone loss (6). Vitamin D deficiency in NS may also have immunomodulatory implications. Vitamin D regulates immune function by influencing T-cell differentiation and cytokine production. Low vitamin D levels may therefore worsen immune dysregulation in NS and potentially influence relapse rates or steroid responsiveness. Selewski found that all children with new-onset idiopathic NS had 25(OH)D levels below 20 ng/mL at diagnosis, and more than half remained deficient even in remission (7, 8). Another author similarly noted more severe deficiency among steroid-dependent and steroid-resistant patients, suggesting an association between vitamin D deficiency and disease severity. It demonstrated that supplementation with vitamin D_3 and calcium significantly increased serum 25(OH) D levels in steroid-sensitive NS but did not significantly improve BMD or reduce relapse rates during six months of follow-up. Some supplemented children also developed hypercalciuria, emphasizing the need for careful monitoring (9). One study reports improved bone mineral content with prolonged supplementation, suggesting benefit over longer durations (10). Persistent deficiency even during remission highlights the chronic nature of these abnormalities. The interplay between vitamin D, calcium, and PTH has important implications for growth and bone development. Abnormalities during childhood and adolescence critical periods for achieving peak bone mass may predispose children to early osteopenia and osteoporosis. Vitamin D deficiency is also linked to impaired immune responses, infection susceptibility, and

Frontier in Medical & Health Research
ISSN: (e) 3007-1607 (p) 3007-1593

dyslipidemia, which may further complicate NS. Given this complex metabolic profile, careful monitoring of calcium and vitamin D levels in children with NS is essential. Routine measurement of serum calcium, phosphorus, 25(OH)D, and PTH provides important insight into bone-mineral metabolism and guides timely supplementation (11). Therefore, evaluating serum vitamin D and calcium in pediatric NS patients is crucial for detecting disease-associated metabolic disturbances and preventing long-term skeletal complications.

Tests of renal function (RFTs), such as serum creatinine, BUN, and electrolytes, are crucial markers that are utilized to evaluate the function of the kidneys. While nephritic syndrome is mainly a disease of the glomerulus, it usually maintains normal renal function in its initial phases; however, frequent relapse episodes, steroid resistance, or chronic it may impair renal function. It is important to monitor RFTs in patients with NS to determine any impairment of renal function. Studies conducted recently have shown an association between vitamin D deficiency and the degree of proteinuria among children with nephrotic syndrome. Vitamin D is believed to be instrumental in protecting podocytes from damage and minimizing inflammatory response in glomeruli. Hence, vitamin D deficiency could be linked to worsening proteinuria and disease progression. Additionally, variations have been noted in the level of vitamin D between children suffering from steroid-sensitive and steroid-resistant nephrotic syndrome (12, 13).

Deficiency of calcium and vitamin D in children with nephrotic syndrome is largely under diagnosed and treated, especially due to the importance of this condition for children with NS. Not all clinicians routinely check the level of those elements in children because of their absence from the management algorithm, which may cause problems in the future (14, 15). As mentioned above, proteinuria causes the loss of vitamins in urine, including vitamin D. Besides, due to hypoalbuminemia, which is also a feature of nephrotic syndrome, the patient experiences a reduction in the level of vitamin D in plasma because most vitamin D binds to albumin. In addition to this, hypoalbuminemia affects the level of total and ionized calcium, because some percentage of that element in plasma is albumin bound. Thus, the level of total and ionized calcium is likely to be changed in children with nephrotic syndrome (16, 17).

Considering all the above factors, the assessment of vitamins D and C levels, as well as assessing the kidney function in children who have been diagnosed with nephrotic syndrome, become extremely important. In addition to providing valuable information regarding the biochemical changes accompanying the disease, these assessments also provide valuable insight into the disease severity, prognosis, and patient response to treatment.

MATERIALS AND METHODS

This retrospective observational study was conducted to evaluate serum vitamin D and calcium levels in children suffering from nephrotic syndrome. The study was carried out at Children Hospital and collaborating pediatric nephrology units over a total duration of 4 months. A total of 91 children were included in the study, with sample size calculated using Cochran's formula. Consecutive sampling was employed, and eligible patients presenting to the participating centers were enrolled consecutively until the target sample size was reached. The inclusion criteria were children aged 1 to 18 years with a clinical diagnosis of nephrotic syndrome and hyperlipidemia as per hospital and pediatric nephrology criteria. The exclusion criteria included known chronic liver disease, chronic gastrointestinal disease, metabolic bone disease, chronic illnesses affecting vitamin D or calcium metabolism, as well as the presence of heavy proteinuria, hypoalbuminemia, or edema.

RESULTS

Demographic Characteristics of Children with Nephrotic Syndrome

A total of 91 children with nephrotic syndrome were included in this study. The average age of the participants was 11.08 years of age with an SD of 4.20, weight was 11.08 kg with SD of 4.20, and height was 84.10 cm with SD of 16.62, indicating that there was some variation in development

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ISSN: (e) 3007-1607 (p) 3007-1593

among the participants. In terms of gender, the distribution is almost equal with 47 (51.6%) being males and 44 (48.4%) being females. Overall, the demographic data show a balanced population in terms of gender and heterogeneous distribution of age, weight, and height, providing a solid baseline for evaluating laboratory parameters in these children.

Table 1: Demographic characteristics of children with nephrotic syndrome

Variable	Mean± SD
Age (years)	11.08±4.20
Weight (kg)	11.08±4.20
Height (cm)	84.10±16.62
Gender	Frequency (Percentage)
Male	47 (51.6%)
Female	44 (48.4%)

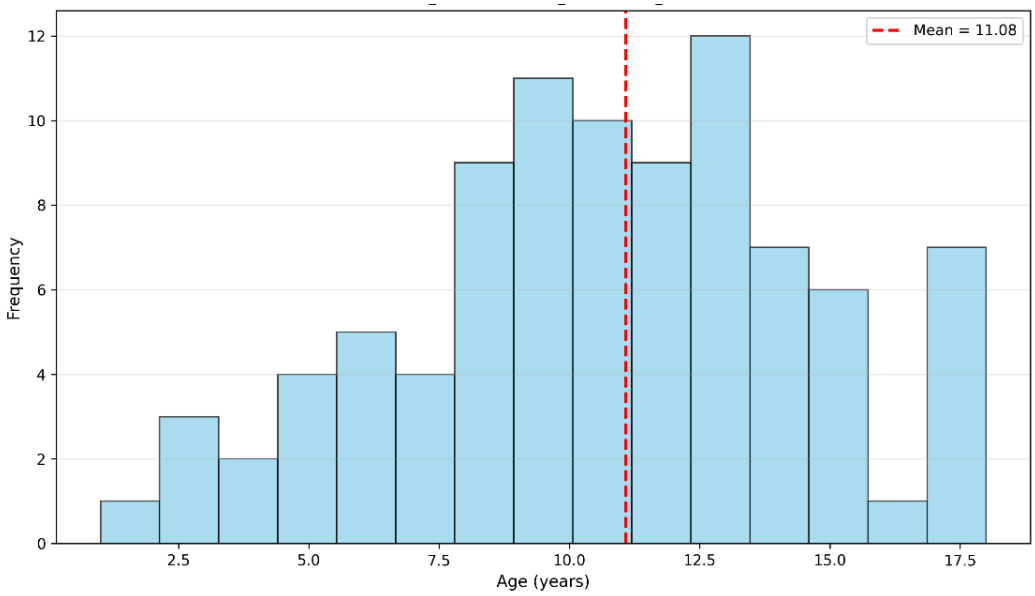

Figure 1: Histogram of Age

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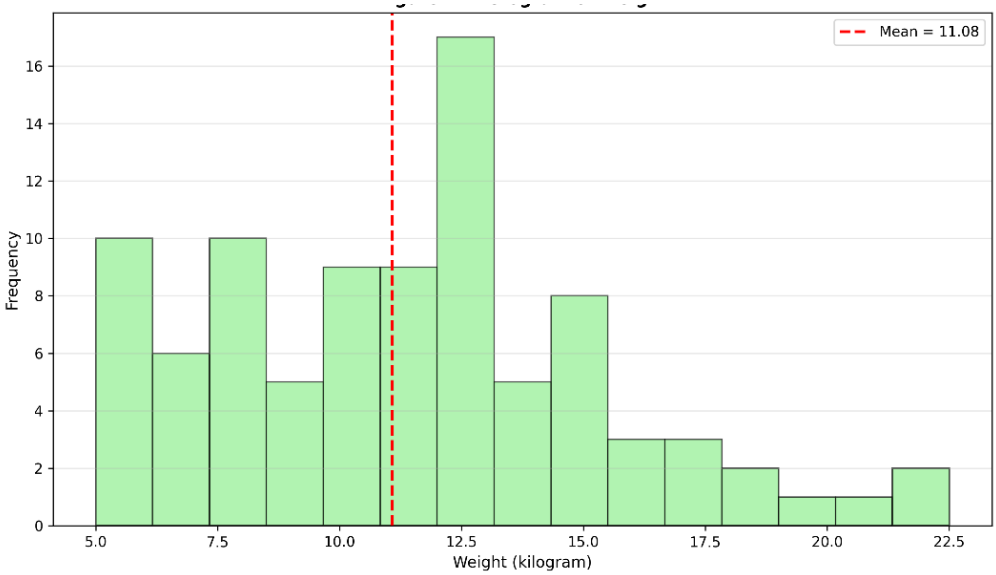

Figure 2: Histogram of Weight

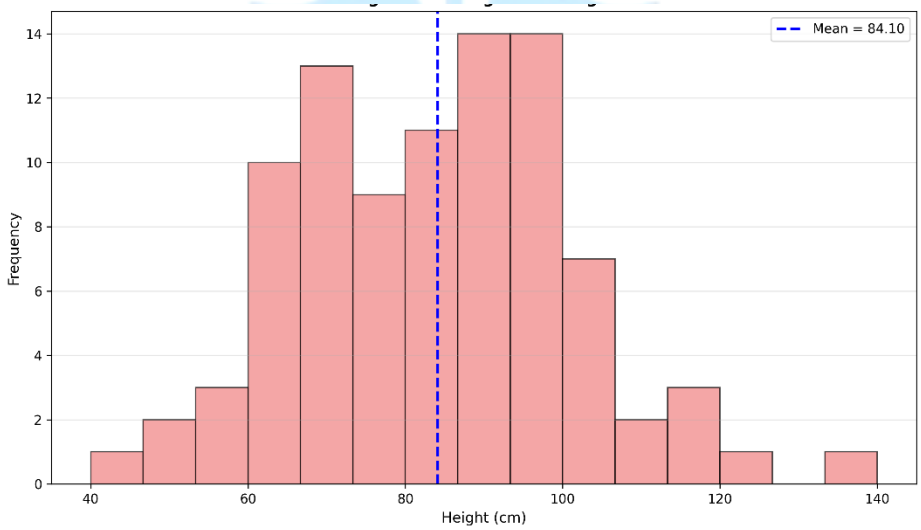

Figure 3: Histogram of Height

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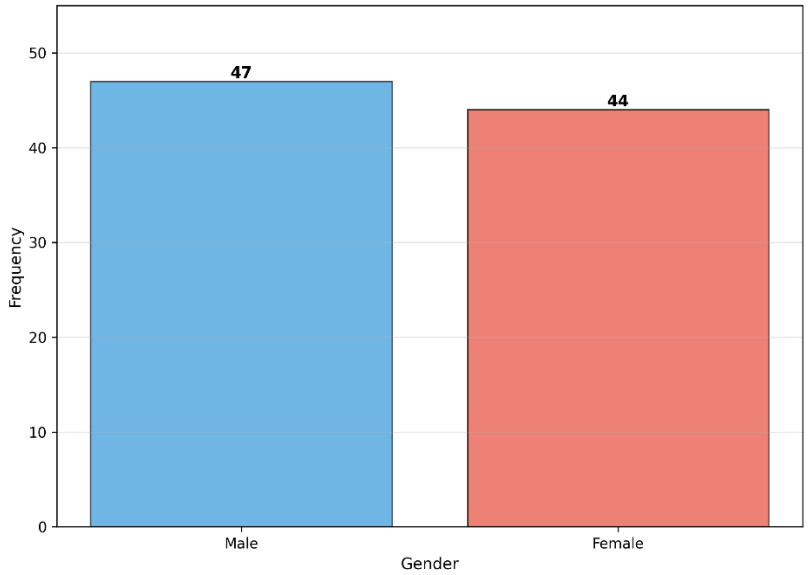

Figure 4: Bar Chart of Gender

Laboratory Parameters of Study Participants

The average serum urea concentration was 150.80 ± 114.76 mg/dl, which showed a big variation in levels of nitrogenous waste accumulation, and the average serum creatinine was 6.80 ± 6.16 mg/dl, which showed differing degrees of renal impairment among participants. The mean serum vitamin D level was 11.16 ± 5.27 ng/dl, showing a trend toward deficiency, which is commonly observed in nephrotic syndrome due to urinary loss of vitamin D–binding protein. Serum calcium levels had also decreased being 6.50 ± 1.48 mg/dl and are in line with the disturbed mineral metabolism in these children. Altogether, these lab results indicate the presence of the impaired kidney effects and vitamin D and calcium deficit commonly observed in the study population.

Table 2: Laboratory parameters of study participants

Parameter	Mean±SD
Serum Urea (mg/dl)	150.80±114.76
Serum Creatinine (mg/dl)	6.80±6.16
Serum Vitamin D (ng/dl)	11.16±5.27
Serum Calcium (mg/dl)	6.50±1.48

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ISSN: (e) 3007-1607 (p) 3007-1593

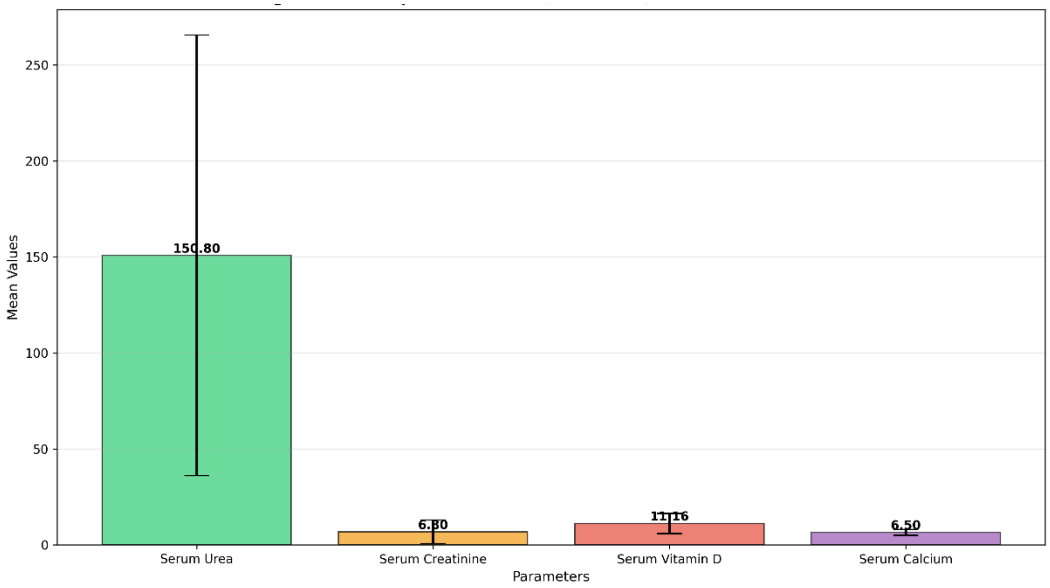

Figure 5: Bar Graph of Vitamin D, Calcium, Serum Urea and Creatinine

Correlation Analysis: Serum Vitamin D, Calcium, Urea, and Creatinine

There was a significant positive correlation between serum urea and serum creatinine (rs=.499, p<.001), as serum urea and serum creatinine share a common renal excretion physiological pathway. More significantly, serum calcium was statistically significantly and weakly negatively correlated with serum creatinine, indicating that increased serum creatinine (signifying diminished renal functioning) is linked with decrease in serum calcium. The correlation between calcium and urea was negative, but was insignificant. There were no statistically significant correlations between serum vitamin D and any other biochemical parameter. No significant correlation existed between calcium and vitamin D.

The high negative correlation between creatinine and calcium indicates that moderate impairment of renal functions could have a notable effect on calcium homeostasis. The high correlation between urea and creatinine confirms internal consistency of renal functions indicators. In general, these results suggest that the level of vitamin D alone does not determine the content of calcium in this group; however, renal activity (measured by creatinine) can be a better factor of the content of serum calcium.

Table 3: Spearman's correlation matrix for serum vitamin d, calcium, urea, and creatinine

Variable	Vitamin D (ng/dl)	Calcium (mg/dl)	Urea (mg/dl)	Creatinine (mg/dl)
Vitamin D (ng/dl)	1.00	0.112	0.123	0.132
Calcium (mg/dl)	0.112	1.00	-0.125	-.229*
Urea (mg/dl)	0.123	-0.125	1.00	.499**
Creatinine (mg/dl)	0.132	-.229*	.499**	1.00

* $p < 0.05$ ** $p < 0.001$

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DISCUSSION

The current study interested correlations between serum vitamin D, calcium, urea and creatinine in 91. The correlation coefficient indicated that serum vitamin D had weak non-significant positive relationships with calcium (r=0.112, p=0.292), urea (r=0.123, p=0.247), and creatinine (r=0.132, p=0.212).There were significantly positive urea-creatinine (r=0.499, p<0.01)and urea-calcium (r=0.618, p=0.01) positive correlations indicating renal excretion dependence between the two and creatinine, respectively. Significantly, serum calcium showed a significant negative association with creatinine (r n= - 0.229, p= 0.029), indicating a negative relationship between high levels of creatinine and low calcium levels.

Wijaya et al. (2022) investigated 68 pediatric CKD between 2-18 years old and discovered that low vitamin D level has a significant correlation with growth retardation (p = 0.005) when compared to vitamin D level. Their analysis also revealed good correlations between calcium level (p = 0.026) and phosphorus level (p = 0.222) and growth retardation. In contrast, our study found a weak, non-significant correlation between vitamin D and calcium (r_s =0.112, p=0.292). Wijaya et al. found positive correlation coefficients of vitamin D (0.427), calcium (0.277) and phosphorus (0.300) with the results of growth. The creatinine – calcium correlation (rn=-0.229, p = 0.029) is similar in magnitude to their calcium one, deeming that although our study did not provide a direct vitamin D-calcium correlation, the renal functioning-calcium one we found could be significant enough in the clinic (18).

A study by Kumar et al. found out 506 CKD pediatric patients and discovered that 25-hydroxyvitamin D deficiency (defined as less than 20 ng/ml) occurred in 28% of the participants during enrolment. Other important predictors of deficiency were advantage, non-whites, an assessment made during winter, milk intake less than daily, not taking nutritional vitamin D supplements, and proteinuria. Comparatively our research found the mean vitamin D serum concentrationof11.16 ng/dlthat isway below the 20 ng/ml level applied in the CKD study. This implies that there is a significantly higher prevalence of vitamin D deficiency in our cohort, which probably is high (above80bythe mean value itself). Another CKD study also found that the lower GFR level, the lower serum 25OHD, the lower calcium level and the higher FGF level were important determinants of secondary hyperparathyroidism. The fact that we have found a significant negative correlation between creatinine and calcium (r n = -0.229, p = 0.029) is consistent with their results that deteriorating renal function (reflected by reduced GFR or increased creatinine) is related to mineral metabolism abnormalities such as hypocalcaemia (19).

Jung et al. (2023) examined 431 pediatric kidney disease patients in the cohort and discovered that the median serum calcium level was relatively normal irrespective of the stage of disease, whereas 1,25-dihydroxy vitamin D level declined significantly with increasing stage of disease. Their study demonstrated that hyperparathyroidism prevalence increased significantly with disease stage, ranging from 37.3% in stage 3a to 52.9% in stage 5(17). In sharp contrast, our research also revealed an average of 6.50mg/dl of serum calcium, quite below the normal reference ranges and also below the median of their samples. Such a contrast implies that we have more severe hypocalcemia than the Korean kidney disease population, which can be due to differences in nutritional calcium intake, supplementation use, or geographical differences in vitamin D status. They also found in their study that, urine calcium-to-creatinine ratios and bone densitometry Z-scores reduced significantly with increasing stage of disease. The discovery of a strong negative relationship between serum creatinine and calcium (r 0 = -0.229) is also in line with their results that indicated that calcium processing becomes increasingly deficient as renal functions deteriorate (intensification of creatinine, decreased eGFR) (20).

In a study by Karava et al. (2023) 53 children at CKD stages 3-4 (GFR < 60 mL/min/1.73 m 2) were analyzed, with results indicating significant correlations between FGF23, vitamin D and iron parameters (21). Comparatively, parameters of FGF23 and iron were not measured in our study,

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ISSN: (e) 3007-1607 (p) 3007-1593

but the weak linkage of vitamin D to calcium (r n = 0.112) is opposed by moderate vitamin D-iron linkage (r n = 0.467). This difference indicates that vitamin D could be more closely related to non-calcium, iron, status parameters rather than the calcium itself in CKD populations. The statistical insignificance of the vitamin D correlations with calcium, urea or creatinine in our study, could be a result of less severe CKD in our cohort, or of less strong association between vitamin D and these parameters compared to their study of vitamin D-FGF23-iron axis (22).

Kamath et al. (2022) report a randomized controlled trial that compared vitamin D deficient children (<30 ng/ml) with children with CKD phases 2-4 who were treated with cholecalciferol either daily, weekly or monthly in Paediatric Nephrology. They found the supplementation raised the level of 25OHD in them with 85% of them having normal levels (19). The most applicable can be seen to relate to our study, where they indicated that a PTH-25OHD (25OHD) change was reported to be inversely correlated with PTH ($r = -0.4$, $p < 0.001$). Conversely, we did not observe a significant correlation between vitamin D and PTH because we did not measure PTH in our observational study. The average of vitamin D in our teamof11.16 ng/dl is an indication of extreme deficiency that would qualify them to be supplemented as per their treatment guidelines (23).

Sawires et al. (2021) had 30 children with pre-dialysis CKD randomized to receive 3 months of either native cholecalciferol or active alfacalcidol before a cross over to the other form of treatment (prospective cross-over study). They discovered that there was a substantial rise in the levels of 25(OH)D3 when the study population was administered native or active vitamin D ($p< 0.001$ in both groups in the first period). Above all, serum calcium change (1st period $p = 0.770$, 2nd period $p = 0.412$) or serum phosphorus changes or serum PTH changes did not significantly differ between both treatment groups. Compared to this, our weak vitamin D-calcium correlation ($rn=0.112$) is comparable to their result indicating that the native and active vitamin D levels do not have any different effects on calcium levels. This indicates that, in pediatric CKD, vitamin D supplementation (native or active) may not directly correlate to increase serum calcium and this result holds weight in support of our finding that other factors, as opposed to that of vitamin D, such as renal (function) and dietary (calcium intakes) and PTH (levels) are more crucial determinants of calcium homeostasis.

CONCLUSION

The present study concludes that children with nephrotic syndrome exhibit significant abnormalities in vitamin D, calcium, and renal function parameters. Serum vitamin D is correlated weakly with serum calcium indicating that the status of vitamin D alone is not a predictor of calcium levels in this population. There is however a substantial negative correlation between serum creatinine and calcium levels meaning that decreasing renal function relates to decreasing calcium levels. These results indicate that in nephrotic syndrome, vitamin D deficiency alone is not as critical as the role of renal function in the regulation of calcium homeostasis, requiring a combination of careful monitoring of both renal and mineral parameters.

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