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Original Article

Dual-Energy X-ray Absorptiometry and Bone Densitometry in CKD Children on Regular Haemodialysis

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ABSTRACT

Background: Children who had chronic kidney disease (CKD) could be usually at high risk of disturbances in bone mineral metabolism, that could impair growth and increase fracture risk. Dual-energy X-ray absorptiometry (DEXA) could be considered as the gold standard evaluation tool for bone mineral density (BMD) among these patients. This study aimed to assess bone mineral density and body composition in children who had CKD and undergoing hemodialysis using DEXA.

Methods: This cross-sectional study involved 39 children with CKD on regular hemodialysis at Zagazig University Children's Hospital. All participants underwent comprehensive clinical assessment, laboratory investigations for mineral metabolism (calcium, phosphorus, parathyroid hormone (PTH), vitamin D, and DEXA scans of the lumbar spine and whole body.

Results: The mean lumbar spine BMD was 0.63 ± 0.1 g/cm², with a median Z-score of +0.6; 43.6% had osteopenia ($Z \leq -1$) and 7.7% had osteoporosis ($Z \leq -2.5$). Higher PTH and phosphorus were significantly associated with lower BMD ($p < 0.01$), while higher calcium and vitamin D were associated with greater BMD ($p < 0.05$). Older age and longer dialysis duration correlated with higher BMD ($p < 0.05$).

Conclusion: Metabolic factors, especially serum calcium, phosphorus, vitamin D, and PTH, were the strongest determinants of BMD in pediatric dialysis patients, outweighing demographic factors or dialysis duration. Regular DEXA screening and early management of mineral disturbances may help reduce fracture risk and improve bone health in this vulnerable group.

Keywords: Dual-Energy X-ray Absorptiometry, Bone Densitometry, Chronic Kidney Disease, Hemodialysis.

INTRODUCTION

Chronic kidney disease (CKD) is considered a long-term condition where there is either structural or functional damage to the kidneys that lasts for at least three months. Usually, functional problems in CKD show up as a steady drop in the estimated glomerular filtration rate (eGFR), or constant protein in the urine, or sometimes both of these issues [1].

Problems with bone metabolism, which used to be called renal osteodystrophy, are very common among CKD patients. Nearly everyone with moderate to severe kidney problems develop some kind of bone disorders, and they have a much greater risk

of bone fractures compared to people without kidney disease [2–4]. Dual-energy X-ray absorptiometry is considered the gold standard method to check bone mineral density because it's accurate, quick, and uses very low doses of radiation [5,6].

It's now well-recognized that changes in minerals among CKD patients are strongly correlated to bone disease and can also lead to harmful calcification outside of the bones. For this reason, the Kidney Disease Improving Global Outcomes (KDIGO) group suggested a more comprehensive term, chronic kidney disease-mineral and bone disorder (CKD-MBD), to better describe these complications in CKD [7]. CKD-MBD refers to a group of problems: unusual blood levels of calcium, parathyroid hormone (PTH), phosphate, as well as vitamin D; bone changes like short stature, poor mineralization, and higher risk of fractures; and calcium building up in places outside the skeleton [8,9]. In children with CKD, these mineral changes can weaken the bones, cause pain and deformities, and increase the chances of fractures [10,11].

While many studies explored bone problems in adults with CKD, there is still a lack of research focused on children, especially regarding the use of DEXA to assess bone mineral density and body composition in pediatric CKD patients on hemodialysis. Also, the relationship between DEXA results and biochemical markers of bone disease in children is not well studied. This gap highlights the need for more research in this specific group. So, this research aimed to assess bone mineral density as well as the body composition among children with CKD undergoing hemodialysis utilizing DEXA, and to explore the possible correlations between DEXA findings with biochemical markers of renal osteodystrophy.

METHODS

This cross-sectional study was conducted at the Pediatric Nephrology Unit of Zagazig University Children's Hospitals from June 2024 to June 2025 after obtaining approval from the Institutional Review Board (IRB :328/28-April) and written informed consent from all cases' relatives. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research.

The sample size was determined based on the number of children with CKD receiving regular hemodialysis at the pediatric nephrology unit at Zagazig University Children's Hospitals during a six-month period, which were total of 48 children. A sample size of 39 was calculated using the OpenEpi program with a confidence interval of 95% and a power of 80%.

Children were included if they had a confirmed diagnosis of chronic renal failure and were on hemodialysis, regardless of sex, and were aged up to 16 years. All included patients had been receiving hemodialysis treatment for at least three months. Children were excluded if the patients not undergoing dialysis treatment or if either the child or their parents refuse to participate in the study, or if they had other diseases known to affect bone mineral density or increase fracture risk.

All participants underwent a detailed medical history review, which included information such as age, sex, comorbid conditions, time since CKD diagnosis and initiation of hemodialysis, frequency of hemodialysis per week, medication use, and family history of kidney disease. A thorough physical examination was performed to evaluate general health, with attention to vital signs including heart rate, blood pressure, and respiratory rate. Anthropometric measurements were taken for each child. Weight was measured using a digital scale and rounded to the nearest kilogram, while height (for ambulatory

children) or length (for non-ambulatory children) was measured and recorded in centimetres. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2).

Laboratory investigations were conducted for all patients including CBCs (involving red blood cells, hemoglobin, white blood cells, and platelets) were analyzed using the Sysmex XN-1000 analyzer (Japan). Serum creatinine and urea were measured with the Biosystem A15 autoanalyzer (Biosystems, Barcelona, Spain). Biochemical assessments for serum calcium, phosphorus, and alkaline phosphatase were performed using the same autoanalyzer, four milliliters of blood were drawn from each child, split into two tubes: 2 ml in an EDTA tube for complete blood count analysis and 2 ml in a plain tube for serum analysis. The blood in the plain tube was allowed to clot at room temperature, then centrifuged, serum was separated, aliquoted and stored at less than 20°Cfor later chemical assays.

Serum parathyroid hormone (PTH) levels were assessed using commercially available ELISA kits (Cat. No.: MBS263675, MyBioSource, San Diego, CA, USA), with levels below 88 ng/L considered normal and levels of 88 ng/L or higher considered elevated. Serum vitamin D was also measured using ELISA kits (Cat. No.: MBS580159, MyBioSource, San Diego, CA, USA). Vitamin D status was considered sufficient (≥ 30 ng/mL), insufficient (20–29 ng/mL), or deficient (< 20 ng/mL).

For bone mineral density (BMD) assessment, all children underwent DEXA scanning of the lumbar spine (L2–L4) using a Challenger Envision osteodensitometer (DMS, England). The scans were performed by an experienced technician with the child lying still and flat on the examination table. The lumbar spine BMD assessed trabecular bone density, while whole-body measurements (excluding the head) were used to reflect cortical bone, following recommendations from the International Society for Clinical Densitometry (ISCD) 2019 [13]. BMD values were recorded in grams per square centimeter. Z-scores for BMD were calculated according to age, sex, and height-matched normative data, using the formula: Z-score = (BMD [g/cm^2] of the patient − BMD predicted for age and sex) / standard deviation (SD) for BMD (age, sex, and height matched). BMD values were classified as normal (Z-score ≥ −1.0), osteopenia (Z-score between −1.0 and −2.5), or osteoporosis (Z-score ≤ −2.5), in accordance with World Health Organization (WHO) criteria [14].

Statistical Analysis

We used SPSS version 27 to examine the data. The chi-square test was used to compare categorical variables, which were shown as frequencies. This was determined by the Shapiro-Wilk test. Mean ± standard deviation or median (IQR) were used to summarize quantitative data where appropriate. Depending on the distribution of the data, the independent samples t-test or the Mann-Whitney U-test were utilized for group comparisons. Whenever possible, we used Pearson's or Spearman's coefficients to assess the strength of the relationships. An independent variable that might be predicted using linear regression was the dependent variable. The threshold for statistical significance was $P<0.05$, and a value of $P\leq0.001$ was regarded as highly significant.

RESULTS

The studied pediatric group (N = 39) comprised slightly more males (22 males, 56.4%) with a mean age of 10.6 years and a wide range in anthropometric data. Glomerular disease was the most common etiology (38.5%), laboratory findings showed moderate anemia (mean hemoglobin 10.06 g/dl), elevated creatinine (mean 6.52 mg/dl), and BUN (mean 51.08 mg/dl). BMD of L1 to L4 ranged from 0.3 to 1.33

with mean value 0.63 G/cm2 and median Z score was 0.6 and 43.6% had osteopenia. However, three patients had osteoporosis (7.7%). BMD of pediatric whole body ranged from 0.2 to 1.28 with mean value 0.71 G/cm2 and median Z score was 0.4 and 28.2% had osteopenia. However, three patients had osteoporosis (5.1%) (Table 1).

Increasing age (unstandardized β=0.046, p<0.001), IPTH (unstandardized β=-0.001, p<0.001) and duration of dialysis (unstandardized β=0.024, p=0.049) were significantly independently associated with total L1 – L4 bone mineral density (Table 2). Increasing calcium level and size of filter were significantly and independently associated with whole body bone mineral density in children (Table 3).

Patients with osteopenia, compared to those with normal bone mineral density, were significantly older, had lower serum calcium and vitamin D levels, higher phosphorus, elevated alkaline phosphatase, and higher iPTH. There were no significant differences between the two groups regarding gender, BMI, or filter size (Table 4).

Both lumbar and whole-body BMD were positively associated with age, disease duration, size of filter, serum calcium, and vitamin D while significant negative correlations were observed with serum phosphorus and iPTH (Table 5, Figure 1&2).

Table 1: Baseline Demographic, Clinical, Laboratory, and Densitometry Data of Studied Pediatric Group (N = 39)

Variable (Unit)	Value	Range/IQR	N (%)
Gender			
- Female			17 (43.6%)
- Male			22 (56.4%)
Age (years)	10.6 ± 2.82	2 – 15	
BMI (kg/m²)	18.23 ± 4.38	12.9 – 35	
Systolic blood pressure (mmHg)	114.87 ± 6.74	100 – 130	
Diastolic blood pressure (mmHg)	78.72 ± 6.86	70 – 95	
Etiology			
- Glomerular disease			15 (38.5%)
- Unknown			14 (35.9%)
- Tubule-interstitial disease			8 (20.5%)
- Cystic disease			1 (2.6%)
- Vascular disease			1 (2.6%)
Family History			
- Negative			33 (84.6%)
- Positive			6 (15.4%)

Variable (Unit)	Value	Range/IQR	N (%)
Ascites			
- Absent			30 (76.9%)
- Present			9 (23.1%)
Size of filter (micron)	5.03 ± 1.06	3 – 7	
Duration of disease (years)	3 (2–4)	1 – 10	
Hemoglobin (g/dl)	10.06 ± 1.25	8 – 14	
Creatinine (mg/dl)	6.52 ± 2.01	1.34 – 10.7	
BUN (mg/dl)	51.08 ± 14.34	25 – 81	
Calcium (mg/dl)	8.23 ± 1.44	5.8 – 10	
Phosphorus (mg/dl)	5.4 ± 1.47	3 – 9	
Magnesium (mg/dl)	2.54 ± 0.53	1.5 – 3.6	
Alkaline Phosphatase (IU/L)	140 (120–300)	90 – 440	
Vitamin D (ng/ml)	23 (16–29)	10 – 33	
IPTH (pg/ml)	100 (30–200)	20 – 400	
L1–L4 BMD (g/cm²)	0.63 ± 0.21	0.3 – 1.33	
Z score L1–L4	-0.6 (-1.7, 0.4)	-3.5, 1.7	
- Normal			22 (56.4%)
- Osteopenia/Osteoporosis			17 (43.6%)
Whole body BMD (g/cm²)	0.71 ± 0.21	0.2 – 1.28	
Z score whole body	0.4 (-1.1, 2.5)	-3.5, 4.5	
- Normal			28 (71.8%)
- Osteopenia/Osteoporosis			11 (28.2%)

SD: Standard deviation; IQR: Interquartile range; BMI: Body mass index; BUN: Blood urea nitrogen; TLC: Total leukocyte count; BMD: Bone mineral density; IPTH: Intact parathyroid hormone; IU/L: International units per liter; g/dl: grams per deciliter; ng/ml: nanograms per milliliter; pg/ml: picograms per milliliter; mmHg: millimeters of mercury; cm: centimeters; kg: kilograms. All continuous variables are expressed as Mean ± SD or Median (IQR) as appropriate. Categorical data are presented as number and percentage.

Table 2: Linear stepwise regression analysis of factors associated with total L1-L4 BMD among studied patients

	Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval	
	β	Std. Error	Beta	t	P	Lower	Upper
(Constant)	0.161	0.08		2.015	0.05	-0.001	0.323
Age (year)	0.046	0.009	0.62	5.29	<0.001**	0.029	0.064
IPTH	-0.001	0.0	-0.438	-4.406	0.001**	-0.001	0.0
Duration of dialysis (y)	0.024	0.012	0.235	2.042	0.049*	.0	0.049

*p<0.05 is statistically significant **p≤0.001 is statistically highly significant

Table 3: Linear stepwise regression analysis of factors associated with pediatric whole body BMD among studied patients

	Unstandardized Coefficients		Standardized Coefficients			95% Confidence Interval	
	β	Std. Error	Beta	t	P	Lower	Upper
(Constant)	-0.145	0.191		-0.757	0.454	-0.533	0.243
Calcium (mg/dl)	0.068	0.019	0.476	3.485	0.001**	0.028	0.107
Size of filter	0.058	0.026	0.302	2.21	0.034*	0.005	0.111

*p<0.05 is statistically significant **p≤0.001 is statistically highly significant

Table 4: Comparison between patients with and without osteoporosis regarding Baseline , Clinical and Laboratory data:

	Normal	Osteopenia	χ^2	p
	N= (%)	N= (%)		
Male gender				
Lumbar	13 (59.1%)	8 (47.1%)	0.559	0.455
Whole body	15 (53.6%)	6 (54.5%)	0.003	0.956
	Mean ± SD	**Mean ± SD**	t	p
Age (year)				
Lumbar	9.48 ± 2.94	12.06 ± 1.87	-3.151	0.003*
Whole body	10.05 ± 2.96	12.0 ± 1.9	-2.017	0.051
BMI (kg/m^2)				
Lumbar	18.45 ± 3.59	17.95 ± 5.33	0.344	0.733
Whole body	18.3 ± 3.49	18.06 ± 6.32	0.147	0.884
	Normal	Osteopenia	t	p
	Mean ± SD	Mean ± SD		
Size of filter				
Lumbar	4.77 ± 1.19	5.35 ± 0.79	-1.734	0.091
Whole body	4.93 ± 1.15	5.27 ± 0.79	-0.97	0.307
	Median (IQR)	Median (IQR)	Z	p
Duration (year)				

	Normal	Osteopenia	χ^2	p
Lumbar	3(1.75 – 4.25)	3(2 – 4.5)	-1.08	0.28
Whole body	3(2 – 4)	4(2 – 5)	-1.523	0.16
	Normal	Osteopenia	t	p
	Mean ± SD	Mean ± SD		
Calcium (mg/dl)				
Lumbar	9.1 ± 0.88	7.15 ± 1.26	5.591	<0.001**
Whole body	8.73 ± 1.27	7.0 ± 1.06	3.976	<0.001**
Phosphorus (mg/dl)				
Lumbar	3.73 ± 1.29	6.59 ± 1.93	-5.186	<0.001**
Whole body	4.41 ± 1.94	6.5 ± 1.86	-3.062	0.004*
Magnesium (mg/dl)				
Lumbar	2.65 ± 0.52	2.41 ± 0.53	1.448	0.156
Whole body	2.54 ± 0.57	2.56 ± 00.45	-0.146	0.885
	Median (IQR)	Median (IQR)	Z	p
Alkaline phosphate				
Lumbar	120(110 – 136.25)	300(250 – 375)	-4.097	<0.001**
Whole body	121.5(110 – 250)	300(250 – 370)	-3.198	<0.001**
Vitamin D				
Lumbar	27(22.5 – 30)	16(11.5 – 17.5)	-3.068	<0.001**
Whole body	25.5(18.25 – 30)	16(12 – 17)	-3.241	<0.001**
IPTH				
Lumbar	40(30 – 62.5)	210(130 – 335)	-3.95	<0.001**
Whole body	42.5(30 – 177.75)	210(110 – 340)	-3.069	<0.001**

t Independent sample t test Z Mann Whitney test, χ^2Chi square test

Table 5: Correlation between L1_L4 BMD& pediatric whole Body BMD and the studied parameters

	Lumbar		**Whole body**	
	r	**p**	**r**	**p**
Age (years)	**0.661**	**<0.001****	**0.273**	**0.092**
BMI(Kg/m2)	**0.168**	**0.306**	**0.007**	**0.965**
Duration (year)	**0.541**	**<0.001****	**0.181**	**0.271**
Size of filter	**0.615**	**<0.001****	**0.4**	**0.012***
Calcium (mg/dl)	0.586	<0.001**	0.362	0.028*
Vitamin D	0.643	<0.001**	0.333	0.038*
Phosphorus (mg/dl)	-0.536	<0.001**	-0.362	0.024*
Magnesium (mg/dl)	0.268	0.099	0.127	0.442
Alkaline phosphate	-0.296	0.067	-0.572	<0.001**
IPTH	-0.635	<0.001**	-0.343	0.032*

Pearson correlation coefficient *p<0.05 is statistically significant **p≤0.001 is statistically highly significant

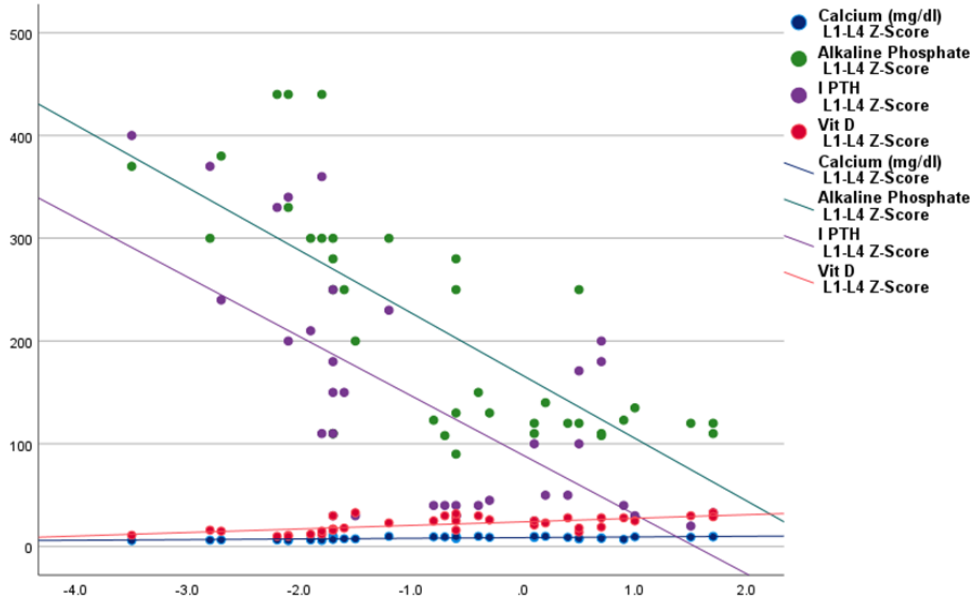

Figure 1: Scatter dot plot showing significant negative correlation between Z score of L1-L4 and IPT and phosphorus and positive correlation between it and both vitamin D and calcium

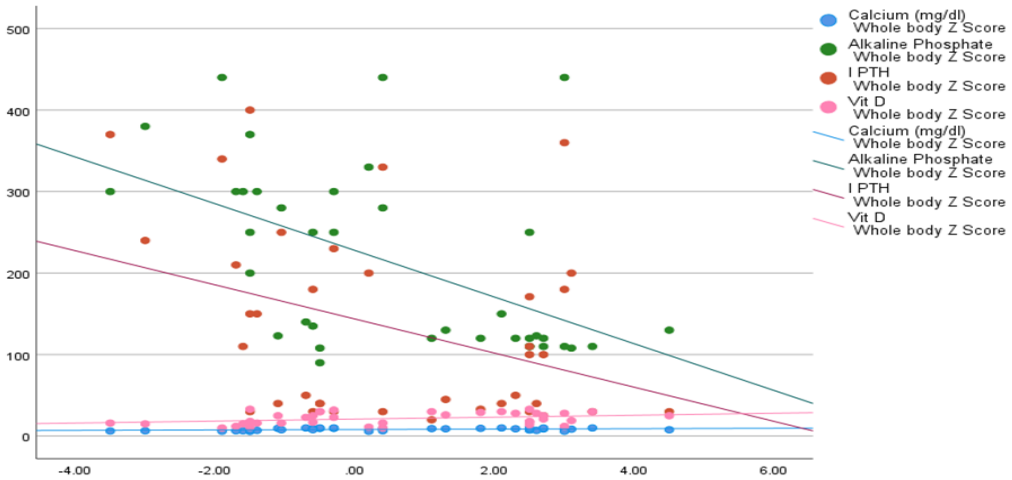

Figure 2: Scatter dot plot showing significant negative correlation between Z score of whole body and IPT and phosphorus and positive correlation between it and both vitamin D and calcium

DISCUSSION

The gold standard for evaluating BMD in children with chronic kidney disease (CKD) is DEXA scan due to its accuracy, speed, and minimal radiation exposure [12]. The relationship between mineral imbalances and bone changes in CKD is increasingly recognized, which led KDIGO to define CKD-mineral and bone disorder (CKD-MBD), as disturbances among calcium, phosphate, PTH, bone deformities, vitamin D, and extra-skeletal calcifications [13,14]. In pediatric CKD, these metabolic disturbances can impair growth, cause bone pain, and increase fracture risk [15].

In our study, children ranged in age from 2 to 15 years, with a mean age of 10.6 years, closely matching the age profiles reported

by Masalskienė et al. [16], who found means between 9.5 and 10.8 years. Our cross-sectional study included 22 males (56.4%) and 17 females (43.6%), which is similar to the distribution reported by Masalskienė et al. [16], who noted a slight male majority and a male-to-female ratio ranging from 1.05:1 to 1.32:1.

The mean BMI of our patients was 18.23 kg/m², within the normal range for school-aged children. This aligns with Prytuła et al. [17], who found most pediatric CKD patients can maintain normal anthropometrics with proper care, though short stature is common. In contrast, Karava et al. [18] reported both undernutrition and obesity patterns among children with CKD, possibly due to differences in patient demographics, intensity of nutritional management, socioeconomic factors, or CKD severity. Our patients' normal BMI likely reflects regular nutritional monitoring and support.

Blood pressures in our patients were normal for age, and most of these children were receiving antihypertensive medications as part of their management. In contrast, Masalskienė et al. [16] found that over 60% of children with CKD had hypertension. Such differences may reflect variations in reporting style, use of antihypertensive drugs, or the distribution of CKD stages across studies.

Glomerular disease was the leading CKD cause in the studied group, followed by unknown and tubulointerstitial etiologies. However, other cohorts more often report congenital anomalies of the kidney and urinary tract (CAKUT) as most common: Masalskienė et al. [16] found CAKUT in 38% of cases and Geleta et al. [19] reported similar rates. These variations likely reflect regional and referral differences.

A positive family history of kidney disease was present in 15% of our cohort, consistent with Sawaf et al. [20], who noted genetic factors (e.g., nephronophthisis, Alport syndrome) play a significant role in pediatric CKD. However, few epidemiological studies report family history in detail, limiting comparisons.

The mean dialysis filter size in our study was 5.03 microns, but this parameter is rarely detailed in published literature, limiting direct comparisons. CKD durations ranged from 1 to 10 years, with a median dialysis duration of 3 years, similar to the Egyptian cohort by El-Gamasy et al. [21], who found a mean of just over 3 years, and Levy Erez et al. [22], who reported a median of 1.5 years.

Regarding laboratory results, the mean hemoglobin in our study was 10.06 g/dL, indicating mild anemia. This aligns with data from the Lithuanian registry, which reported that more than 73% of children with advanced CKD were anemic [16]. Similarly, El-Hawy et al. [23] also found that hemoglobin levels were significantly lower in CKD patients compared to healthy controls.

The current study revealed that the mean serum calcium was slightly below the normal pediatric range. This finding coincides with Sharba et al. [24], who revealed that children with CKD on hemodialysis had significantly lower calcium levels compared to both non-dialysis CKD patients and healthy controls, highlighting the challenges in mineral metabolism control in various settings. In contrast, Ketteler et al. [25] reported that pediatric CKD patients, regardless of dialysis status, maintained comparable mean calcium levels (around 9.2 mg/dL) when standardized dialysate calcium and vitamin D supplementation were used. Such differences likely result from variability in clinical practices, including dialysate calcium concentration, vitamin D therapy, and dietary intake. Evenepoel et al. [26] further emphasized that well-managed

dialysis can lead to normal or elevated calcium, depending on supplementation and therapy.

Our patients' serum phosphorus was elevated, with a mean of 5.4 ± 1.47 mg/dL, which is consistent with many studies reporting that hyperphosphatemia becomes more common as kidney function declines. Jung et al. [27] showed that hyperphosphatemia prevalence rises with advancing CKD stage, reaching up to 41% in stage V. Sharba et al. [24] also confirmed higher phosphate levels in children on dialysis compared to non-dialysis CKD and healthy controls. Such findings underscore the difficulty of controlling phosphate balance in pediatric CKD, especially on dialysis.

The present study revealed that alkaline phosphatase (ALP) was mildly elevated, with a median of 140 IU/L (IQR: 120–300) and a range from 90 to 440 IU/L, reflecting increased bone turnover a recognized feature in advanced CKD. Schini et al. [28] also documented rising bone turnover markers, including ALP, with CKD progression. Similarly, Sharba et al. [24] observed higher ferritin in CKD patients on dialysis, supporting the link between mineral disturbance and altered bone and iron metabolism.

Vitamin D deficiency was prevalent among our patients, with a median 25(OH)D level of 23 ng/mL. This finding is consistent with several reports noting widespread vitamin D insufficiency in pediatric CKD. Parra-Ortega et al. [29] found similar results in a cross-sectional analysis, reporting a mean 25(OH)D level of 22.5 ng/mL in pediatric dialysis patients.

The mean intact parathyroid hormone (iPTH) level in our study was moderately elevated at 100 ± 104.7 pg/mL, consistent with secondary hyperparathyroidism in CKD. Jung et al. [27] reported similar patterns, with iPTH increasing as CKD progresses. Such elevations are typical and reflect disrupted mineral metabolism.

Focusing on bone outcomes, mean lumbar spine BMD in the current study was 0.63 g/cm² with a median Z-score of +0.6. Osteopenia and osteoporosis were observed in 43.6% and 7.7% of cases, respectively, indicating a significant burden of low bone mass. This is consistent with Jung et al. [27], who showed progressively lower DEXA Z-scores in children as CKD stage advanced. Hauge et al. [30] reported that dialysis groups frequently have high rates of low BMD than healthy cohorts.

Whole-body BMD measurements followed a similar pattern, with slightly lower prevalence of low BMD compared to the spine. Other studies, such as Magallares et al. [31], also reported that whole-body DEXA tends to show better Z-scores than the spine, but a substantial minority of children still have low values. Site-specific variation is expected, as trabecular bone (like the spine) is more susceptible to early loss in CKD.

The rates of osteopenia and osteoporosis in the present study were in line with the recognized burden of CKD–MBD among children who were on dialysis. Ananvutisombat et al. [32] and LeBoff et al. [33] also reported high frequencies of "low BMD" in similar populations, although inter-study differences can stem from patient characteristics, CKD duration, and differences in how low BMD is defined.

We found that older age and longer duration of dialysis, with a median of 3 years (IQR: 2–4 years), were associated with higher BMD in both the spine and whole body. This likely reflects normal skeletal growth, as bone mass accrues with age. Zhu et al. [34] noted that pediatric bone mass increases with growth, and Iseri et al. [35] found that longer dialysis duration correlated with better bone turnover and growth. This finding contrasts with adult studies, where

prolonged dialysis is linked to bone loss potentially due to children's ongoing growth and differences in management strategies [36]. Lalayiannis et al. [15] also found that longer dialysis was linked to better bone and growth outcomes in pediatric CKD.

In the present study, higher iPTH levels (mean 100 ± 104.7 pg/mL) were strongly associated with lower lumbar and whole-body BMD. This is consistent with Lalayiannis et al. [15], who found that in children on dialysis, elevated PTH and alkaline phosphatase predicted lower cortical bone density, with multivariate models showing PTH (β=–0.43, $p<0.0001$) and ALP (β=–0.36, $p<0.0001$) as significant negative predictors of bone mass. Similarly, our osteopenic patients had significantly higher PTH and ALP levels.

Conversely, Hajizadeh et al. [37] reported a positive correlation between PTH and DEXA BMD Z-scores in children with CKD, and Elsayed et al. [38] also observed that higher PTH correlated with higher BMD Z-scores. These conflicting findings may reflect differences in patient populations (CKD stage, nutritional status), or how bone turnover is influenced by treatment regimens.

Higher serum phosphorus in our study was associated with lower BMD, matching established physiology where persistent hyperphosphatemia impairs bone mineralization. However, Elsayed et al. [39] reported that children with the lowest BMD actually had lower serum phosphate—a result that might reflect more intensive phosphate binder therapy in those with more severe bone disease or confounding by high bone turnover. These paradoxical findings could also occur if increased PTH in advanced CKD temporarily buffers phosphate.

We observed that higher serum calcium and higher 25(OH) vitamin D levels were linked to better BMD and Z-scores. Lalayiannis et al. [15] also demonstrated a positive correlation between serum calcium and cortical BMD in children with CKD, and Elsayed et al. [38] found that children with low BMD had significantly lower serum calcium and vitamin D. This fits with broader pediatric data that adequate calcium and vitamin D intake supports bone health, and that vitamin D sufficiency enhances bone mineralization [38].

Notably, we found a novel association: larger dialyzer filter surface area was independently associated with higher whole-body BMD. While there are no previous pediatric studies focusing on dialyzer membrane size, this finding likely reflects the confounding effect of body size, as larger children require bigger filters and have greater bone mass. It is also possible that larger filters, with increased dialysis efficiency, contribute to improved removal of uremic toxins that impact bone health.

Limitations of study

The cross-sectional design limits causal interpretation and tracking of BMD changes over time. The relatively small, single-center sample may reduce generalizability and statistical power. Lack of bone biopsy means we could not distinguish bone turnover states. Finally, because DEXA was not size-adjusted, BMD may be underestimated in smaller children.

Conclusion

In pediatric dialysis patients, better bone health was mainly linked to higher calcium and vitamin D, while elevated PTH and phosphate were harmful. Older age, longer dialysis, and larger filter size also related to higher BMD, but metabolic factors had the strongest effect. Routine DEXA, correcting mineral imbalances, and personalized dialysis may help reduce fracture risk and support healthy growth.

Conflict of Interest or financial disclosure:
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Availability of Data: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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REFERENCES

1. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002;39(2 Suppl 1):S1–266.
2. Alem AM, Sherrard DJ, Gillen DL, Weiss NS, Beresford SA, Heckbert SR, et al. Increased risk of hip fracture among patients with end-stage renal disease. Kidney Int. 2000;58(1):396–403.
3. Jadoul M, Albert JM, Akiba T, Akizawa T, Arab L, Bragg-Gresham JL, et al. Incidence and risk factors for hip or other bone fractures among hemodialysis patients in the Dialysis Outcomes and Practice Patterns Study. Kidney Int. 2006;70(7):1358–66.
4. Coco M, Rush H. Increased incidence of hip fractures in dialysis patients with low serum parathyroid hormone. Am J Kidney Dis. 2000;36(5):1115–21.
5. Malluche H, Faugere MC. Renal bone disease: an unmet challenge for the nephrologist. Kidney Int. 1990;38(1):193–211.
6. Christiansen C. Osteoporosis: diagnosis and management today and tomorrow. Bone. 1995;17(5 Suppl):513S–6S.
7. Moe S, Drüeke T, Cunningham J, Goodman W, Martin K, Olgaard K, et al. Definition, evaluation, and classification of renal osteodystrophy: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int. 2006;69(11):1945–53.
8. Wesseling-Perry K, Salusky IB. Phosphate binders, vitamin D and calcimimetics in the management of chronic kidney disease-mineral bone disorders (CKD-MBD) in children. Pediatr Nephrol. 2013;28(4):617–25.
9. Ketteler M, Block GA, Evenepoel P, Fukagawa M, Herzog CA, McCann L, et al. Diagnosis, evaluation, prevention, and treatment of chronic kidney disease–mineral and bone disorder: synopsis of the kidney disease: improving global outcomes 2017 clinical practice guideline update. Ann Intern Med. 2018;168(6):422–30.
10. Fang Y, Ginsberg C, Sugatani T, Monier-Faugere MC, Malluche H, Hruska KA. Early chronic kidney disease–mineral bone disorder stimulates vascular calcification. Kidney Int. 2014;85(1):142–50.
11. Shroff R, Wan M, Nagler EV, Bakkaloglu S, Fischer DC, Bishop N, et al. Clinical practice recommendations for native vitamin D therapy in children with chronic kidney disease Stages 2–5 and on dialysis. Nephrol Dial Transplant. 2017;32(7):1098–113.
12. Slart RHJA, Punda M, Ali DS, Bazzocchi A, Bock O, Camacho P, et al. Updated practice guideline for dual-energy X-ray absorptiometry (DXA). Eur J Nucl Med Mol Imaging. 2025;52(2):539–63.
13. Evenepoel P, Cunningham J, Ferrari S. Bone histomorphometry in CKD: state-of-the-art and future directions. Nephrol Dial Transplant. 2021;36(4):565–73.
14. Rysz J, Franczyk B, Rokicki R, Gluba-Brzózka A. The influence of dietary interventions on chronic kidney disease–mineral and bone disorder (CKD-MBD). Nutrients. 2021;13(6):2041.
15. Lalayiannis AD, Soeiro EMD, Moysés RMA, Shroff R. Chronic kidney disease mineral bone disorder in childhood and young adulthood: a 'growing' understanding. Pediatr Nephrol. 2024;39(3):723–39.
16. Masalskienė J, Rudaitis Š, Vitkevič R, Čerkauskienė R, Dobilienė D, Jankauskienė A. Epidemiology of chronic kidney disease in children: a report from Lithuania. Medicina (Kaunas). 2021;57(2):112.
17. Prytuła A, Grenda R. Anthropometric measures and patient outcome in pediatric chronic kidney disease. Pediatr Nephrol. 2023;38(10):3207–10.
18. Karava V, Dotis J, Kondou A, Printza N. Malnutrition patterns in children with chronic kidney disease. Life (Basel). 2023;13(3):713.
19. Geleta BE, Bekele BA, Afework M. Spectrum of congenital anomalies of the kidney and urinary tract in a pediatric nephrology clinic in Ethiopia and factors associated with advanced chronic kidney disease: a cross-sectional study. PLOS Glob Public Health. 2024;4(11):e0003869.
20. Sawaf H, Gudura TT, Dorobisz S, Sandy D, Wang X, Bobart SA. Genetic susceptibility to chronic kidney

disease: links, risks and management. Int J Nephrol Renovasc Dis. 2023;16:1–15.
21. El-Gamasy M, Elshehaby W. Evaluation of ventricular functions in Egyptian pediatric patients with end stage kidney disease. Neonat Pediatr Med. 2018;4:156.
22. Levy Erez D, Krause I, Dagan A, Cleper R, Falush Y, Davidovits M. Impact of pediatric chronic dialysis on long-term patient outcome: single center study. Int J Nephrol. 2016;2016:2132387.
23. El-Hawy MA, Nowir AAM, Zaki SAR, El-Haruon MS. Assessment of erythroferrone levels in children with chronic kidney disease on regular hemodialysis. Voprosy gematologii/onkologii i immunopatologii v pediatrii. 2023;22(3):130–5.
24. Sharba IR, Aljabery HA, Al-Khakani MF. Eritroferrona como um novo biomarcador associado à anemia em pacientes iraquianos com DRC. Periódico Tchê Química. 2021;18(37):252–60.
25. Ketteler M, Evenepoel P, Holden RM, Isakova T, Jørgensen HS, Komaba H, et al. Chronic kidney disease–mineral and bone disorder: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int. 2025;107(3):405–23.
26. Evenepoel P, Jørgensen HS, Bover J, Davenport A, Bacchetta J, Haarhaus M, et al. Recommended calcium intake in adults and children with chronic kidney disease—a European consensus statement. Nephrol Dial Transplant. 2024;39(2):341–66.
27. Jung J, Lee KH, Park E, Park YS, Kang HG, Ahn YH, et al. Mineral bone disorder in children with chronic kidney disease: Data from the KNOW-Ped CKD (Korean cohort study for outcome in patients with pediatric chronic kidney disease) study. Front Pediatr. 2023;11:1110435.
28. Schini M, Vilaca T, Gossiel F, Salam S, Eastell R. Bone turnover markers: basic biology to clinical applications. Endocr Rev. 2023;44(3):417–73.
29. Parra-Ortega I, Zurita-Cruz JN, Villasis-Keever MA, Klünder-Klünder M, Vilchis-Gil J, Zepeda-Martinez C, et al. Cardiometabolic factors and vitamin D deficiency in pediatric patients with chronic kidney disease. Front Nutr. 2024;11:1328243.
30. Hauge SC, Hjortkjær HØ, Persson F, Theilade S, Frost M, Jørgensen NR, et al. Bone mineral density and the risk of kidney disease in patients with type 1 diabetes. J Diabetes Complications. 2025;39(2):108927.
31. Magallares B, Malouf J, Codes-Méndez H, Park HS, Betancourt J, Fraga G, et al. Pediatric densitometry: is the Z score adjustment necessary in all cases? Front Endocrinol (Lausanne). 2025;16:1373132.
32. Ananvutisombat N, Tantiworawit A, Punnachet T, Hantrakun N, Piriyakhuntorn P, Rattanathammethee T, et al. Prevalence and risk factors predisposing low bone mineral density in patients with thalassemia. Front Endocrinol (Lausanne). 2024;15:1393865.
33. LeBoff MS, Greenspan SL, Insogna KL, Lewiecki EM, Saag KG, Singer AJ, et al. The clinician's guide to prevention and treatment of osteoporosis. Osteoporos Int. 2022;33(10):2049–62.
34. Zhu X, Zheng H. Factors influencing peak bone mass gain. Front Med. 2021;15(1):53–9.
35. Iseri K, Dai L, Chen Z, Qureshi AR, Brismar TB, Stenvinkel P, et al. Bone mineral density and mortality in end-stage renal disease patients. Clin Kidney J. 2020;13(3):307–17.
36. Ginsberg C, Ix JH. Diagnosis and management of osteoporosis in advanced kidney disease: a review. Am J Kidney Dis. 2022;79(3):427–36.
37. Hajizadeh N, Mehrkash M, Fahimi D, Qorbani M, Shafa N. Association of bone mineral density with biochemical markers of bone turnover in hemodialysis children. J Renal Inj Prev. 2016;5(4):174–8.
38. Elsayed AS, Omar HR, Khater HT, Mohammed SE. Bone densitometry in children with chronic renal failure. Benha J Appl Sci. 2020;5(6 Pt 2):217–21.

Figure legend

Figure 1: Scatter dot plot showing significant negative correlation between Z score of L1-L4 and IPT and phosphorus and positive correlation between it and both vitamin D and calcium.

Figure 2: Scatter dot plot showing significant negative correlation between Z score of whole body and IPT and phosphorus and positive correlation between it and both vitamin D and calcium.

Citation

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