DOI: https://dx.doi.org/10.4314/ejpch.v21i3.2

Ethiopian Journal of Pediatrics and Child Health, 2026, 21(3)
Open access articles distributed in terms of Creative Commons Attribution Licence [CC by 4.0]
ISSN 2413-2640 *eISSN* 2519-0334

OPEN ACCESS

Original article

Mineral bone disorders in children with chronic kidney disease in a teaching hospital in Addis Ababa, Ethiopia

Woldemariam Asmare[1], Damte Shimelis[1]*

[1] Department of Pediatrics and Child Health, School of Medicine, Addis Ababa University, Addis Ababa, Ethiopia.

*Corresponding author: dshimelis2017@gmail.com

Abstract

Background *: Metabolic bone disease (MBD) in children with chronic kidney disease (CKD) is associated with high morbidity and mortality, and it has been reported that it starts early in the course of the disease and worsens as the kidney damage progresses. Therefore, we aimed to determine the magnitude and pattern of mineral bone disease among patients with chronic kidney disease on follow-up at the pediatric renal clinic of Tikur Anbessa Specialized Hospital in Addis Ababa.*

Methods*: An institution-based cross-sectional study with retrospective data collection was conducted on 86 children with CKD from stages 1 to 5. The study was conducted at Tikur Anbessa Specialized Hospital, Department of Pediatrics and Child Health, renal unit, from September 2024 to February 2025. Blood levels of Ca, P, PTH, alkaline phosphatase, and 25-Hydroxyvitamin D (25 OHD) were checked from patient records, and the prevalence of MBD according to the stages of CKD was assessed.*

Results*: The age range is from 6 months to 15 years, including 53 males and 33 females, with a male-to-female ratio of 1.6:1. The prevalence of hypocalcemia increased with CKD stages: 12.2%, 36.8%, and 71.3% for stages 3, 4, and 5, respectively. Similarly, hyperphosphatemia was observed in 24.4%, 21.1%, and 42.9% of patients across stages 3, 4, and 5. The prevalence of hyperparathyroidism was 79.3%, 86.7%, and 100% at stages 3, 4, and 5. The prevalence of high total alkaline phosphatase increased from 4% to 21.1% and 28.6% across those stages. Similarly, the prevalence of low 25-OHD rose significantly with increasing CKD stage: 71.1%, 73.7%, and 100% at stages 3, 4, and 5.*

Citation : Asmare W., Shimelis D., Mineral bone disorders in children with chronic kidney disease in a teaching hospital in Addis Ababa, Ethiopia. *Ethiop J Pediatr Child Health*. 2026;21 (3): 261-275 ***Submission date:*** *15 November 2025* ***Accepted****: 18 August 2026* ***Published****: 1 September 2026*

Conclusion: *The results demonstrated that mineral bone disorders are common in Ethiopian children with CKD in a single facility, beginning in the early stages and worsening as the disease progresses.*

Keywords: *CKD, MBD, Hyperparathyroidism, Hyperphosphatemia, Hypocalcemia.*

Background

Mineral bone disease in children with CKD is a multisystemic disorder of bone mineral metabolism caused by CKD. It can manifest with one or multiple abnormalities, such as electrolyte abnormalities (calcium and phosphorus deficiency), vitamin D deficiency, growth deficiency, bone fractures, and extra-skeletal calcification (1). Mineral homeostasis gradually deteriorates with declining kidney function, which leads to abnormal serum and tissue calcium and phosphorus levels, and hormonal imbalances in the circulation. These include, fibroblast growth factor-23 (FGF-23), parathyroid hormone (iPTH), 1, 25-dihydroxyvitamin D, and other vitamin D metabolites (2). Impaired 25(OH) D conversion to 1, 25(OH) 2 D reduces intestinal calcium absorption and increases iPTH. In the early stages of CKD, FGF-23 starts to increase, causing a reduction in phosphate excretion by the kidneys, inhibiting 1α-hydroxylase activity and, as a result, reducing the active form of Vitamin D. The kidneys do not react appropriately to FGF-23 activity or to PTH, which ordinarily encourages phosphaturia and calcium absorption (3).

Chronic kidney disease (CKD) mineral bone disorder is caused by renal insufficiency, which is linked to secondary hyperparathyroidism. This includes phosphorus retention (hyperphosphatemia) as the renal glomerular filtration rate decreases and a decline in calcitriol levels occurs due to a decrease in the metabolically active renal mass (4).

It is well known that patients with CKD stages 3a to 5 experience higher fracture rates than the general population. Additionally, incident hip fractures are linked to significant morbidity and mortality (5).

The kidneys' ability to remove phosphorus declines starting in stage 3 of CKD, leading to increased iPTH and phosphorus levels, as well as decreased 1,25 (OH)2D levels, which are associated with higher FGF-23 levels. Calcium absorption in the intestine decreases due to impaired conversion of 25 (OH) D to 1,25 (OH) 2D (6,7).

This study aimed to assess the magnitude and patterns of CKD-MBD among pre-dialysis pediatric CKD patients at Tikur Anbessa Specialized Hospital in Addis Ababa, Ethiopia.

Materials and Methods

Study design and period: Institution-based, cross-sectional study design with retrospective data collection was employed from September 2024 to February 2025.

Study Area: The study site was Tikur Anbessa Specialized Hospital (TASH) in Addis Ababa, Ethiopia. TASH is one of the oldest and largest referral and teaching hospitals in Ethiopia, affiliated with Addis Ababa University, with over 700 beds. It offers 17 specialty programs and numerous subspecialty programs, including the pediatric kidney subspecialty. This program provides training and services for referral cases from across Ethiopia. It offers inpatient and outpatient care for children with various kidney conditions.

Source population: All children who had follow-up at Tikur Anbessa Specialized Hospital pediatric renal clinic in 2024/25.

Study population: all eligible children with CKD who have follow-up at Tikur Anbessa Specialized Hospital pediatric renal clinic in 2024.

Sample size determination and Sampling procedure: Sample size was calculated based on a single-sample size estimation with a 95% confidence interval and a margin of error of 5%. P< 0.05 was considered statistically significant. Prevalence of mineral bone disease was taken as 69.1% and the sample size was 381, and by adding a 10% non-response rate, the total sample size was 420. Since the total population was less than 10,000, the adjusted sample size became 88.

Sampling technique: A convenience sampling technique was employed to select the sample population from the target /source.

Inclusion and exclusion criteria: All records of CKD patients at the pediatric renal follow-up clinic of TASH, except those with primary bone disease or traumatic fractures in the last 6 months and who were on prolonged steroid therapy, were included.

Study variables

Independent variables: Age, sex, duration of illness, Weight, Height

Dependent variables: Serum ALP, Ca, P, Vitamin D3 level, parathyroid hormone level (iPTH), and bone fracture with CKD.

Data collection technique

A pre-tested structured checklist was used to gather data. The checklist was adapted from various sources in the literature and modified to fit this study's variables and goals. The checklist was prepared in English. Data was collected by trained data collectors trained on the data collection tools.

Data quality control

A structured, validated, and reliable adapted questionnaire was used to collect data; training for data collectors and supervisors lasted half a day. To ensure the quality of the data collection tool, a pre-test was conducted on 5% of the total calculated sample size before the actual data collection. The questionnaire was checked for clarity, understandability, and simplicity. After the pre-test, the questionnaires were reviewed and reformatted based on the feedback and comments from senior nephrologists. The

completed questionnaires were checked for completeness to maintain data quality. Both the principal investigator and supervisors were responsible for on-site supportive supervision and for reviewing all completed questionnaires daily.

Data processing and analysis

The data was checked for completeness and consistency. The data were coded and entered into EpiData for cleaning, then transferred to SPSS version 25 for further analysis. Continuous variables are presented as median and mean ± standard deviation. The level of each variable according to the stage of CKD was compared with Kruskal-Wallis test. The relationship between each variable to the stage of CKD was assessed by using ANOVA. Finally, the data was processed both descriptively and analytically. Statistical significance was considered when $P<0.05$.

Descriptive statistics, including frequencies, percentages, tables were used to present the study's results.

Operational definitions

Chronic Kidney Disease (CKD): Is defined as abnormalities of kidney structure or function, present for > 3 months, with health implications. CKD is classified based on Cause and GFR category (G1-G5)

- Stage 1: GFR ≥ 90 mL/min/1.73 m² with evidence of kidney damage
- Stage 2: GFR 60-89 mL/min/1.73 m² with evidence of kidney damage
- Stage 3a: GFR 45-59 mL/min/1.73m2
- Stage 3b: GFR 30-44 mL/min/1.73m2
- Stage 4: GFR 15-29 mL/min/1.73 m²
- Stage 5: GFR < 15 mL/min/1.73 m² or on dialysis

Biochemical Mineral and Bone Disorder (MBD) in CKD is a systemic disorder of mineral and bone metabolism due to CKD, manifested by one or a combination of the following (6):

- Hypocalcemia and hypercalcemia: Defined as serum-corrected total calcium levels < 8.8 mg/dl and > 10.8 mg/dl, respectively.
- Hyperparathyroidism was categorized based on CKD stage: serum iPTH > 70 pg/ml for CKD stages 1 through 3, > 110 pg/ml for CKD stage 4, and > 300 pg/ml for CKD stage 5
- Vitamin D deficiency was defined as 25-hydroxyvitamin D < 30 ng/ml and 1,25-dihydroxyvitamin D < 60 ng/ml
- Hyperphosphatemia was determined according to age-specific normal phosphate levels: serum phosphate > 8.2 mg/dl (0–5 days), > 7.35 mg/dl (6 days to <1 year), > 6.5 mg/dl (1–3 years), > 5.6 mg/dl (4–11 years), > 5.4 mg/dl (12–15 years), and > 4.7 mg/dl (16–19 years) (12).

Results

There were 53 males out of 86 CKD patients (61.6%), with a male-to-female ratio of 1.6:1. The majority of children, 34 out of 86 (39.5%), were between five to ten years old, while 27 out of 86 (31.4%) were less than five years of age. Among children under five years with CKD, 17 out of 27 (62.9.2%) had stage III CKD, while 29.4% of children aged five to ten years had stage IV CKD. Their gender was statistically associated with their CKD stage, as shown in Table 1, with a P-value of 0.049. Among all patients, 75 (87.2%) had congenital anomalies of the kidney and urinary tract (CAKUT).

Table 2 displays the distribution and patterns of mineral bone disease across different CKD stages. Regardless of the CKD stage, the mean serum calcium level remained within the normal range, though it showed significant variation between stages (p-value=0.004). Serum iPTH levels steadily increased as CKD progressed from stage 3 to stage 5 and the variation was statistically significant (P = 0.019), and the mean serum phosphate levels (mg/dl) were 4.7, 5.2, 4.9, and 6.3 in CKD stages 3a, 3b, 4, and 5, respectively and the variation was statistically significant (P=0.016). The median value of iPTH is higher than mean value in different CKD stages because of the high variability of the results. Similarly, as CKD advanced from stage 3 to stage 5, 25-dihydroxyvitamin D3 levels declined, although this difference was not statistically significant (P=0.549). Serum alkaline phosphatase level increased as the CKD stage progresses from stage 1 to 5 and this was statistically significant (P=0.022)

Table 1: Demographic characteristics of pediatric patients attending the pediatric kidney clinic of TASH with respect to the stage of CKD, Addis Ababa, Ethiopia

Variable	**Stage I**	**Stage II**	**Stage IIIa**	**Stage IIIb**	**Stage IV**	**Stage V**	**P-value**
Age in years							
<5	0	5 (35.7%)	11	6 (37.5%)	5 (26.3%)	0	
5-10	3 (60%)	3 (21.4%)	7 (28%)	6 (37.5%)	10 (52.6%)	5 (71.1%)	0.267
>10	2 (40%)	6 (42.9%)	7 (28%)	4 (25%)	4 (21.1%)	2	
Patient number	5	14	25	16	19	7	0.241
mean± SD	8.4±4.6	8±4.35	7±4.54	7.1±4.29	6.9±4.0	8.7±2.43	
Median (min-	5 (5-14)	10 (2-14)	5 (1-17)	6.5 (1-13)	7 (1-15)	8 (6-12)	
Duration of CKD							
Patient number	5	14	25	16	19	7	0.576
mean± SD	2.6±0.8	4±2.98	2.8±1.84	2.94±1.53	3±2.38	3.7±2.06	
Median (min-max)	2 (2-4)	3 (1-10)	2 (1-8)	3 (1-6)	2 (1-10)	4 (1-6)	
Sex							
Male	3 (60%)	12 (85.7%)	16 (64%)	11 (68.8%)	10 (52.6%)	1 (14.3%)	
Female	2 (40%)	2 (14.3%)	9 (36%)	5 (31.3%)	9 (47.4%)	6	0.049

Table 2. The distribution of laboratory mineral bone disorder with respect to the stage of CKD in patients attending the pediatric renal clinic of TASH, Addis Ababa, Ethiopia

Variable	Stage I	Stage II	Stage IIIa	Stage IIIb	Stage IV	Stage V	P-value
Serum calcium mg/dl							
Patient number	5	14	25	16	19	7	0.004
mean± SD	7.56±1.03	9.53±0.80	9.28±1.04	9.3±0.60	9.54±1.1	7.7±1.66	
Median (min-max)	9(4-10)	9.7(8 -11)	9(6-11)	9(8-10)	8.9(6.1-10.1)	8(5 -10)	
Serum phosphates (mg/dl)							
Patient number	5	14	25	16	19	7	0.016
mean± SD	4.1±0.61	4.6±10.3	4.7±1.05	5.2±0.88	4.9±01.26	6.3±2.36	
Median (min-max)	4(3.2-4.9)	4.35(2.8-6.1)	4.7(3-6.7)	5(3.2-6.6)	4.9(3-7.6)	5.1(4.6-11.1)	
Serum intact parathyroid hormone (pg/ml)							
Patient number	No data	3	16	13	15	6	0.019
mean± SD	No data	91.7±67.1	182.5±227.1	108.9±65.9	265.6±266.2	504.8±321.3	
Median (min-max)	No data	120(15-140)	114.5(58.3-973)	112(32-246)	176(32-1067)	577(112-918)	
Serum 25 hydroxyvitamin D3 (ng/ml)							
Patient number	2	9	23	15	19	6	0.549
mean± SD	34±26.8	27±11.62	25.3±16.75	25.4±15.37	21.3±14.2	15.9±2.59	
Median (min-max)	34(15-53)	30.9(13-48)	19.9(4-87)	19.6 (8.1-67)	16(9-67)	16.4(11-18)	
Serum Alkaline phosphate (IU/L)							
Patient number	5	14	25	16	19	7	0.022
mean± SD	248.6±44.84	325.8±102	294.2±99.4	322.6±68.1	356.4±131.3	420.7±53.2	
Median (min-max)	256(205-316)	312(188-645)	306(82-528)	316 (215-436)	324(170-721)	415(352-529)	
Serum creatinine (mg/dl)							
Patient number	5	14	25	16	19	7	0.001
mean± SD	3.4±0.65	0.69±0.17	1.18±0.85	1.04±0.32	1.9±0.47	6.64±6.18	
Median (min-max)	0.5(0.2-15.1)	0.7(0.4-0.9)	1(0.6-5.1)	1(0.6-1.8)	2(1.2-2.6)	4.4(0.5-17)	

Table 3 shows the prevalence of MBD in relation to the stage of CKD and the treatments administered. Dialysis was done for 4 patients, 2 patients had fracture history and 4 patients had bone pain. In our study, 83% of patients developed hyperparathyroidism, 23% hypocalcemia, and 24.4% hyperphosphatemia. Among mineral bone abnormalities, hyperparathyroidism was the most commonly observed. The prevalence of hypocalcemia was not statistically significant, but it tends to increase in patients with advanced stages of CKD (20%, 14.3%, 16%, 6.3%, 36.8%, and 71.3% at CKD stages 1, 2, 3a, 3b, 4, and 5, respectively; P = 0.049). Furthermore, the prevalence of hyperparathyroidism and vitamin D deficiency significantly increased from 3a to 5 CKD stages (P = 0.012 and 0.007 respectively) which is statistically significant. Though 6/86 patients had muscle weakness the magnitude increased as the stage of CKD increased from 3a to 5 (P=001). Overall, 69.80% of patients had MBD.

Table 3: Prevalence of MBD with respect to the stage of CKD and treatments given in patients who were on follow-up at the pediatric renal clinic of TASH, Addis Ababa, Ethiopia

Variable	Stage I	Stage II	Stage IIIa	Stage IIIb	Stage IV	Stage V	P-value
Hypocalcemia	1/5 (20%)	2/14 (14.3%)	4/25 (16%)	1/16 (6.3%)	7/19 (36.8%)	5/7 (71.3%)	0.049
Hypercalcemia	0	1/14 (7.1%)	2/25 (8%)	0	0	0	
Hyperphosphatemia	0	4/14 (28.6%)	5/25 (20%)	5/16 (31.3%)	4/19 (21.1%)	3/7 (42.9%)	0.575
hyperparathyroid-ism	no data	2/3 (66.7%)	14/16 (87.5%)	9/13 (69%)	13/15 (86.7%)	6/6(100%)	0.012
Vitamin D deficien-cy	1/2 (50%)	4/9 (44.4%)	17/23 (73.9%)	10/15 (66.7%)	14/19 (73.7%)	6/6(100%)	0.007
High alkaline phos-phate	no data	1/14 (7.1%)	1/25 (4%)	0	4/19 (21.1%)	2/7 (28.6%)	0.099
Muscle weakness							
Yes	0	0	3(12%)	0	2 (10.5%)	4(57.1%)	
No	5(6.5%)	14 (18.2%)	22(88%)	16 (100%)	17 (89.5%)	3(42.9%)	0.001
Treatments							
Phosphate binder							
Yes	0	0	2(8%)	4(25%)	1(5.3%)	2(28.6%)	0.110
No	5(100%)	14(100%)	23(92%)	12(75%)	18 (94.7%)	5(71.4%)	
Vitamin D analogs							
Yes	1(20%)	4(28.6%)	17(68%)	10 (62.5%)	15 (78.9%)	6(85.7%)	0.012
No	4(80%)	10 (71.4%)	8(32%)	6(37.5%)	4 (12.1%)	1(14.3%)	
Low phosphate diet							
Yes	0	1(7.1%)	4(16%)	6(37.5%)	5 (26.3%)	4(57.1%)	0.057
No	5(100%)	13 (92.9%)	21(84%)	10 (62.5%)	14 (73.7%)	3(42.9)	
Calcium supplements							
Yes	1(20%)	4(28.6%)	13(52%)	10 (62.5%)	13 (68.4%)	5(71.4%)	0.113
No	4(80%)	10 (71.4%)	12(48%)	6(37.5%)	6 (31.6%)	2(28.6%)	

Discussion

MBD is a significant complication that requires careful monitoring in pediatric patients with CKD. In our study, 53 out of 86 (61.6%) participants were boys, with a male-to-female ratio of 1.6:1, similar to previous studies in children, as congenital urologic abnormalities are more common in boys (9,10). The median serum calcium concentration remained fairly stable across different CKD stages, except in cases of end-stage renal disease, where it measured 9, 9.7, 9, 8.9, and 8 from stage 1 to stage 5, respectively. The consistent serum calcium levels until the eGFR dropped below 15 ml/min/1.73 m^2 align with findings from a previous study on pediatric CKD patients, reporting values of 9.2, 9.3, 9.3, 9.3, 9.5, and 9.6 from stages 1 to 5, respectively (11,12). In our study, the prevalence of hypocalcemia was 23%, slightly higher than the 16% reported in a study from Korea. (13). A study from India observed hypocalcemia in 21.5%, 33.9%, and 48.9% of CKD stage 3, 4, and 5 cases, respectively (3). In our setting, the prevalence was 12.1%, 36.8%, and 71% in stages 3, 4, and 5, respectively. The higher prevalence of hypocalcemia in our setup may be due to lower dietary calcium intake, fewer prescriptions of calcium supplementation, and patients presenting later to the clinic.

However, when analyzing the proportions of patients with hypercalcemia and hypocalcemia separately, both show an increasing trend across CKD stages, with hypocalcemia being more common. This finding aligns with research in adult CKD, where hypocalcemia usually appears in the later stages (6,7) due to increased iPTH levels starting from stage 3.

In our study, hyperphosphatemia was observed in 23.8%, 21%, and 42% of cases in CKD stages 3, 4, and 5, respectively, which aligns with findings from a Korean cohort study (hyperphosphatemia: 17.39%, 23.68%, and 41.18% in CKD stages 3–5, respectively) and a single-center study in India (hyperphosphatemia: 13.8%, 26.4%, and 63.4% in CKD stages 3, 4, and 5, respectively (3). Another cross-sectional study by Behra T.R et al. from India showed serum phosphate levels of 11.1%, 25.5%, and 63% from stages 3 to 5, respectively, which is similar to our findings (13). Our cross-sectional study highlighted an inverse relationship between serum phosphate levels and eGFR, with the highest serum phosphate levels seen in stage 5 CKD. The higher median phosphate levels in advanced CKD stages might be influenced by differences in age distribution, which affect baseline phosphate levels, as well as the smaller sample size in stage 5 CKD.

In our study, the median iPTH level increased significantly with advancing CKD stages, particularly in stages 4 and 5, which aligns with established data on hyperparathyroidism in CKD stages 4–5 from Indian and Korean groups (11,13). However, the prevalence of

hyperparathyroidism was high across CKD stages 2 to 5, recorded at 66.7%, 87.5%, 69%, 86.7%, and 100% in stages 2, 3a, 3b, 4, and 5, respectively. These results are also similar to a study by Borisa K.D. in India, where hyperparathyroidism was seen in 66% of CKD stage 4 patients and 89.4% of CKD stage 5 patients, as well as another Indian study by Behra T.R. et al (3, 13).

However, in the Korean cohort, parathyroid hormone levels increased from CKD stages 1 through 3b and remained stable in stages 4 and 5 (11). In our study, no iPTH samples were collected for stage 1 CKD, which resulted in the lowest recorded value. A study on adult patients in Tanzania who were on maintenance hemodialysis, with some not on dialysis, showed a progressive increase in iPTH levels as kidney disease advanced (12). The highest prevalence in stage 5 CKD may be due to the smaller sample size and the fact that iPTH was measured for all stage 5 patients at a late stage of presentation. Two adult studies from Nigeria on non-dialytic CKD patients, as well as studies on hemodialysis patients from Lahore and South Africa, showed a high prevalence of mineral bone disease as the disease progressed (11, 15, 16). Another adult study in South Africa indicated a higher prevalence of mineral bone disease in Black patients compared to White patients at a younger age, with higher median levels of iPTH and vitamin D (17).

Studies have shown that CKD is linked to low levels of 25(OH) vitamin D, decreased 1,25 $(OH)_2$ vitamin D (calcitriol), and vitamin D resistance (10). In our study, serum 25-hydroxy vitamin D deficiency remained high across all stages of CKD (>44.4% in each stage), with its prevalence increasing as the disease progressed. Low 25(OH)D3 levels were found in 70.2% of patients, similar to the 73.6% reported in a study conducted at a teaching hospital in central India (3). However, this prevalence is lower than the 88% reported by Moorani K.N. et al. in children with CKD in Pakistan (10). Another study by Kari J.A., et al. in children with CKD in Saudi Arabia showed 45% of their CKD patients had vitamin D insufficiency, 30% vitamin D deficiency, and 12.5% had severe deficiency. Their study also indicated a positive correlation between low vitamin D levels and the stage of CKD, which aligns with our findings (18).

However, these results are significantly higher compared to international data. Stein D. R. et al. reported that among 100 American children with CKD, 16% were deficient in 25 (OH) D (≤20 ng/mL), 24% were insufficient (≤30 ng/mL), and 40% had suboptimal levels (9). The higher prevalence of vitamin D deficiency in CKD patients in our setting may be due to additional factors such as primary malnutrition and limited access to timely and accessible healthcare facilities.

Elevated total ALP levels suggest high-turnover bone disease and should be interpreted within the proper clinical context. . In this

study, elevated ALP was observed in 21.1% of patients with CKD stage 4 and in 28.6% of patients with stage 5. Conversely, only two patients in stages 1 to 3 had increased ALP levels in our setting. KDIGO guidelines recommend managing mineral and bone disorder (MBD) based on trends in biochemical parameters rather than isolated abnormalities (8). An Indian study reported raised ALP levels in 5.9% and 45.7% of patients with CKD stages 4 and 5, respectively (3), and it shared similarities with our study, as both observed the highest ALP levels at end-stage renal disease. A Senegalese study by Seck S.M. et al. and a Nigerian study by Abdu A. et al. on adult CKD patients, along with similar research, showed a high prevalence of mineral bone disease and recommended early dialysis and medical treatment to prevent complications (19-21). This Ethiopian study in pre-dialytic pediatric CKD patients contributes to filling the gap in the knowledge and development of contextualized management guideline for CKD-MBD.

This study has some limitations because it is a single-center study, no assessment of dietary intake or socioeconomic factors, and small sample size for advanced stages. but the results could be extrapolated to similar institutions in Sub-Saharan Africa.

Conclusion and recommendation

We observed that low 25(OH)D levels, hyperparathyroidism, and hyperphosphatemia were key indicators of CKD-MBD in our patients. Mineral bone disorders are common in CKD and begin early, worsening as the disease advances, which increases morbidity and decreases quality of life. This emphasizes the importance of early detection and the development of management strategies to prevent progression, ultimately reducing morbidity and mortality. Patients with CKD should have regular assessments of phosphate, calcium, and iPTH levels to enable early diagnosis and treatment of metabolic bone disease.

Declarations

Ethical Considerations

Ethical approval was secured from the Pediatrics and Child Health Department's Research and Publications Committee of the School of Medicine, College of Health Sciences, and Addis Ababa University. All participants in the study were kept anonymous during analysis and dissemination.

Conflict of interest

The authors declare that there is no competing interest.

Authors contribution

WA drafted the primary idea, wrote the proposal, collected data and did the primary analysis, and write-up. DS synthesized and enriched the primary idea, reviewed the proposal, analyzed data, and did the final write-up.

Funding

This study was funded by the College of Health Sciences of Addis Ababa University as part of postgraduate research program. The college did not have any role in the study.

Acknowledgement

We would like to thank the Department of Pediatrics and Child Health of Addis Ababa University for their support during the study and the renal team for their unreserved cooperation.

Reference

1. Wesseling-Perry K, Salusky I.B. Chronic kidney disease: mineral and bone disorder in children. Semin Nephrol. 2013 Mar;33 (2):169-79. doi: 10.1016/ j.semnephrol.2012.12.017. PMID: 23465503; PMCID: PMC4209124

2. Rukshana C Shroff. Chronic Kidney Disease-Renal Bone Disorder. In Pediatric Kidney Disease. Claus P Schmitt, Rukshana C Shroff, eds. P 2-40. Aug 2017.

3. Borisa, K.D., Bone disorders in chronic kidney disease patients: A teaching hospital -based study in central India. IJARM 2019; 1(2): 151-156. DOI: https:// doi.org/10.22271/27069567.2019.v1.i2b.42 4

4. Nasim A., Rafique Z., Talal A., Afzal A., Asrar A, Prevalence of Bone Mineral Disorder in Hemodialysis patients: A Single Centered Study of Local Population. PJMHS 2022; 16 (07), 134-136; DOI: https://doi.org/10.53350/ pjmhs22167134

5. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024 Apr;105(4S): S117-S314. doi: 10.1016/ j.kint.2023.10.018. PMID: 38490803

6. Langman CB, Salusky IB. K/DOQI clinical practice guidelines for bone metabolism and disease in children with chronic kidney disease. Am J Kidney Dis. (2005) 46(4): S48–64. doi: 10.1053/ j.ajkd.2005.07.028

7. Esfandiar N., Shakiba M., Mirzaei, Z. Vitamin D Deficiency in Children with Chronic Kidney Disease. Journal of Clinical & Diagnostic Research 2019; 13 (2), SC13-SC16. DOI: 10.7860/ JCDR/2019/38044.12633

8. Okoye, J.U., Arodiwe, E.B., Ulasi, I.I., Ijoma, C.K., Onodugo, O.D. Prevalence of CKD-MBD in pre-dialysis patients using biochemical markers in Enugu, South-East Nigeria. African health sciences 2015, 15 (3), 941-48.

9. Stein D.R., Feldman H.A, Gordon C.M. Vitamin D status in children with chronic kidney disease. Pediatr Nephrol. 2012 Aug;27(8):1341-50. doi:10.1007/s00467-012-2143-7.

10. Kayani A, Jamali A.A, Moorani K.N, Fatima S, Jamil A. Vitamin D deficiency in children with Chronic Kidney Disease-Mineral Bone Disorder (CKD-MBD) and factors affecting response to cholecalciferol therapy: A quasi-experimental study from low-middle income setting. Caspian J Pediatrs Mar 2022; 8(1): 615-24.

11. RajiY.R., Ajayi S.O., Adeoye A.M., Amodu O., Tayo B.O., Salako B.L., 2022. Fibroblast Growth Factor 23 (FGF 23) and intact parathyroid hormone (iPTH) as markers of mineral bone disease among Nigerians with non-diabetic kidney disease. African Health Sciences, 22(1), 344-51. https://dx.doi.org/10.4314/ahs.v22i1.42

12. Mungulluh F. F., Ruggajo P., Furia F.F. Mineral Bone Disease in Chronic Kidney Disease Patients (CKD-MBD) and its Associated Factors at Muhimbili National Hospital in Dar as Salaam, Tanzania: A Cross-sectional Study. AJRN 2021, 4(1): 36-49,

13. Jung J, Lee K.H, Park E., Park Y.S., Hee K.G., AhnY.H.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. Frontiers in pediatrics 17 February 2023, DOI 10.3389/fped.2023.994979, 1-9

14. Behera T.R., Mohanty B., Sahu A., Naik S. Mohanty J.N.. Prevalence of Mineral Bone Disorders in Chronic Kidney Disease Patients. Academia Journal of Medicine 2019; 2(2), pp.60-63.

15. Nasim A., Rafique Z., Talal A., Afzal A., Asrara S. Prevalence of Bone Mineral Disorder in Hemodialysis patients: A Single-Centered Study of Local Population. J M H S July 2022; DOI: https://doi.org/10.53350/pjmhs221671

16. Yokoyama J.S., Matsuda A.M., Denburg M.R., Kumar J., Warady B.A., Furth S.L. et al., Association between chronic kidney disease–mineral bone disease (CKD-MBD) and cognition in children: Chronic Kidney Disease in Children (CKiD) Study. Kidney medicine 2020, 2(4), pp.398-406.

17. Waziri B., Duarte R., Dickens C., Peek T.D., George J., Rekhviashvili V., Paget G. Naicker S. Racial variations in the markers of mineral bone disorders in CKD patients in South Africa. Kidney international reports, 2018;3(3), pp.583-591

18. Kari J.A., El-Desoky S.M, El-Morshedy S.M, Habib H.S. Vitamin D insufficiency and deficiency in children with chronic kidney disease. Ann Saudi Med 2012; 32 (5): 473-8

19. Seck SM, Dahaba M, Ka EF, Cisse MM, Guéye S, Tal AOL. Mineral and Bone Disease in Black African Hemodialysis Patients: A Report from Senegal. Nephro-Urol Mon. 2012;4(4):613-16. DOI: 10.5812/numonthly.4225

20. Abdu A., Abdu A., Arogundade F.A. Prevalence and Pattern of Chronic Kidney Disease-Mineral Bone Disorders among Hemodialysis Patients in Kano, Northwest Nigeria. Ann Afr. Med 2019 Oct-Dec;18(4):191–195. doi: 10.4103/aam.aam_18_19

21. Melkeneh S., Melkie A. Magnitude and Determinants of Biochemical Mineral Bone Disease Abnormalities among Predialysis Chronic Kidney Disease Patients in Tikur Anbessa Specialized Hospital. EMJ 2025, 63(1); 300-9. https://dx.doi.org/10.4314/emj.v63i1.2.