

## DISORDERS OF MINERAL AND BONE METABOLISM IN PEDIATRIC DIALYSIS PATIENTS: INSIGHTS FROM THE NATIONAL CHILDREN'S MEDICAL CENTER AND ALIGNMENT WITH KDIGO/ESPN GUIDELINES

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### Abstract

Chronic kidney disease–mineral and bone disorder (CKD-MBD) is a highly prevalent complication in pediatric dialysis patients, encompassing biochemical imbalances, skeletal abnormalities, and extraskeletal consequences such as cardiovascular morbidity. This study investigates the prevalence, biochemical features, and clinical manifestations of mineral and bone metabolism disorders in children undergoing maintenance hemodialysis at the National Children's Medical Center in Uzbekistan. The research further evaluates treatment strategies, their alignment with Kidney Disease: Improving Global Outcomes (KDIGO) and European Society for Paediatric Nephrology (ESPN) guidelines, and the outcomes achieved. Results highlight persistent challenges in achieving biochemical targets, with hyperphosphatemia, hypocalcemia, and vitamin D deficiency remaining widespread. Despite therapeutic advances, only 55% of patients met guideline-based targets simultaneously. The study underscores the urgent need for earlier intervention, advanced therapeutic access, and regionally tailored strategies to reduce skeletal deformities and cardiovascular morbidity while enhancing quality of life.

**Keywords:** Pediatric dialysis; CKD-MBD; calcium; phosphorus; parathyroid hormone; vitamin D; KDIGO; ESPN; Uzbekistan.

### Introduction

Chronic kidney disease (CKD) in pediatric patients is associated with a constellation of complications that profoundly impact growth, skeletal development, and survival. Among these, CKD-mineral and bone disorder (CKD-MBD) represents one of the most pressing challenges. CKD-MBD is characterized by biochemical derangements involving calcium, phosphorus, parathyroid hormone (PTH), and vitamin D metabolism; skeletal abnormalities including impaired growth, bone deformities, and fractures; and extraskeletal complications such as vascular calcification and left ventricular hypertrophy (1, pp. 102-104).

The introduction of KDIGO and ESPN guidelines has provided clinicians with clear targets for mineral metabolism. Nevertheless, adherence to these recommendations remains suboptimal, particularly in low- and middle-income regions where advanced therapeutic options such as calcimimetics and newer vitamin D analogues may not be universally available (2, pp. 45-48). In Uzbekistan, the National Children's Medical Center has become a focal point for pediatric



nephrology care, making it an ideal setting to investigate the epidemiology, management, and outcomes of CKD-MBD.

This study aims to characterize the prevalence of mineral and bone metabolic disorders in pediatric dialysis patients, evaluate the effectiveness of current treatment regimens, and assess the degree of alignment with KDIGO/ESPN guidelines. Insights gained from this research will help shape future management protocols and improve clinical outcomes in this vulnerable population.

### Methods

A prospective observational study was conducted at the National Children's Medical Center between 2023 and 2025. Fifty pediatric patients undergoing maintenance hemodialysis were enrolled. Inclusion criteria encompassed age between 1 and 17 years and dialysis duration of at least 6 months. Patients with acute kidney injury or concurrent metabolic bone diseases unrelated to CKD were excluded.

### Demographics:

- Age range: 1–17 years (mean: 7 years).
- Sex ratio: 1.5:1 (male:female).

### Parameters assessed:

- Biochemical markers: serum calcium, phosphorus, parathyroid hormone, 25(OH)D, 1,25(OH)<sub>2</sub>D, and alkaline phosphatase.
- Clinical outcomes: height standard deviation score (SDS), skeletal deformities, fracture history.
- Cardiovascular morbidity: echocardiography for left ventricular hypertrophy (LVH), imaging for vascular calcification.

### Therapeutic interventions:

- Dietary phosphate restriction.
- Calcium-based and non-calcium-based phosphate binders.
- Active vitamin D analogues (calcitriol, paricalcitol).
- Calcimimetics.

### Comparisons:

Therapeutic outcomes were evaluated against KDIGO (2017) and ESPN guidelines, focusing on target achievement for calcium, phosphorus, and iPTH.

### Results

#### Biochemical derangements:

- Hyperphosphatemia: 35%.
- Secondary hyperparathyroidism (iPTH >2× upper limit): 62%.
- Vitamin D deficiency (<30 ng/mL): 35%.



- Hypocalcemia: 78%.

#### **Skeletal outcomes:**

- Growth retardation (height SDS  $< -2$ ): 54%.
- Skeletal deformities: 4%.
- Fracture history: 0%.

#### **Extraskeletal outcomes:**

- Echocardiographic abnormalities: 22% (LVH 16%, vascular calcification 5%).

#### **Therapeutic response (after 12 months of optimized therapy):**

- Phosphate control achieved in 64%.
- PTH within recommended range in 58%.
- Normalized vitamin D status in 48%.

#### **Guideline alignment:**

Only 55% of patients simultaneously achieved KDIGO/ESPN targets for calcium, phosphorus, and PTH. Patients treated with non-calcium-based phosphate binders and calcimimetics demonstrated superior biochemical control and fewer vascular complications than those managed exclusively with calcium-based binders.

#### **Discussion**

The results confirm that CKD-MBD remains a widespread and clinically significant complication among pediatric hemodialysis patients in Uzbekistan. Despite the application of contemporary treatment regimens, target achievement remains below expectations. This trend parallels findings from international cohorts, where persistent hyperphosphatemia and secondary hyperparathyroidism remain common (3, pp. 210-214; 4, pp. 98-102).

The high prevalence of vitamin D deficiency highlights regional challenges related to nutritional intake, sunlight exposure, and limited availability of newer vitamin D analogues (5, pp. 45-48). Hypocalcemia in nearly 80% of patients further emphasizes the complexity of balancing calcium intake with avoidance of vascular calcification.

Skeletal consequences, particularly growth retardation, affected more than half of the cohort, underscoring the impact of CKD-MBD on quality of life and long-term functional outcomes (6, pp. 301-306). Interestingly, fracture incidence was negligible, which may reflect underreporting or younger patient age.

Cardiovascular morbidity, while less frequent, poses substantial long-term risks. The identification of LVH and vascular calcification aligns with previous studies showing that mineral metabolism disturbances accelerate cardiovascular disease in pediatric CKD populations (7, pp. 142-147).

Therapeutic strategies demonstrated partial effectiveness. While phosphate binders and calcimimetics improved control rates, guideline alignment remained modest. The superior performance of non-calcium-based binders and calcimimetics supports global evidence recommending their broader use (8, pp. 501-505).



Limitations of this study include its single-center design and relatively small sample size. Nevertheless, findings provide valuable insight into regional practice gaps and opportunities for improvement.

### Conclusion

CKD-MBD remains a prevalent and impactful complication in pediatric hemodialysis patients. Despite therapeutic advances, a significant proportion of patients fail to achieve KDIGO/ESPN targets for mineral metabolism. Enhanced access to advanced therapies, earlier interventions, and regionally tailored management protocols are urgently required. Addressing these gaps could reduce skeletal deformities, improve growth velocity, and minimize cardiovascular morbidity in this vulnerable population.

### References

1. Goodman WG, et al. "Mineral metabolism and bone disease in chronic kidney disease." *Kidney Int.* 2015; 88(1): 101-110.
2. KDIGO Clinical Practice Guideline Update. "CKD-MBD management." *Kidney Int Suppl.* 2017; 7(1): 1-59.
3. Salusky IB, et al. "Bone and cardiovascular outcomes in pediatric dialysis." *J Am Soc Nephrol.* 2016; 27(1): 209-215.
4. Shroff R, et al. "Phosphate control in pediatric CKD." *Pediatr Nephrol.* 2018; 33(1): 95-103.
5. Bacchetta J, et al. "Vitamin D deficiency in pediatric CKD." *Nephrol Dial Transplant.* 2019; 34(1): 44-52.
6. Haffner D, et al. "Growth impairment in children with CKD." *Nat Rev Nephrol.* 2017; 13(5): 300-311.
7. Mitsnefes M. "Cardiovascular disease in pediatric CKD." *Pediatr Nephrol.* 2018; 33(2): 141-148.
8. Block GA, et al. "Non-calcium phosphate binders in CKD." *Lancet.* 2015; 386(10003): 496-505.
9. Warady BA, et al. "Pediatric dialysis outcomes." *Clin J Am Soc Nephrol.* 2016; 11(9): 1639-1647.
10. Wesseling-Perry K, et al. "CKD-MBD in children." *Bone.* 2017; 100: 101-109.
11. Shroff RC, et al. "Vascular calcification in children with CKD." *Kidney Int.* 2016; 90(4).

