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Original Research Article

Stage-specific patterns of FGF23 and CKD-mineral bone disorder biomarkers in children with predialysis chronic kidney disease: a cross-sectional study

Shabnam Sharmin[1]*, Latifa Akter Shumi[2]

[1]Department of Paediatric Nephrology, Shaheed Suhrawardi Medical College Hospital, Dhaka, Bangladesh
[2]Department of Pediatrics, Shaheed Suhrawardi Medical College Hospital, Dhaka, Bangladesh

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***Correspondence:**
Dr. Shabnam Sharmin,
E-mail: kariul@hotmail.com

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ABSTRACT

Background: Chronic kidney disease-mineral and bone disorder (CKD-MBD) begins early in pediatric CKD, yet conventional biochemical markers often fail to detect early disturbances. Fibroblast growth factor-23 (FGF23) has emerged as a potential early regulator of mineral metabolism.
Methods: This cross-sectional study included 32 children aged 2-18 years with predialysis CKD stages 2–5. Plasma FGF23, serum calcium, phosphate, parathyroid hormone (PTH), and vitamin D were measured. CKD-MBD was defined based on abnormalities in mineral metabolism parameters. Stage-wise comparisons of FGF23 between children with and without CKD-MBD were performed, and correlations between FGF23, phosphate, and PTH were analyzed.
Results: Mean FGF23 concentrations increased progressively with CKD stage and were significantly higher in children with CKD-MBD at every stage, beginning in stage 2 (102±4.05 vs. 88±2.09 RU/ml; p=0.042) and peaking in stage 5 (345±15.01 vs. 185±4.03 RU/ml; p=0.011). FGF23 showed a moderate positive correlation with PTH (r=0.535, p=0.006) and a strong positive correlation with serum phosphate (r=0.638, p<0.001).
Conclusions: FGF23 rises early in predialysis pediatric CKD, increases in a stage-dependent manner, and is strongly associated with phosphate and PTH. These findings support FGF23 as a sensitive early biomarker of CKD-MBD in children.

Keywords: Fibroblast growth factor-23, Chronic kidney disease, CKD-mineral bone disorder, Pediatric nephrology, Predialysis CKD

INTRODUCTION

Chronic kidney disease (CKD) is increasingly recognized as a major global health problem that also affects pediatric populations, with growing evidence that metabolic disturbances emerge long before kidney failure develops.[1] Among these, CKD-MBD represents one of the most significant and systemic complications, involving biochemical, skeletal, and vascular abnormalities that worsen with disease progression.[2,3] The disease process begins early in predialysis CKD, when subtle disruptions in phosphate handling, calcium homeostasis, and vitamin D metabolism progressively activate secondary endocrine responses, leading to disordered bone turnover and impaired growth in children.[1,4]

Traditionally, CKD-MBD has been assessed through measurements of serum calcium, phosphate, parathyroid hormone (PTH), and vitamin D metabolites. However,

these conventional biomarkers often fail to detect early abnormalities, since phosphate and PTH remain within normal limits until later CKD stages.[2,5] This diagnostic lag contributes to late recognition and inadequate early intervention, especially in children with slowly progressive disease. Recent findings indicate that fibroblast growth factor 23 (FGF23), a phosphaturic hormone secreted by osteocytes, rises much earlier than these classical markers and therefore may serve as a sensitive early indicator of mineral dysregulation in CKD.[4,5]

FGF23 plays a pivotal role in mineral metabolism by enhancing urinary phosphate excretion and suppressing 1α-hydroxylase, thereby reducing the synthesis of active vitamin D [1,25$(OH)_2$D].[6,7] Through these actions, FGF23 links bone and kidney physiology as part of an endocrine feedback loop involving the co-receptor α-Klotho, maintaining phosphate balance under normal circumstances.[6,8] In CKD, declining renal function reduces Klotho expression, leading to FGF23 resistance and progressive accumulation of the hormone. This maladaptive response contributes not only to secondary hyperparathyroidism and bone demineralization but also to cardiovascular complications such as left ventricular hypertrophy and vascular calcification.[9,10]

The pathophysiological relevance of FGF23 extends beyond mineral metabolism, with evidence linking elevated levels to increased mortality, cardiovascular risk, and bone fragility in both adult and pediatric CKD.[11,12] These associations emphasize the need for early, stage-specific monitoring of FGF23 in CKD patients before overt biochemical or structural abnormalities appear. Several investigations have demonstrated that serum FGF23 increases progressively from CKD stage 2 onward, preceding measurable changes in calcium, phosphate, or PTH, particularly in children.[2,4,13] Furthermore, both FGF23 and Klotho concentrations have been shown to correlate with bone microarchitectural deterioration and trabecular bone score in early CKD, even when bone mineral density remains unchanged.[14]

Despite these advances, pediatric data remain limited compared with adult studies, leaving significant gaps in understanding the developmental differences in CKD-MBD pathophysiology.[10] Most evidence arises from adult or mixed-age cohorts, and extrapolation to children is problematic given their distinct skeletal dynamics, growth velocity, and hormonal milieu.[1] Moreover, regional and ethnic variations further influence CKD-MBD patterns and biomarker kinetics, highlighting the need for population-specific evaluation.[11,15] Therefore, systematic characterization of FGF23 and related biochemical markers across CKD stages in children, particularly during the predialysis period, remains an unmet research priority.

The predialysis stage offers a critical therapeutic window in CKD-MBD management, as interventions during this period may prevent irreversible skeletal and cardiovascular sequelae.[3,9] Early-stage identification of dysregulated mineral metabolism allows dietary phosphate control, vitamin D optimization, and pharmacologic interventions before overt bone or vascular damage occurs.[16,17] This approach aligns with emerging evidence that integrated biomarker assessment, combining FGF23 with classical CKD-MBD parameters, enhances prediction of renal decline and systemic complications.[18]

Given these observations, the present study aims to explore the stage-specific behavior of FGF23 and CKD-MBD biomarkers among children with predialysis CKD, providing quantitative insights into early biochemical derangements and their interrelationships. By delineating these trajectories, this research seeks to strengthen the evidence base for biomarker-driven, stage-tailored CKD-MBD evaluation in pediatric nephrology, addressing a key gap between pathophysiologic understanding and clinical application.

METHODS

This cross-sectional study was conducted between August 2020 and February 2022 in the Department of Paediatric Nephrology, in collaboration with the Departments of Biochemistry and Microbiology and Immunology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka. Children aged 2–18 years of both sexes with predialysis chronic kidney disease (CKD stages 2-5) attending inpatient or outpatient services were enrolled using purposive sampling. CKD was defined according to KDIGO criteria as abnormalities of kidney structure or function persisting for more than three months. Children receiving dialysis, those on active calcium or vitamin D supplementation, and those whose parents or guardians declined consent were excluded. A total of 32 children were included, exceeding the calculated minimum sample size of 26. Estimated glomerular filtration rate (eGFR) was calculated using the revised Schwartz formula, and participants were categorized into CKD stages 2 (eGFR 60-89 ml/min/1.73 m^2), stage 3 (30-59 ml/min/1.73 m^2), stage 4 (15-29 ml/min/1.73 m^2), and stage 5 (<15 ml/min/1.73 m^2). Baseline demographic data, clinical history, anthropometric measurements, and blood pressure were recorded using a structured data sheet; hypertension was defined according to age-, sex-, and height-specific American Academy of Pediatrics guidelines. Five milliliters of venous blood were collected aseptically, with plasma separated in EDTA tubes and serum obtained from plain tubes. Serum creatinine, calcium, phosphate, parathyroid hormone (PTH), and 25-hydroxyvitamin D were analyzed in the BSMMU Biochemistry Laboratory using automated chemistry analyzers, while plasma fibroblast growth factor-23 (FGF23) concentrations were measured using a

commercially available C-terminal enzyme-linked immunosorbent assay, with results expressed in relative units per milliliter (RU/ml). CKD-mineral bone disorder (CKD-MBD) was defined by the presence of abnormalities in serum calcium, phosphate, PTH, and/or vitamin D, in accordance with KDIGO guidelines. Data were analyzed using SPSS version 22; continuous variables were expressed as mean±standard deviation and categorical variables as frequencies and percentages. Comparisons of FGF23 levels between CKD patients with and without CKD-MBD across stages were performed using unpaired Student's t-tests, and associations between FGF23 and biochemical markers were assessed using Pearson correlation coefficients. A p value <0.05 was considered statistically significant. Ethical approval was obtained from the Institutional Review Board of BSMMU, and written informed consent was secured from parents or legal guardians prior to participation.

RESULTS

In this predialysis pediatric CKD cohort (n=32), the mean age was 9.48±3.06 years, with stage-wise mean ages ranging from 8.00±3.03 years in CKD stage 3 to 11.33±3.16 years in stage 2. Overall, males predominated, 20 (62.5%), and this male excess persisted across stages, increasing from 4 (57.1%) in stage 2 and 5 (55.6%) in stage 3 to 6 (75.0%) in stage 5, while females comprised 12 (37.5%) of the total sample. Mean BMI was 22.0±3.3 kg/m² overall, with slightly higher values in stages 2 and 3 (24.0±1.3 and 24.0±1.2 kg/m², respectively) compared with stages 4 and 5 (both 22.0±3.3 kg/m²). Hypertension was common, affecting 22 (68.8%) participants overall, and demonstrated a clear stage-related gradient, rising from 2 (28.6%) in stage 2 and 5 (55.6%) in stage 3 to 7 (87.5%) in stage 4 and 8 (100.0%) in stage 5.

Table 1: Baseline demographic and clinical characteristics of children with predialysis CKD by stage (n=32).

Characteristics	Total (n=32)	CKD Stage 2 (n=7)	CKD Stage 3 (n=9)	CKD Stage 4 (n=8)	CKD Stage 5 (n=8)
Age, years, mean±SD	9.48±3.06	11.33±3.16	8.00±3.03	9.22±3.19	9.22±3.07
Male sex, N (%)	20 (62.5)	4 (57.1)	5 (55.6)	5 (62.5)	6 (75.0)
Female sex, N (%)	12 (37.5)	3 (42.9)	4 (44.4)	3 (37.5)	2 (25.0)
BMI, kg/m², mean±SD	22.0±3.3	24.0±1.3	24.0±1.2	22.0±3.3	22.0±3.3
Hypertension, N (%)	22 (68.8)	2 (28.6)	5 (55.6)	7 (87.5)	8 (100.0)

Table 2: Etiology of chronic kidney disease by CKD stage.

Etiology	Total (n=32)	Stage 2 (n=7)	Stage 3 (n=9)	Stage 4 (n=8)	Stage 5 (n=8)
	N (%)	N (%)	N (%)	N (%)	N (%)
Obstructive uropathy	12 (37.5)	2 (28.6)	1 (11.1)	4 (50.0)	5 (62.5)
Congenital anomalies of kidney and urinary tract (CAKUT)	8 (25.0)	3 (42.9)	1 (11.1)	2 (25.0)	2 (25.0)
Glomerulonephritis	6 (18.8)	2 (28.6)	2 (22.2)	1 (12.5)	1 (12.5)
Cystic kidney disease	4 (12.5)	2 (28.6)	1 (11.1)	1 (12.5)	0 (0.0)
Other causes	2 (6.2)	0 (0.0)	0 (0.0)	0 (0.0)	2 (25.0)

Table 3: Stage-wise comparison of plasma FGF23 concentrations in children with predialysis CKD with and without mineral bone disorder.

CKD Stage (eGFR, ml/min/1.73 m²)	FGF23 (RU/ml), mean ± SD		P value
	CKD without MBD (n=12)	CKD with MBD (n=20)	
Stage 2 (60-89)	88±2.09	102±4.05	0.042
Stage 3 (30-59)	99±9.09	132±1.02	0.034
Stage 4 (15-29)	110±5.05	242±10.02	0.031
Stage 5 (<15)	185±4.03	345±15.01	0.011

Across the 32 children with predialysis CKD, obstructive uropathy was the most frequent etiology, present in 12 (37.5%) cases, and showed a stage-related concentration toward advanced disease, increasing from 2 (28.6%) in stage 2 and 1 (11.1%) in stage 3 to 4 (50.0%) in stage 4 and 5 (62.5%) in stage 5. Congenital anomalies of the kidney and urinary tract (CAKUT) accounted for 8 (25.0%) cases, with the highest proportion in stage 2, 3 (42.9%), and a relatively even distribution thereafter, 1 (11.1%) in stage 3 and 2 (25.0%) each in stages 4 and 5. Glomerulonephritis contributed 6 (18.8%) cases, appearing across all stages but at lower proportions in

stages 4 and 5, 1 (12.5%) each, compared with stages 2 and 3, 2 (28.6%) and 2 (22.2%). Cystic kidney disease represented 4 (12.5%) cases, most commonly in stage 2, 2 (28.6%), and was absent in stage 5. Other causes were uncommon overall, 2 (6.2%), and were observed only in stage 5, 2 (25.0%).

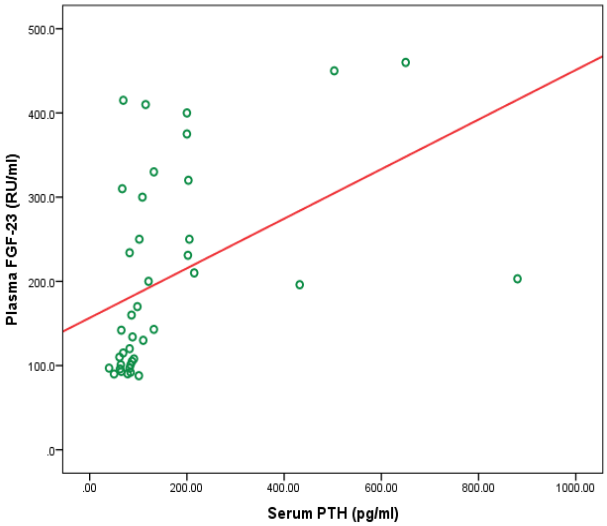


Figure 1: Scatter diagram showing correlation of plasma FGF-23 with serum PTH (r=0.535, p=0.006); plasma FGF-23 showed a statistically significant, moderate positive correlation with serum PTH (r= 0.535, p=0.006), indicating that higher PTH levels were generally associated with higher circulating FGF-23 in this predialysis pediatric CKD COHORT.

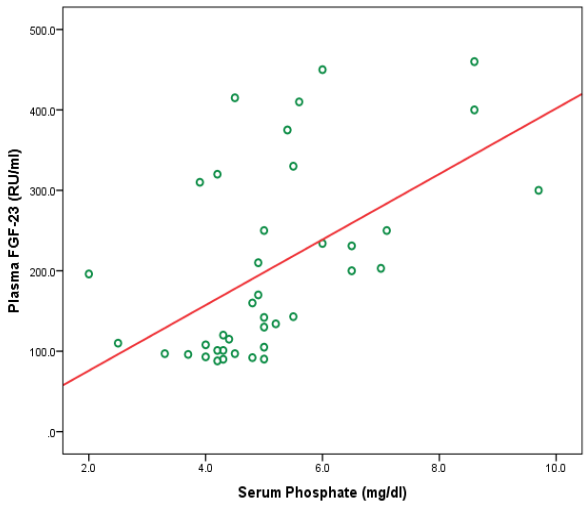


Figure 2: Scatter diagram showing correlation of plasma FGF-23 with serum phosphate (r=0.638, p<0.001); plasma FGF-23 demonstrated a strong and statistically significant positive correlation with serum phosphate (r = 0.638, p<0.001), showing that higher phosphate levels were associated with higher FGF-23 concentrations in children with predialysis CKD.

Plasma FGF23 concentrations increased progressively with worsening CKD stage and were consistently higher among children with CKD-MBD than those without MBD at every stage. In CKD stage 2 (eGFR 60-89 ml/min/1.73 m²), mean FGF23 was 88±2.09 RU/ml in the non-MBD group versus 102±4.05 RU/ml in the CKD-MBD group, showing a significant between-group difference (p=0.042). This separation widened in stage 3 (eGFR 30-59), where FGF23 rose to 99±9.09 RU/ml without MBD compared with 132±1.02 RU/ml with MBD (p=0.034). In stage 4 (eGFR 15-29), FGF23 increased further to 110±5.05 RU/ml in the non-MBD group but showed a marked elevation to 242±10.02 RU/ml in the CKD-MBD group (p=0.031). The highest levels were observed in stage 5 (eGFR <15), with mean FGF23 of 185±4.03 RU/ml without MBD versus 345±15.01 RU/ml with MBD, again statistically significant (p=0.011), indicating an accentuated stage-dependent divergence in FGF23 levels in the presence of mineral bone disorder.

DISCUSSION

The present study evaluated stage-specific variations in FGF23 and associated mineral bone disorder (MBD) biomarkers among children with predialysis CKD, revealing that plasma FGF23 concentrations increased progressively with CKD severity, with significantly higher levels in those with CKD-MBD compared to those without. This elevation began as early as stage 2, intensified in stages 3 and 4, and peaked in stage 5, highlighting FGF23 as a sensitive indicator of mineral dysregulation preceding overt biochemical abnormalities in phosphate, calcium, or PTH. The early and progressive rise observed in this cohort aligns with previous reports emphasizing FGF23 as one of the earliest endocrine responses to declining renal function, preceding hyperphosphatemia and secondary hyperparathyroidism.[4,19] The significant between-group differences across CKD stages further suggest that FGF23 elevation represents a pathophysiologic hallmark of CKD-MBD rather than an isolated laboratory finding.

The observed stage-dependent increase in FGF23 in this study is consistent with both pediatric and adult data showing similar trends. In large pediatric cohorts, such as the KNOW-PedCKD and 4C studies, FGF23, PTH, and phosphate levels have been shown to rise proportionally with CKD progression.[13,20] This pattern supports the hypothesis that phosphate retention acts as a principal stimulus for FGF23 secretion, with secondary activation of PTH contributing to the amplification of the mineral dysregulation loop.[21] The magnitude of the FGF23 increase in our predialysis COHORT, particularly in stage 4 and 5 CKD, closely mirrors findings from West African and European populations, where children with CKD-MBD exhibited significantly higher FGF23 and PTH levels than non-MBD counterparts.[11,22] These consistencies across geographically and ethnically

diverse cohorts reinforce the biological universality of FGF23 upregulation in CKD progression.

Our data also demonstrated strong positive correlations between FGF23, serum phosphate, and PTH, both of which remained statistically significant and directionally consistent across all CKD stages. This mirrors earlier studies showing linear relationships between FGF23 and both phosphate and PTH in predialysis CKD, with these markers collectively reflecting the degree of phosphate retention and skeletal remodeling.[23,24] The current findings extend this relationship to children, illustrating that even in the absence of dialysis, FGF23 functions as an integrated biochemical signal linking phosphate burden to hormonal adaptations. Furthermore, the increasing dispersion of FGF23 values observed at higher phosphate and PTH levels suggests an amplification of mineral dysregulation as CKD advances, a phenomenon similarly noted in both adult and pediatric populations with advanced disease.[25]

The early divergence in FGF23 levels between CKD-MBD and non-MBD groups observed in this cohort may be of particular clinical importance. Previous studies have confirmed that FGF23 rises prior to overt changes in serum phosphate or calcium, serving as an early biomarker for disordered mineral metabolism and future cardiovascular complications.[26,27] The clear statistical significance of stage-wise differences in FGF23 across all stages reinforces its diagnostic potential. Such early detection is critical in pediatric CKD, where interventions targeting phosphate control or vitamin D modulation can meaningfully alter long-term skeletal and cardiovascular outcomes.[11,20]

From a mechanistic perspective, the interplay between FGF23, PTH, and phosphate represents a tightly regulated yet pathologically self-reinforcing loop. The FGF23-Klotho-PTH endocrine axis mediates phosphate homeostasis and calcitriol suppression; however, in CKD, Klotho deficiency and phosphate retention perpetuate unregulated FGF23 secretion.[21,28] This feedback failure may explain the persistent and exaggerated FGF23 elevation observed in the present study, even in early CKD stages. Collectively, the current findings, in conjunction with previous evidence, position FGF23 as an early, sensitive, and clinically relevant biomarker of CKD-MBD in children, preceding classical biochemical abnormalities and mirroring disease severity. Further longitudinal and interventional research is warranted to define its predictive role in skeletal and cardiovascular outcomes and to establish stage-specific therapeutic targets for phosphate and hormonal regulation in pediatric CKD.

Limitations

This study was limited by its single-center, cross-sectional design and relatively small sample size, which may restrict generalizability and preclude causal inference. Additionally, the absence of longitudinal follow-up prevented evaluation of temporal changes in FGF23 and their predictive value for clinical outcomes.

CONCLUSION

This study demonstrates that fibroblast growth factor-23 (FGF23) rises early in children with predialysis chronic kidney disease and increases progressively with advancing CKD stage. FGF23 levels were significantly higher in children with CKD-mineral bone disorder compared with those without MBD across all stages, beginning as early as stage 2 and peaking in stage 5. The strong, statistically significant correlations between FGF23, serum phosphate, and parathyroid hormone highlight the central role of FGF23 within the mineral dysregulation cascade of CKD. These findings support FGF23 as a sensitive and stage-specific biomarker of CKD-MBD in pediatric patients, preceding overt abnormalities in conventional biochemical markers and reflecting disease severity during the predialysis period.

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