Pediatr Urol Case Rep 2022; 9(6):216-217 DOI: 10.14534/j-pucr.20222675593

PEDIATRIC UROLOGY CASE REPORTS
ISSN 2148-2969
http://www.pediatricurologycasereports.com

A potential link between pediatric hypercalciuria and FFF23

Ashenafi Kebede*

Department of Pediatrics, Jimma University, Jimma, Ethiopia

✉ ***Ashenafi Kebede***
Department of Pediatrics,
Jimma University,
Jimma, Ethiopia
E-mail: kebedehena@gmail.com

Received: *30-Nov-2022, Manuscript No. PUCR-22-83124;* ***Editor assigned:*** *2-Dec-2022, PreQC No. PUCR-22-83124 (PQ);* ***Reviewed:*** *15-Dec-2022, QC No. PUCR-22-83124;* ***Revised:*** *22-Dec-2022, Manuscript No. PUCR-22-83124 (R);* ***Published:*** *30-Dec-2022, DOI: 10.14534/j-pucr.20222675593*

Description

The most frequent metabolic disorder linked to paediatric urolithiasis is hypercalciuria, and some patients show a decrease in bone mass. Many different mechanisms, including decreased renal calcium reabsorption, increased gut calcium absorption, increased bone resorption, or any combination of the above, due to either genetic or environmental effects, have been proposed to explain the generalised dysregulation of calcium homeostasis in hypercalciuria. To maintain ideal calcium and phosphate homeostasis, FGF23, calcitriol, and PTH are all engaged in mineral metabolism in the gut, bone, and kidney. Therefore, these regulating hormones may contribute to the pathophysiology of hypercalciuria as well as urine calcium excretion. In the current investigation, we looked into FGF23's potential contribution to paediatric hypercalciuria.

Previous research, including ours, showed that both adult and paediatric hypercalciuric individuals had lower bone mineral density. The pathophysiologic mechanism behind the abnormal bone remodelling process in hypercalcemic patients is yet unknown, though. FGF23 is an osteocyte-derived hormone, hence increased levels in stone-forming individuals might be a sign of aberrant bone physiology. Recent research, however, found that the expression of FGF23 in osteocytes from individuals with hypercalciuria was not different from that of controls and did not depend on histomorphometric variables. On the other hand, these researchers discovered that there was no connection between serum PTH and bone resorption in these patients, which might be attributed to $1,25(OH)_2D$. Similar to how the TP/GFR and serum phosphate dropped in the presence of similar mean PTH concentrations, FGF23 was likely involved. Therefore, we suggest that other or additional mechanisms, such as those related to the environment and in particular the impact of nutrition, are at play in hypercalciuria. Recent research has indicated that hypertensive adults who follow the DASH diet had lower incidences of urolithiasis. Similar effects were seen in children; specifically, dietary sodium intake was reduced while potassium intake was increased, which lowered calciuria and stone formation. After receiving anticalciuric therapy, our patients' urine calcium excretion levels returned to those of healthy controls. As a result, the level of serum FGF23 dropped, and as was predicted, the levels of TP/GFR and serum phosphate rose. It appears that a chain of unidentified events that led to a decrease in the plasma FGF23 content occurred as calcium retention was improved. As was previously mentioned, the change might have been caused by a drop in serum $1,25(OH)_2D_3$ levels or activity on the bone. Our research has some drawbacks. Unfortunately, the serum $1,25(OH)_2D_3$ concentration was not tested due to financial restrictions. The missing piece in the puzzle for understanding the reported occurrences may have

been changes in its level before and after anticalcciuric therapy. The control group's access to blood and urine data may have revealed differences from the results seen in the hypercalcemic patients. Our study's limitations include the absence of information on $1,25(OH)_2D_3$ levels as well as serum PTH, creatinine, calcium, and phosphate values in controls. Nevertheless, we think that the aforementioned limitations do not affect the findings of this investigation, which suggest that FGF23 is unlikely to play a direct role in juvenile hypercalciuria and alterations in its metabolism following the introduction of pharmacologic anticalciuric treatments.

Conclusion

Plasma FGF23 levels between hypercalcemic and control children were not different, according to our study. Patients receiving pharmaceutical treatment exhibited significantly reduced plasma FGF23 levels, raised TP/GFR, and higher blood phosphate levels without appreciable changes in serum PTH values. They also had significantly decreased urine calcium excretion rates. Thus, it appears that the reversal of hypercalciuria may have an impact on phosphate metabolism either directly or indirectly. We advise future research to distinguish between treated and untreated patients and to add $1,25(OH)_2D_3$ among all other variables being assessed.