Original Article

ISSN (O): 2635-0823; ISSN (P): 2635-0815

Osteodystrophy in Children with Urolithiasis Caused by Primary Hyperparathyroidism

Abdusattor Akhatovich Nasirov[1], Fatkhillo Fayzievich Bayakhmedov[1], Timur Turaevich Norbayev[1], Mukhamadjon Abdurakhmonovich Norov[2], Sultanmurad Bakhtiyorovich Abdurakhmanov[2]

[1]Department of Faculty Pediatric Surgery, Tashkent Medical Pediatric Institute, Tashkent, Uzbekistan, [2]Tashkent Medical Pediatric Institute, Tashkent, Uzbekistan.

Abstract

Carried out the retrospective analysis 52 children, ill nefrolitiasis of etiology hyperparathyroidism. By purpose showed up to study degree demineralization bony cloth as to forms primary hyperparathyroidism at children. The studies of the activity of alkalized phosphatase and ehoosteometrii will lend possibility to appreciate degree demineralization bony cloth and accordingly diferation PGPT as to forms and the weights of disease. Hyperparathyroidism contributes development demineralization the bony cloth of various intensity which is connected by physical charge on bonus cloth. Indicators characterizing bony changes in stone disease of etiology hyperparathyroidism were more at children in miscellaneous form PGPT and accordingly high indicators are noted at children with the disorder of the function of buds and had direct relationship with quantity and size of formed stone and osteoporosis of bones.

Keywords: Urolithiasis, Primary Hyperparathyroidism, Kidneys, Alkaline Phosphatase, Pediatric Urology.

Corresponding Author: Abdusattor Akhatovich Nasirov, Department of Faculty Pediatric Surgery, Tashkent Medical Pediatric Institute, Tashkent, Uzbekistan.

Received: 19 July 2021 **Revised: 18 September 2021** **Accepted: 29 September 2021** **Published: 05 October 2021**

Introduction

In children suffering from urolithiasis, enamel destruction and tooth loss, pain and curvature of limbs and joints are noted. Attempts to reveal the cause of these phenomena by studying the mineral composition of blood and urine do not give positive results.[1–5] In recent years, the hyperparathyroid nature of these changes has been more often mentioned, especially when combined with bone demineralization and the formation of kidney stones.[6,7] Excessive production of parathyroid hormone enhances the activity of osteoclasts and osteoclastic resorption of bone tissue, which is accompanied by the mobilization of minerals from the bone tissue (calcium and inorganic phosphorus) and their excessive excretion by the kidneys.[3]

Purpose of the study Assessment of the degree of bone tissue demineralization depending on the form of primary hyperparathyroidism (PH) and urolithiasis.

Materials and Methods

The study involved 2100 children aged 1 to 15 years, patients with urolithiasis, of which PGPT was diagnosed in 52 (2.5%) children. There were 29 (55.7%) patients with renal form (PF) PHPT, and 23 (44.2%) patients with mixed form (kidney and bone damage) (SF). PGPT was diagnosed on the basis of studying the content of total calcium and inorganic phosphorus in the blood and in daily urine, ionized calcium in the blood, stress tests with calcium and sodium chloride, the content of parathyroid hormone (PTH), calcitonin (CT), vitamin D and cyclic 3,5-adenosine monophosphate (cAMP) in serum. To detect damage to bone tissue, we studied the activity of alkaline phosphatase (ALP) and X-ray densitometry of the bones of the hand and ultrasound osteometry of tubular bones.[8,9] To clarify the severity of canine formation and assess the functional state of the kidneys, ultrasound, radiological (survey, excretory or infusion urography), radioisotope (radioisotope renography, renal scanning, dynamic nephroscintigraphy) studies were used.[10–13]

The ALP activity was determined by the photocalorimetric method using reagents from Lachema. Ultrasound examination of the bone tissue was investigated with the device ”Echoosteometer EOM - 02 Ts”, in four bones - tibial, radial, clavicle and lower jaw.[14,15]

Results and Discussion

Clinical symptoms characteristic of bone changes were: pain in the limbs in 30 (57.65%) children, change in gait - in 19 (36.5%) children, deformity and curvature of the bones of the skull, spine and limbs - in 21 (40.3%) of a child, fractures of tubular bones were observed in 7 (13.4%) children. In 50 (96.1%) children, the disease was complicated by pyelonephritis, in 24 (46.1%) children, gross renal dysfunction was noted. Patients with HGPT significantly lagged behind in physical development.

The alkaline phosphatase activity in patients with urolithiasis without PGPT (comparison group) did not significantly differ from that of healthy children [Table 1].

The alkaline phosphatase activity in 46 (88.4%) children with PGPT was higher than that in the control group. With PPHPT, the ALP activity significantly increased (1.23? 0.06 μmol / liter) p? 0.05. If renal failure was added to it, then this indicator increased 2.17 times (1.87 ? 0.08 μmol / liter) compared with children in the control group (p?0.01).

With SF PHPT, the ALP activity was 2.8 times higher, p ? 0.001. If renal failure was associated, then this indicator increased significantly and was equal to 6.38 ? 0.22 μmol / liter, which is 7.8 times higher than the indicator of children in the control group (p ? 0.001).

To identify the degree of impairment of the mineral saturation of bone tissue during PGPT in children with urolithiasis, an ultrasound examination of bones was performed [Table 2]. The difference in echoosteometry indices in children with ICD without PHPT with healthy children was significant only in the tibia, in the rest of the bones there was a tendency to decrease.

In children with the renal form of PGPT, the echo osteometry indices in all bones were significantly lower (p ? 0.05). Bone tissue in the mixed form of PHPT was distinguished by a significantly low (1.8 times) conductivity of ultrasound signals (P?0.001) compared with children, the renal form of PHPT. Echoosteometry indices in children with renal form of PHPT complicated by renal failure were 1.41 times lower (P?0.05), in children with a mixed form - 2.1 times lower (P?0.001).

Analysis of the research results showed that the activity of alkaline phosphatase and echoosteometry in children with PHPT significantly differs from the indicators of children in the control group ($p < 0.05$), which is a characteristic sign of evaluating the process of bone tissue demineralization. A significantly high level of alkaline phosphatase and a low level of echoosteometry was observed in children with a mixed form of PHPT, especially in children with impaired renal function ($p < 0.001$).

Conclusion

Thus, the study of the activity of alkaline phosphatase and echo-osteometry of bones makes it possible to assess the severity of osteystrophy and, accordingly, to differentiate PHPT by forms (renal and mixed) and severity of the disease. High indices of alkaline phosphatase and low indices of echoosteometry were observed in children with impaired renal function and had a direct relationship with the number and size of formed calculi and bone osteoporosis.

References

1. Colak H, Dülgergil CT, Dalli M, Hamidi MM. Early childhood caries update: A review of causes, diagnoses, and treatments. J Nat Sci Biol Med. 2013;4(1):29–38. Available from: https://dx.doi.org/10.4103/0976-9668.107257.
2. Marcus R. Laboratory diagnosis of primary hyperparathyroidism. Endocrinol Metab Clin North Am. 1989;18(3):2550219.
3. Suzuki S, Fukushima T, Ami H, Asahi S, Onogi H, Nakamura I, et al. Pre- and postoperative bone metabolism of primary hyperparathyroidism. Biomed Pharmacother. 2000;54(1):90–96. Available from: https://doi.org/10.1016/s0753-3322(00)80021-9.
4. Bandeira F, Cusano NE, Silva BC. Bone disease in primary hyperparathyroidism. Arq Bras Endocrinol Metabol. 2014;58(5):553–561.
5. Douglass JM, Douglass AB, Silk HJ. A practical guide to infant oral health. Am Fam Physician. 2004;70(11):2113–2120.
6. Krajisnik T, Björklund P, Marsell R, Ljunggren O, Akerström G, Jonsson KB, et al. Fibroblast growth factor-23 regulates parathyroid hormone and 1alpha-hydroxylase expression in cultured bovine parathyroid cells. J Endocrinol. 2007;195(1):125–156. Available from: https://doi.org/10.1677/joe-07-0267.
7. Moosgaard B, Christensen SE, Vestergaard P, Heickendorff L, Christiansen P, Mosekilde L. Vitamin D metabolites and skeletal consequences in primary hyperparathyroidism. Clin Endocrinol (Oxf). 2007;68(5):707–715. Available from: https://doi.org/10.1111/j.1365-2265.2007.03109.x.
8. Rossini M, Gatti D, Isaia G, Sartori L, Braga V, Adami S. Effects of oral alendronate in elderly patients with osteoporosis and mild primary hyperparathyroidism. J Bone Miner Res. 2001;16(1):113–122. Available from: https://doi.org/10.1359/jbmr.2001.16.1.113.
9. Kollars J, Zarroug AE, Heerden JV, Lteif A, Stavlo P, Suarez L, et al. Primary hyperparathyroidism in pediatric patients. Pediatrics. 2005;115(4):974–80. Available from: https://doi.org/10.1542/peds.2004-0804.
10. Nosirov AA, Sobitov IZ. Osteodystrophy in Children with the Renal form of Primary Hyperparathyroidism. Asian J Res Rep Urol. 2019;2(1):1–5.
11. Abdusattor AN, Temur TN, Fatkhulla FB. Frequency and Risk Factors of Stone Formation in Kidneys of Children Experience of the Urology Department of Clinic of Tashpmi. JAMDSR. 1995;9(4):71–76.

Table 1: Alkaline phosphatase activity in children with ice (μmol / liter)

Study groups children	n	Alkaline phosphatase activity (μmol / liter)	P
Healthy children.	20	0,82 ±0,04	p>0,05
Children with urolithiasis without PGPT (control).	20	0,91±0,06	p<0,05 ;P1<0,05
Children with the renal form of PGPT.	16	1,23 ±0,06	p<0,01;p1<0,01
Mixed form of PGPT.	12	2,3 ±0,1	p<0,01;p1<0,01
Renal form of PGPT complicated by renal failure.	13	1,87 ±0,08	p<0,001
Mixed form of PGPT complicated by renal failure.	11	6,38 ±0,22	p1<0,001

R - Compared with the indicators of healthy children.
P1- Compared with the indicators of children with urolithiasis (control).

Table 2: Echoosteometry indices in the examined children

	Study groups of children	n	Echoosteometry (meter / seconds)			
			Tibia	Elbow bone	Collarbone	Lower jaw
1	Healthy children	20	3898±43	3786±57	3591±63	3642±61
2	Children with urolithiasis disease without HGPT (control)	20 p	3609±37 p < 0,05	3447±37 p < 0,05	3492±62	3501±57
3	Renal form PGPT	16 p P 1	3210±43 p < 0,05 1 < 0,05	3228±41 p < 0,05 1 < 0,05	3270±38 p < 0,05 1 < 0,05	3322±49 p < 0,05 1 < 0,05
4	Mixed form PGPT	12 p P1	2208±41 p < 0,01 1 < 0,01	2354±46 p < 0,01 1 < 0,01	2482±53 p < 0,01 1 < 0,01	2456±51 p < 0,01 1 < 0,01
5	Renal form PGPT complicated by renal failure	13 p P1	2786±39 p < 0,05 1 < 0,05	3049±42 p < 0,05 1 < 0,05	3152±49 p < 0,05 1 < 0,05	3040±61 p < 0,05 1< 0,05
6	Mixed PHPT complicated by renal failure	11 p P1	1888±42 p < 0,001 1 < 0,001	1960±54 p < 0,001 1 < 0,001	2080±61 p < 0,001 1 < 0,001	2070±59 p < 0,001 1 < 0,001

R - Comparedwith the indicators of healthy children.
P1- Comparedwith the indicators of children with urolithiasis (control).

12. Elkoushy MA, Yu AX, Tabah R, Payne RJ, Dragomir A, Andonian S. Determinants of urolithiasis before and after parathyroidectomy in patients with primary hyperparathyroidism. Urology. 2014;84(1):22–26. Available from: https://doi.org/10.1016/j.urology.2014.01.016.
13. Gentle DL, Stoller ML, Bruce JE, Leslie SW. Geriatric urolithiasis. J Urol. 1997;158(6):2221–2225. Available from: https://doi.org/10.1016/s0022-5347(01)68203-x.
14. Sellaturay S, Fry C. The metabolic basis for urolithiasis. Surgery. 2008;26(4):136–140. Available from: https://doi.org/10.1016/j.mpsur.2008.03.002.
15. Rowlands C, Zyada A, Zouwail S, Joshi H, Stechman MJ, Scott-Coombes DM. Recurrent urolithiasis following parathyroidectomy for primary hyperparathyroidism. Ann R Coll Surg Engl. 2013;95(7):523–528. Available from: https://doi.org/10.1016/j.mpsur.2008.03.002.

How to cite this article: Nasirov AA, Bayakhmedov FF, Norbayev TT, Norov MA, Abdurakhmanov SB. Osteodystrophy in Children with Urolithiasis Caused by Primary Hyperparathyroidism. Adv Clin Med Res. 2021;2(4):8-10.

Source of Support: Nil, **Conflict of Interest:** None declared.