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Analysis of salt transport indices, calcification markers, and FGF23 in patients with nephrolithiasis and crystalluria

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Abstract. ***Background.*** *Nephrolithiasis (NL) is a common polyetiological urological disease that is frequently associated with reduced bone mineral density. One of the shared key factors contributing to both osteoporosis and NL is insufficient intake of calcium-rich products. Another common pathogenic mechanism involves the activation of calcification factors, such as osteopontin (OPN), osteocalcin (OC), and fibroblast growth factor 23 (FGF23). The purpose was to evaluate indices of salt transport, calcification markers (OC, OPN), and FGF23 in patients with nephrolithiasis and crystalluria.* ***Materials and methods.*** *The study was conducted at the Department of Urology of the Bogomolets National Medical University, and the Urology Department of the Kyiv Regional Clinical Hospital. The work was carried out in accordance with the research plan and is a fragment of the research project "Optimization of the management of patients with urolithiasis with concomitant osteoporosis" (state registration number 0125U000958). The diagnosis of NL was established based on the criteria according to the 2023 guidelines of the European Association of Urology. The assessment of salt transport, osteopontin, and FGF23 was carried out in a certified laboratory (Medical Laboratory "DILA", Kyiv, Ukraine). Osteocalcin was measured in the Ukrainian Osteoporosis Center and Department of Clinical Physiology and Pathology of Locomotion Apparatus of the State Institution "D.F. Chebotarev Institute of Gerontology of the National Academy of Medical Sciences of Ukraine" (Kyiv, Ukraine).* ***Results.*** *Higher rates of oxaluria were observed in patients with NL and crystalluria, which was predominant in the study groups compared to other indicators studied. The levels of calcification markers (OPN, OC) and FGF23 were significantly lower in patients from group I compared to those with NL and crystalluria.* ***Conclusions.*** *The level of FGF23 showed a positive association with serum phosphate levels, which is consistent with its physiological mechanism of action. Further study is needed to determine the specific changes in calcification markers (OPN, OC) and FGF23 depending on changes in bone mineral density.*

Keywords: *urolithiasis; vascular calcification; osteopontin; osteocalcin; FGF23*

Introduction

Nephrolithiasis (NL) is a common polyetiological urological disorder that is often associated with reduced bone mineral density (BMD). In a large cohort study including 531,431 patients with nephrolithiasis, 23.6 % were found to have a diagnosis of osteoporosis or fractures [1]. Moreover, cross-sectional data from NHANES (13,357 participants) demonstrated that reduced BMD (osteopenia and osteoporosis) was statistically associated with a higher risk of kidney stone formation (OR 1.24 and 1.41, respectively) [2].

One of the key shared risk factors for both osteoporosis and NL is insufficient dietary intake of calcium-rich foods. When calcium intake is low, the amount of free oxalates in the intestine increases, enhancing their absorption and urinary excretion, which in turn raises the risk of oxalate stone formation [3].

Another common pathogenic mechanism is the activation of calcification factors such as osteopontin (OPN) and osteocalcin (OC). Elevated OC levels are associated with an increased risk of coronary atherosclerosis and myocardial

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infarction, partly through vascular calcification as an intermediate mechanism [Mendelian randomization analysis: OR ≈ 1.07–1.29] [4]. Elevated OPN levels have also been linked to cardiovascular complications, particularly in type 2 diabetes, where higher concentrations were associated with a 32 % increased risk of CVD and a 25 % increased risk of diabetic retinopathy per 1 SD increase [2]. However, data remain limited on the role of these calcification markers in patients with nephrolithiasis and crystalluria.

Vascular calcification is also a key comorbid factor. One previous study demonstrated a significant association between abdominal aortic calcification (AAC) and the formation of calcium oxalate stones (OR ≈ 5.76) [5]. Additionally, other studies have shown correlations between fetuin-A levels, cortical bone porosity, and vascular calcification in patients with NL, highlighting the interconnections between bone structure, vascular pathology, and urological risk [8].

In the rapidly developing field of biogerontology, the nephron — the fundamental structural and functional unit of the kidney — has emerged as a key regulator of systemic aging. This paradigm shift has been largely driven by the discovery of the Klotho protein, known for its anti-aging properties, and its regulatory partner fibroblast growth factor 23 (FGF23). Together, these two molecules orchestrate essential biological processes, including mineral metabolism, vascular stability, and cellular homeostasis. Their coordinated activity is now recognized as critical not only for maintaining physiological organ function but also for promoting healthy longevity and resilience to age-related decline [7].

Within the context of osteo-vascular interactions, the FGF23-Klotho axis has been discussed as an important regulator of skeletal-vascular homeostasis and a potential therapeutic target [8]. Furthermore, recent literature suggests that elevated FGF23 may serve as the most sensitive marker of nephron injury. A meta-analysis of 11 studies involving 1,946 patients with acute kidney injury (AKI) evaluated the diagnostic accuracy of plasma FGF23 levels for detecting AKI. The findings showed a sensitivity of 82 % (95% CI: 66–91), specificity of 77 % (95% CI: 67–85), and an AUC of 0.86 (95% CI: 0.82–0.88). Thus, elevated FGF23 appears to be a sensitive biomarker of nephron injury, capable of detecting early stages of AKI, often preceding traditional markers such as cystatin C and creatinine [9].

Taken together, current evidence highlights a deep pathogenetic interplay between nephrolithiasis, impaired bone density, vascular calcification, and regulatory biomarkers (osteocalcin, osteopontin, FGF23/Klotho). This underlines the need for further integrated research to develop effective diagnostic, preventive, and therapeutic approaches for these comorbid conditions.

The purpose. To assess indices of salt transport, calcification markers (osteocalcin, osteopontin), and FGF23 in patients with nephrolithiasis and crystalluria.

Materials and methods

The study was conducted at the Department of Urology, Bogomolets National Medical University, and the Urology Department of Kyiv Regional Clinical Hospital. The work was carried out in accordance with the research plan and is a fragment of the research project Department of Urology "Optimization of the management of patients with urolithiasis with concomitant osteoporosis" (state registration number 0125U000958). The diagnosis of NL was established based on the criteria according to the recommendations of the European Association of Urology in 2023 [10].

The assessment of salt transport, osteopontin, and FGF23 was carried out in a certified laboratory (Medical Laboratory "DILA", Kyiv, Ukraine).

Osteocalcin measurement was carried out on the Ukrainian Osteoporosis Center and Department of Clinical Physiology and Pathology of Locomotion Apparatus of the State Institution "D.F. Chebotarev Institute of Gerontology of the National Academy of Medical Sciences of Ukraine", Kyiv, Ukraine (Chief of the Department Prof. N.V. Grygorieva).

Patients with chronic kidney disease (CKD) and estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73 m^2, calculated by the CKD-EPI formula according to KDIGO 2017 guidelines [11], were excluded from the study.

A total of 104 patients were enrolled: 21 men (20.19 %) and 83 women (79.81 %), with a mean age of 57 years [23–83]. The mean duration of menopause among women was 8 years [0–20]. Patients were divided into three groups:

— **Group I:** 41 participants without urolithiasis or crystalluria, who did not meet exclusion criteria (28 women [68.3 %], 13 men [31.7 %]); mean age 58 years [50–65]; mean duration of menopause in women 8 years [0–18].

— **Group II:** 39 participants with urolithiasis (22 women [56.4 %], 17 men [43.6 %]); mean age 55 years [52–61.5]; mean menopause duration 1 year [0–13.5].

— **Group III:** 24 participants with crystalluria (20 women [83.3 %], 4 men [16.7 %]); mean age 57.5 years [52.5–63.75]; mean menopause duration 11.5 years [2.75–26.5].

Statistical analysis. Data processing was carried out using MS Excel and Statistica EZR version 1.62-2023 statistical programs. The Shapiro-Wilk W test was used to test the distribution for normality. The frequency of quantitative indices was indicated in absolute (n) and relative (%) frequencies. The quantitative indices are presented in the form of median (Me) for variables with a distribution that was different from the normal one and the interquartile range [IQR] of QI ÷ QIII indices. The Mann-Whitney U test was used to assess differences between groups. The difference between the groups was considered statistically significant at $p < 0.05$.

Results

Comparative analysis revealed no significant differences in age, BMI, or duration of menopause among women, serum calcium, phosphorus, creatinine, eGFR, urinary pH, and urinary excretion of calcium and phosphorus among the groups ($p > 0.05$).

Urinary oxalate excretion was significantly higher in groups II and III compared to group I ($p = 0.001$ and $p < 0.001$, respectively), with no significant difference between groups II and III. Urinary uric acid levels were significantly higher in group III compared to group I ($p = 0.002$), although all values remained within the reference range (Table 1).

FGF23 levels were also significantly elevated in groups II and III compared to group I ($p < 0.001$ for both), without significant differences between patients with urolithiasis and those with crystalluria (Fig. 1). OC levels were significantly elevated in groups II and III compared to group I ($p = 0.003$ and $p = 0.002$, respectively) (Fig. 2). Similarly, OPN levels were significantly higher in groups II and III compared to group I ($p < 0.001$ for both) (Fig. 3).

Correlation analysis revealed a strong association serum calcium and urinary phosphorus ($r = 0.59$, $t = 7.43$, $p = 3.288e\text{-}11$); and serum calcium and urinary uric acid ($r = 0.49$, $t = 5.7$, $p < 0.001$). FGF23 was correlated with serum phosphorus levels ($r = 0.42$, $t = 3.14$, $p = 0.002$). eGFR was negatively correlated with the duration of menopause ($r = –0.39$, $t = –4.32$, $p < 0.0001$).

Discussion

Literature evidence supports that elevated FGF23 is an early and sensitive biomarker of nephron tubular injury, outperforming serum creatinine and cystatin C in detecting early kidney damage [12]. FGF23 is a peptide hormone primarily secreted by osteocytes and osteoblasts, as well as renal tubular cells. Its main target is the kidney, where it maintains phosphate homeostasis by promoting urinary phosphate excretion through inhibition of proximal tubular phosphate reabsorption. In addition, FGF23 suppresses renal synthesis of 1,25-dihydroxyvitamin D_3, thereby further modulating mineral metabolism.

Unlike other members of the fibroblast growth factor family, FGF23 requires the obligate co-receptor Klotho for binding and activation of specific FGF receptors (FGFRs). This dependence highlights the unique selectivity and tightly regulated nature of the FGF23-Klotho signaling axis, which plays a pivotal role in systemic phosphate and vitamin D homeostasis, vascular integrity, and pathways related to ageing [13].

In chronic kidney disease (CKD) and ageing, renal Klotho expression declines, accompanied by reduced active vitamin D synthesis and elevated FGF23 levels. This dysregulation is further aggravated by reduced activity of renal 1α-hydroxylase, impairing vitamin D activation and disrupting the normal feedback regulation of parathyroid hormone (PTH). The resulting maladaptive hormonal loop — characterized by Klotho deficiency, excess FGF23, vitamin D deficiency, and secondary hyperparathyroidism — contributes

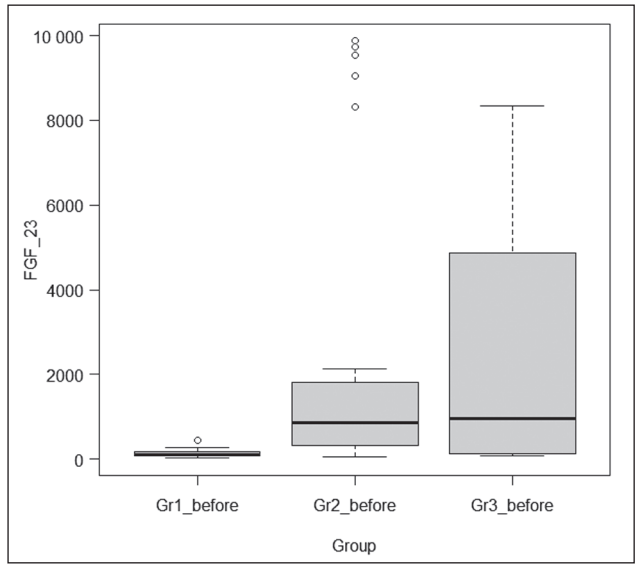

Figure 1. Comparison of FGF23 in patients with and without NL

Notes: interval estimation of the average values of FGF23 (the average value, the error of the average and the 95% probable interval of the average are indicated); * — significant difference between the indicators, p < 0.05.

Table 1. Comparative evaluation of salt transport parameters in patients depending on the presence of urolithiasis

Parameters	Group I (n = 41)	Group II (n = 39)	Group III (n = 24)	p-value
	Me [QI÷QIII], M ± m			
BMI, kg/m²	23.8 [22–25.9]	25.9 [24.5–27.02]	24.77 [22.85–26]	> 0.05
Serum uric acid, μmol/L	256 [220–310]	308 [257–351]	269 [233–321.7]	> 0.05
Serum calcium, mmol/L	1.24 [1.22–1.28]	1.23 [1.2–1.28]	1.26 [1.22–1.29]	> 0.05
Serum phosphorus, mmol/L	1.23 ± 0.11	1.24 ± 0.24	1.53 ± 1.72	> 0.05
Urinary oxalates, mg/day	16.2 [9.67–23.34]	**24.01 [13.9–36.34]***	**28.67 [20.66–38.66]***	
Urinary pH	6 [6–6.3]	6 [6–6.5]	6 [6–6.2]	> 0.05
Urinary calcium, mmol/day	4.1 [2.9–5.6]	4.5 [2.65–6.35]	4.95 [4.4–5.6]	> 0.05
Urinary phosphorus, mmol/day	21.46 ± 7.89	26.34 ± 10.06	25.88 ± 8.79	> 0.05
Urinary uric acid, μmol/day	2672 [1958–3756]	3490 [2625–4127]	**4185 [3519–4368]***	> 0.05
Serum creatinine, μmol/L	69.5 [56–87]	77 [59.5–86.99]	67.5 [55–85.49]	> 0.05
eGFR, ml/min/1.73 m²	84.27 ± 27.63	89.33 ± 17.41	85.20 ± 26.64	> 0.05
Osteocalcin, ng/ml	20 [12.5–25.3]	**26.7 [19.7–28.4]***	**28.3 [23.1–30.34]***	> 0.05
Osteopontin, ng/ml	9.7 [8.2–11.9]	**13.09 [12.4–18.2]***	**17.3 [15–19.4]***	> 0.05
FGF23, pg/ml	97.4 [66.96–177.59]	**840.26 [312.05–1805.33]***	**969.16 [283.4–931.79]***	> 0.05

Notes: eGFR — estimated glomerular filtration rate; * — differed significantly compared with group I (p < 0.05).

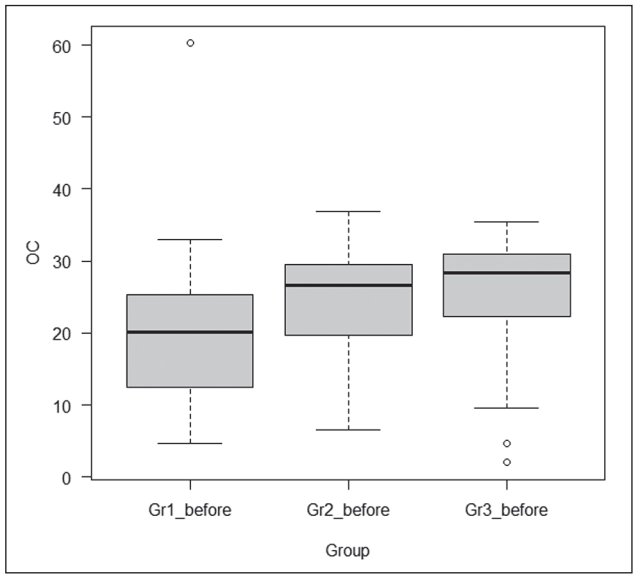

Figure 2. Comparison of OC in patients with and without NL

Notes: interval estimation of the average values of OC (the average value, the error of the average and the 95% probable interval of the average are indicated); * — significant difference between the indicators, p < 0.05.

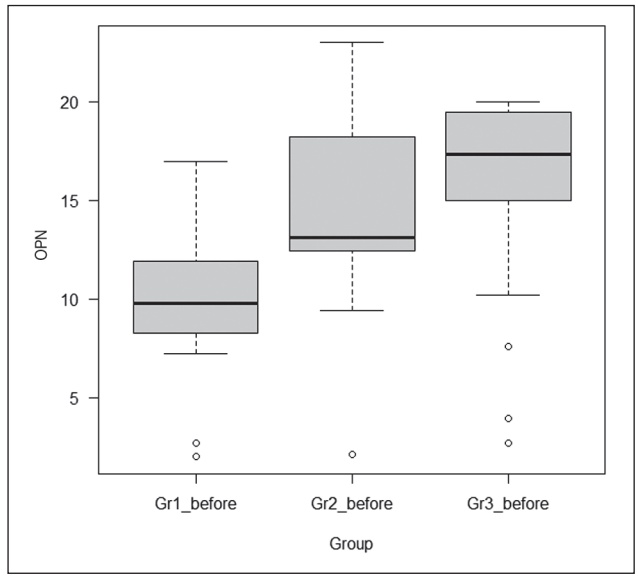

Figure 3. Comparison of OPN in patients with and without NL

Notes: interval estimation of the average values of OPN (the average value, the error of the average and the 95% probable interval of the average are indicated); * — significant difference between the indicators, p < 0.05.

significantly to vascular calcification and arterial stiffness, thereby increasing cardiovascular risk in CKD patients [8].

Since FGF23 promotes phosphate excretion in proximal tubules, it may increase calcium-phosphate precipitation within the tubular fluid. High-phosphate diets can exacerbate this process by raising solubility thresholds. Hyperphosphatemia is a well-recognized risk factor for vascular calcification and cardiovascular events; thus, dietary phosphate restriction and phosphate binders are recommended in CKD patients with hyperphosphatemia [14]. Importantly, hyperphosphatemia itself can directly induce tubular and interstitial injury. As a phosphaturic hormone, FGF23 serves as a surrogate marker of phosphate load on nephrons. Its elevation should therefore be recognized as a signal of excessive phosphate intake relative to nephron mass, justifying early phosphate restriction even in the absence of overt hyperphosphatemia. Indeed, some authors propose initiating phosphate binder therapy at CKD stage 2–3 when serum FGF23 levels rise, despite normal serum phosphate concentrations [15].

Taken together, our findings suggest that elevations in calcification markers — particularly FGF23 — may reflect tubular injury and contribute to crystalluria, representing an additional risk factor for urolithiasis and BMD violation.

Conclusions

Higher rates of oxaluria were observed in patients with Kidney Stone Diseases and crystalluria, which was predominant in the study groups compared to other indicators studied. The levels of calcification markers (OPN, OC) and FGF23 were significantly lower in patients from group I compared with those with NL and crystalluria. The level of FGF23 showed a positive association with serum phosphate levels, which is consistent with its physiological mechanism of action. Further study is needed to determine the specific changes in calcification markers (OPN, OC) and FGF23 depending on changes in BMD.

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References

1. Ganesan C, Thomas IC, Romero R, et al. Osteoporosis, Fractures, and Bone Mineral Density Screening in Veterans With Kidney Stone Disease. J Bone Miner Res. 2021 May;36(5):872-878. doi: 10.1002/jbmr.4260.

2. Luo D, Xie L, Zhang J, Liu C. Exploring the association between osteoporosis and kidney stones: a clinical to mechanistic translational study based on big data and bioinformatics. Biol Direct. 2025 Mar 31;20(1):42. doi: 10.1186/s13062-025-00627-w.

3. Rendina D, De Filippo G, Iannuzzo G, Abate V, Strazzullo P, Falchetti A. Idiopathic Osteoporosis and Nephrolithiasis: Two Sides of the Same Coin? Int J Mol Sci. 2020 Oct 31;21(21):8183. doi: 10.3390/ijms21218183.

4. Guo X, Hou C, Liu F, et al. Genetic insights into circulating osteocalcin for cardiovascular diseases and the role of vascular calcification. Nutr Metab Cardiovasc Dis. 2025 Jul;35(7):103870. doi: 10.1016/j.numecd.2025.103870.

5. Li B, Tang Y, Zhou L, et al. Association between aortic calcification and the presence of kidney stones: calcium oxalate calculi in

focus. Int Urol Nephrol. 2022 Aug;54(8):1915-1923. doi: 10.1007/s11255-021-03058-4.

6. Rodrigues FG, Neves RFCA, Ormanji MS, et al. Vascular Calcification Is Associated with Fetuin-A and Cortical Bone Porosity in Stone Formers. J Pers Med. 2022 Jul 10;12(7):1120. doi: 10.3390/jpm12071120.

7. Salloum JS, Garsetti DE, Rogers MB. Genetic background influences the impact of KLOTHO deficiency. Physiol Genomics. 2020 Oct 1;52(10):512-516. doi: 10.1152/physiolgenomics.00094.2020.

8. Wei X, Huang X, Liu N, Qi B, Fang S, Zhang Y. Understanding the Stony Bridge between Osteoporosis and Vascular Calcification: Impact of the FGF23/Klotho axis. Oxid Med Cell Longev. 2021 Aug 30;2021:7536614. doi: 10.1155/2021/7536614.

9. Sun S, Liu Z, Chen C, et al. Serum fibroblast growth factor 23 for early detection of acute kidney injury in critical illness. Am J Transl Res. 2021 Nov 15;13(11):12141-12151.

10. Geraghty RM, Davis NF, Tzelves L, et al. Best Practice in Interventional Management of Urolithiasis: An Update from the European Association of Urology Guidelines Panel for Urolithiasis 2022. Eur Urol Focus. 2023 Jan;9(1):199-208. doi: 10.1016/j.euf.2022.06.014.

11. Isakova T, Nickolas TL, Denburg M, et al. KDOQI US Commentary on the 2017 KDIGO Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD). Am J Kidney Dis. 2017 Dec;70(6):737-751. doi: 10.1053/j.ajkd.2017.07.019.

12. Patel ML, Gupta S, Shyam R, Sachan R, Ali W. Serum Fibroblast Growth Factor 23 Levels as a Predictor of Acute Kidney Injury in Critically Ill Obstetric Women in Obstetric Intensive Care Units: A North Indian Study. Adv Biomed Res. 2025 Jul 21;14:66. doi: 10.4103/abr.abr_146_23.

13. Hu MC, Shiizaki K, Kuro-o M, Moe OW. Fibroblast growth factor 23 and Klotho: physiology and pathophysiology of an endocrine network of mineral metabolism. Annu Rev Physiol. 2013;75:503-33. doi: 10.1146/annurev-physiol-030212-183727.

14. Kestenbaum B, Sampson JN, Rudser KD, et al. Serum phosphate levels and mortality risk among people with chronic kidney disease. J Am Soc Nephrol. 2005 Feb;16(2):520-8. doi: 10.1681/ASN.2004070602.

15. Kuro-O M. Molecular Mechanisms Underlying Accelerated Aging by Defects in the FGF23-Klotho System. Int J Nephrol. 2018 May 21;2018:9679841. doi: 10.1155/2018/9679841.

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Authors' contributions. O. Nikitin — concept and design of study, collection and processing of the material; I. Kordubailo, O. Nishkumay — collection and processing of the material, analysis of the data, text writing; Mike K.S. Chan, D. Klokol — analysis of the data, text writing.

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Аналіз показників транспорту солей, маркерів кальцифікації та FGF23 у пацієнтів із нефролітіазом та кристалурією

Резюме. Актуальність. Сечокам'яна хвороба (СКХ) — це поширений поліетіологічний урологічний стан, що часто поєднується з порушеннями мінеральної щільності кісткової тканини. Одним із спільних ключових факторів остеопорозу і СКХ є недостатнє вживання кальційвмісних продуктів. Також спільними механізмами розвитку є активація факторів кальцифікації (остеопонтин (OPN), остеокальцин (ОС)) та фактора росту фібробластів 23 (FGF23). **Мета:** провести оцінку показників транспорту солей, маркерів кальцифікації (ОС, OPN) та FGF23 у пацієнтів із сечокам'яною хворобою та кристалурією. **Матеріали та методи.** Дослідження проводилося на базі кафедри урології Національного медичного університету імені О.О. Богомольця, відділення урології КНП КОР «Київська обласна клінічна лікарня» відповідно до плану науково-дослідних робіт і є фрагментом НДР «Оптимізація ведення хворих на уролітіаз із супутнім остеопорозом» (номер державної реєстрації 0125U000958). Аналіз транспорту солей, остеопонтину та FGF23 виконували у сертифікованій лабораторії ТОВ «МЛ «ДІЛА». Рівень остеокальцину визначали на базі Українського центру остеопорозу та відділу клінічної фізіології та патології опорно-рухового апарату Державної установи «Інститут геронтології імені Д.Ф. Чеботарьова Національної академії медичних наук України» (Київ, Україна). **Результати.** Вірогідно вищі показники оксалатурії спостерігалися в пацієнтів із СКХ та кристалурією, яка переважала в досліджуваних групах порівняно з іншими показниками, що вивчалися. Рівні маркерів кальцифікації (OPN, ОС) та FGF23 були значно нижчими в пацієнтів групи I порівняно з тими, хто мав СКХ та кристалурію. **Висновки.** Уміст FGF23 продемонстрував позитивну асоціацію з рівнем фосфатів у сироватці крові, що узгоджується з його фізіологічним механізмом дії. Потрібне подальше вивчення особливостей динаміки маркерів кальцифікації (OPN, ОС) та FGF23 залежно від змін мінеральної щільності кісткової тканини.

Ключові слова: сечокам'яна хвороба; кальцифікація судин; остеопонтин; остеокальцин; FGF23