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Clinical Features of Urolithiasis in Patients With Comorbid Conditions

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ABSTRACT

Urolithiasis is a common disorder of the urinary system. The disease often becomes recurrent, characterized by rapid calculus growth, a trend toward staghorn and multiple stone formation, emergency complications, and disability – particularly in patients with metabolic disorders.

This article presents a clinical case of a patient with a long history of recurrent urolithiasis that developed against the background of comorbid pathology associated with metabolic syndrome and complicated by chronic kidney disease. The case analysis highlights the crucial role of metabolic disorders in the progression of nephrolithiasis and renal dysfunction. The paper describes specific features of surgical treatment in comorbid patients, emphasizing adherence to the "golden hour" principle and risk minimization. It underscores the necessity of a multidisciplinary approach, early metabolic correction, and systemic metaphylactic measures to prevent stone recurrence and reduce the risk of chronic kidney disease development in this patient category.

Keywords: urolithiasis, metabolic syndrome, comorbidity, chronic kidney disease

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Особенности течения уролитиаза на фоне коморбидной патологии

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РЕЗЮМЕ

Мочекаменная болезнь является распространенным заболеванием мочевыделительной системы. Нередко заболевание приобретает рецидивирующий характер с быстрым ростом конкрементов, склонностью к образованию коралловидных и множественных камней, возникновением неотложных состояний и инвалидизации особенно у пациентов на фоне метаболических нарушений.

Представлен клинический случай пациента с длительным анамнезом рецидивирующей мочекаменной болезни, развившейся на фоне коморбидной патологии, ассоциированной с метаболическим синдромом, осложненными хронической болезнью почек. Анализ случая подчеркивает ключевую роль метаболических нарушений в прогрессировании нефролитиаза и почечной дисфункции. Описаны особенности хирургиче-

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ского лечения у коморбидного пациента с акцентом на соблюдение принципа «золотого часа» и минимизации рисков. Подчеркивается необходимость междисциплинарного подхода, ранней метаболической коррекции и системной метафилактики для предотвращения рецидивов камнеобразования и снижения риска развития хронической болезни почек у данной категории пациентов.

Ключевые слова: мочекаменная болезнь, метаболический синдром, коморбидная патология, хроническая болезнь почек

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии финансирования при проведении исследования.

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INTRODUCTION

Urolithiasis (UL) remains one of the most prevalent diseases of the urinary system, affecting approximately 10% of the global population [1]. In the Russian Federation, the incidence of UL reaches 5–7%, with an annual increase of 1–2%. The high prevalence of the disease among the working-age population [2] positions it not only as a medical but also as a socio-economic problem. The disease often follows a recurrent course characterized by rapid calculus growth, a propensity for staghorn and multiple stone formation, the onset of emergency conditions, and disability, particularly in patients with underlying metabolic disorders. Furthermore, existing methods for calculus removal do not prevent the recurrence of UL [3].

Currently, most researchers are convinced that UL should be considered as a condition associated with metabolic syndrome (MetS), with proven involvement of its components in the mechanisms of stone formation [4].

The components of MetS (insulin resistance, abdominal obesity, dyslipidemia, arterial hypertension (AH), hyperuricemia, etc.) are now recognized as determinants of the severe course of associated pathologies. They impact the rates of disability and mortality, contributing to a significant decline in the quality of life of the population [5, 6].

The role of UL as an independent risk factor for chronic kidney disease (CKD) deserves particular attention. Recurrent stone formation, urinary tract obstruction, and chronic inflammation lead to renal parenchymal damage, interstitial fibrosis, and a progressive decline in kidney function. This progression, in combination with MetS components and other associated pathologies (atherosclerosis, diabetes mellitus (DM)), increases the risk of developing CKD.

A clinical case of a patient with recurrent UL against the background of MetS-associated conditions demonstrates the complex interplay of comorbid states. This interplay is characterized by mutual exacerbation, which aggravates the adverse outcome.

CLINICAL CASE

Patient K., 69 years old, was admitted to the urology department on 02.06.2025 for elective surgical treatment with a diagnosis of Urolithiasis. Stones in both kidneys. Right-sided staghorn calculus, K4.

The medical history for UL was significant. The first episode was recorded 30 years ago with spontaneous passage of a stone from the right kidney. In subsequent years, the disease relapsed with episodes of stone passage from both sides up to 2–3 times per year. In 2017, percutaneous nephrolithotomy (PNL) was performed on the right side, achieving 100% effectiveness according to the stone-free rate (SFR) criterion. Stone composition analysis revealed calcium oxalate (90%) and carbonate apatite (10%). In 2019, a follow-up examination again detected a stone in the right kidney. However, an attempt at its dissolution in the outpatient setting for one month using a preparation of potassium citrate + sodium citrate + citric acid yielded no results. A surgical intervention was recommended and was performed during the current hospitalization.

From the life history, it is known that the patient has been under long-term follow-up by a therapist for

chronic conditions: coronary artery disease: stable angina, FC I; post-infarction cardiosclerosis (PICS) since 2016, state after stenting of the right coronary artery (RCA) in 2017. Stage 3 hypertension, controlled. Grade 2 obesity. Dyslipidemia. Hyperuricemia. Cardiovascular risk category 4 (very high). Stage I chronic heart failure with preserved ejection fraction (EF 63%). Type 2 diabetes mellitus (HbA1c – 9.25%). Nephropathy of mixed genesis. Stage C4 CKD (eGFR – 25 ml / min).

The patient has been receiving continuous medication: dapagliflozin 10 mg, insulin detemir 12–12 units daily, insulin glulisine 8–10–8 units, losartan 50 mg, bisoprolol 5 mg, allopurinol 300 mg.

Objective findings: in addition to a standard physical examination, extended anthropometric measurements were performed to assess the degree of obesity and the nature of fat distribution. Height – 164 cm, body weight – 102 kg, calculated body mass index (BMI) – 37.9 kg / m², corresponding to grade 2 obesity; waist circumference – 115 cm, hip circumference – 100 cm, and sagittal abdominal diameter – 28 cm, indicative of abdominal obesity.

Results of the blood biochemistry dated 26.05.2025: creatinine – 150.5 μmol / l (eGFR 25 ml / min), glucose – 11.0 mmol / l, uric acid – 0.38 mmol / l, triglycerides – 2.6 mmol / l, low-density lipoproteins (LDL) – 2.08 mmol / l, total calcium – 2.7 mmol / l, magnesium – 0.56 mmol / l.

The acidity of the 24-hour urine sample (pH) dated 03.06.2025 was 5.5; microalbuminuria – 300 mg / day. No bacterial flora was detected.

According to the results of spiral computed tomography (CT) of the retroperitoneal organs from 14.02.2025: on the right, a staghorn calculus filling its shape; 39 × 35 × 20 mm, 460 HU. On the left, in the upper group of calyces, a calculus was seen measuring 5 × 7 mm, 300 HU (Fig. 1).

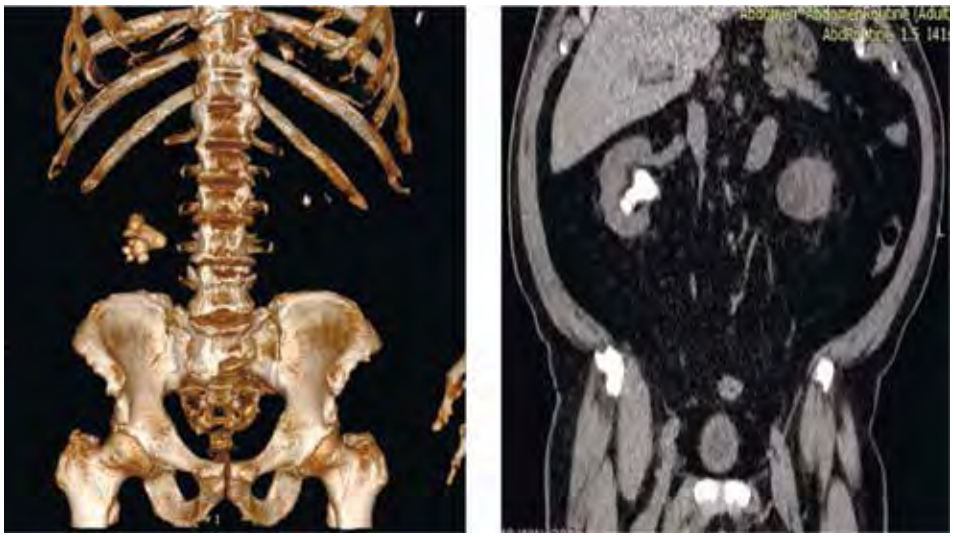

Fig. 1. Results of spiral computed tomography of the retroperitoneal organs dated 14.02.2025: on the right — a staghorn calculus (39 × 35 × 20 mm, 460 HU) filling its shape; on the left, in the upper group of calyces — a calculus of 5 × 7 mm, 300 HU

According to the STONE nephrolithometry scale, the case complexity was assessed at 11 points (high level).

To assess the cardiometabolic status, indirect calorimetry was performed using the COSMED equipment (Italy). The following parameters were determined at rest: oxygen consumption (VO_2) – 354 ml / min, carbon dioxide production (VCO_2) – 266 ml / min, respiratory quotient (RQ) – 0.75 (VCO_2 / VO_2), resting energy expenditure (REE) – 2,405 kcal / day. The obtained data demonstrate a pronounced reduction in aerobic capacity, impaired metabolic flexibility, and signs of a hypermetabolic state, which is characteristic of obesity and insulin resistance and indicates significantly reduced cardiorespiratory endurance.

The patient underwent right PNL in the prone position. Dilation of the percutaneous tract up to 24 CH was performed (Fig. 2).

A combined method using a thulium fiber laser FiberLase U2 and an electrohydraulic lithotripter Urolit 105M was employed for stone fragmentation; disintegration of the staghorn calculus into fragments was performed, and fragments were extracted with forceps.

During revision of the upper calyceal group, a residual stone fragment of up to 6 mm in diameter was found. Complete visualization and removal were impossible due to anatomical features of the pelvicalyceal system, limited instrument mobility, and a high risk of renal parenchyma injury. Given the operation duration of over two hours, high surgical risk (ASA III), reduced renal function, and high risk of infectious complications, a decision was made to terminate the surgical procedure with the placement of a nephrostomy drain. In the early postoperative period, the patient was prescribed prolonged antibiotic prophylaxis to prevent infectious complications. The postoperative period was uneventful. The nephrostomy drain was removed on day 2 with restoration of normal urine passage. The patient was discharged in satisfactory condition on day 5 after the surgery.

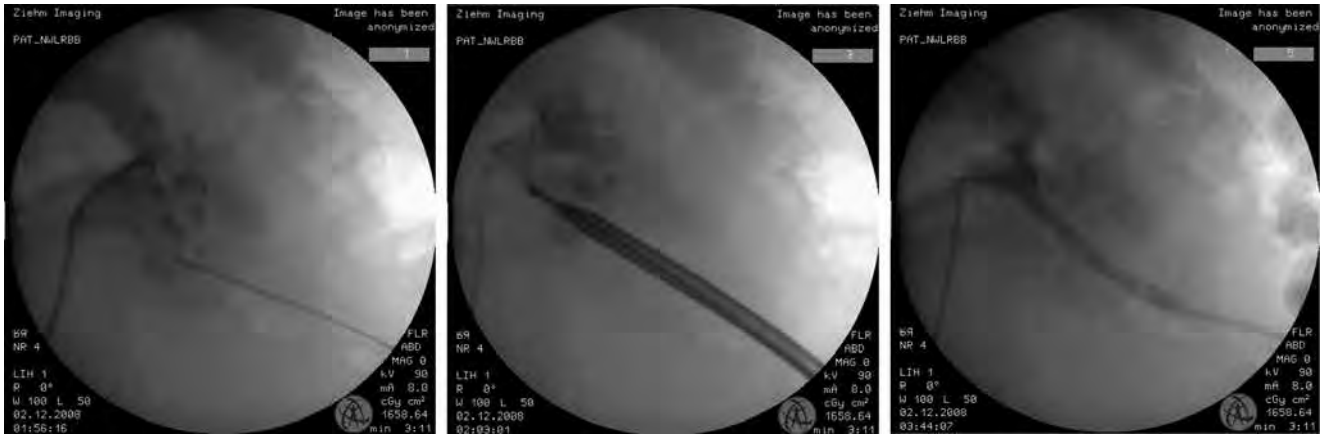

Fig. 2. Intraoperative radiograph (C-arm) during right percutaneous nephrolithotomy.

DISCUSSION

This clinical case presents a classic example of severe comorbid pathology, where UL develops against the background of a full-blown clinical presentation of MetS. The patient exhibited all key components of MetS: abdominal obesity, type 2 DM, AH, dyslipidemia, and hyperuricemia. This combination created ideal conditions for the formation of recurrent nephrolithiasis with the development of CKD [3].

A key aspect of this observation is the pronounced mutual exacerbation of comorbid conditions. The systemic nature of the damage, manifested by the formation of type 2 cardiorenal syndrome (a combination of CAD, stage I CHF, and stage C4 CKD) and metabolic disorders (type 2 DM, dyslipidemia, and hyperuricemia), deserves special attention.

According to the Quebec Cardiovascular Study, the risk of developing CAD in men with MetS is 20 times higher than in the general population. The pathogenesis of cardiovascular complications is based on macro- and microvascular damage. Macrovascular changes are associated with atherosclerosis, which develops under the influence of oxidized LDL, triggering a proinflammatory reaction in the vascular wall involving immune-competent cells and the cytokines they synthesize. In patients with type 2 DM and dyslipidemia, the production of oxygen free radicals increases, which reduces the synthesis of nitric oxide and prostacyclin, promoting vasoconstriction and the progression of atherosclerosis [7].

Furthermore, renal artery involvement in generalized atherosclerosis in patients with type 2 DM is more common over the age of 60. Retrospective data from angiographic studies confirm widespread lesions of the coronary, brachiocephalic, renal, and peripheral arteries [8].

A common pathogenetic link between UL and the aforementioned comorbid pathology is inflammation and free-radical oxidation, leading to renal tubular damage and formation of Randall's plaques, which serve as a foundation for subsequent crystal deposition, initiating nephrolithiasis [9].

Chronic heart failure associated with AH, CAD, and CKD, coupled with the activation of the renin – angiotensin – aldosterone system, is initially compensatory but subsequently leads to cardiac and renal remodeling with the development of fibrosis [10].

The rapid formation of a staghorn calculus, characteristic of patients with MetS-associated diseases, warrants special attention [11]. This is

due to chronic urine acidification and impaired calcium metabolism [12]. Microalbuminuria and a reduced GFR of 25 ml / min indicate significant renal parenchymal damage and confirm the link between UL and CKD [13].

According to current data, the risk of developing UL in patients with MetS increases by 2–3 times, with 50-60% of them experiencing a recurrent course of the disease [14]. Of particular concern is the fact that the combination of these pathologies triples the risk of developing CKD compared to isolated UL [13]. The pathogenesis of this interaction involves several key aspects. Central to this interaction is insulin resistance, leading to reduced citrate excretion and the development of hypercalciuria due to impaired calcium – phosphate homeostasis [12].

Hyperuricemia plays an important role in the pathogenesis, not only promoting urate crystallization but also activating tubulointerstitial inflammation [15]. Elevated uric acid levels are a significant factor in the development and progression of CKD, due to the development of AH against the background of endothelial dysfunction and reduced nitric oxide production. The deposition of uric acid crystals in ischemic areas of renal tissue against the background of elevated blood pressure leads to renal parenchymal damage [16]. Moreover, a high uric acid level itself may indicate reduced renal function due to impaired excretion [17].

Abdominal obesity as a component of MetS independently contributes to reduced renal function through a complex of mechanisms. Under conditions of chronic hemodynamic and metabolic disturbances, increased production of endothelin-1 takes place, which causes pronounced spasm of efferent arterioles and an increase in intrarenal pressure. Simultaneously, possessing growth factor properties, endothelin-1 stimulates the proliferation of cellular elements of renal tissue, leading to oligonephronia, hypertrophy of the remaining nephrons, and ultimately contributing to glomerulosclerosis and the progression of CKD [18].

An additional risk factor is dyslipidemia, which alters the lithogenic properties of urine through the development of hyperoxaluria, hypercalciuria, and hyperphosphaturia against the background of hypocitraturia. These changes significantly increase the risk of stone formation and exacerbate renal tissue damage [19]. These processes explain why in patients with MetS and UL, the rate of GFR decline reaches 5–8 ml / min / year, which is 4–5 times higher than in patients without metabolic disorders [20]. It is also known that in patients with CAD complicated by CHF, nephrolithiasis occurs 1.5 times more frequently, and the rate of GFR decline can be 4 ml / min / year [21]. Patients with MetS form staghorn calculi 3–4 times more often than the general population [12]. Rapid progression of renal dysfunction is noted: the 5-year risk of end-stage CKD is 15–20% compared to 3–5% in patients without MetS [22]. The early appearance of microalbuminuria, which serves as a marker of both diabetic and urate-induced nephropathy, is also characteristic [23]. The GUBBIO study noted that an increase in BMI by 4 kg / m² increased albumin excretion by 1.83 times in men [18].

A particular point of attention in this clinical case is the lack of consistent metaphylaxis over the 30-year history of the disease. The patient had all indications for aggressive metaphylaxis: recurrent nephrolithiasis, metabolic acidosis (pH 5.5), and concomitant metabolic disorders [11, 14]. Despite the recurrent nature of nephrolithiasis (2–3 episodes per year) and identified metabolic disorders, the patient received only episodic therapy (blemaren for 1 month), which is clearly insufficient for secondary prevention. This fact reflects systemic problems in modern urological practice. According to the Urologists' Attitudes on Metabolic Evaluation (NAME, 2021) study, only 38% of urologists regularly prescribe a metabolic evaluation after the first episode of UL [24]. In 72% of cases, they limit themselves to stone composition analysis without assessing urinary lithogenic factors [24]. Metaphylaxis should become a mandatory component of treatment for all patients with UL, especially in the presence of metabolic disorders. Timely initiation of preventive measures is the most effective way to prevent severe complications, including end-stage CKD.

The performed surgical intervention in the volume of PNL in this patient with staghorn nephrolithiasis and MetS-associated diseases demonstrated a number of important clinical aspects. The patient had severe comorbid pathology, which required minimizing intraoperative risks [25, 26]. Limiting the active intervention time to two hours was a justified decision, considering the high surgical risk (ASA III) and reduced renal function (GFR 25 ml / min); the placement of a nephrostomy drain ensured adequate urine drainage. It is important to note that in modern urology, during PNL, it is recommended to adhere to the golden hour rule – achieving maximum stone fragmentation efficiency within the first hour of surgery, which reduces the risk of complications

and improves long-term outcomes [25]. The absence of complications in the postoperative period, rapid restoration of independent urine passage, and stable renal function parameters confirmed the correctness of the chosen strategy, combining sufficient intervention radicality with patient safety [27]. However, the presence of a residual fragment indicates the need for further follow-up and possible retreatment.

CONCLUSION

Diseases associated with MetS are significant risk factors for the recurrent course of UL and the progression of CKD. This clinical case highlights the importance of timely diagnosis of metabolic disorders and their elimination for effective secondary prevention of MetS-associated diseases, including UL, preventing end-stage renal failure, and improving the quality of life.

Medical management of patients with UL against the background of comorbid pathology requires a multidisciplinary approach involving urologists, therapists, nephrologists, endocrinologists, and others. Timely correction of metabolic disorders and effective prevention of stone recurrence can significantly slow the progression of CKD in this category of patients.

REFERENCES

1. Braun T., Bittner A., Guttmann A., Reichert M.C., Schneider M.P., Gasser T. Epidemiology and risk factors of kidney stones in the world: an update of the global burden. *Journal of Clinical Medicine.* 2023;12(3):1048. DOI: 10.3390/jcm12031048.
2. Tiktinskii O.L., Aleksandrov V.P. Urolithiasis. Saint Petersburg: Piter; 2020:336. (In Russ.).
3. Shen Z., Xie L., Luo D., Xie H., Chen H., Liu C. Incidence of metabolic syndrome in patients with unilateral or bilateral staghorn renal stones and its impact on percutaneous nephrolithotomy outcomes. *BMC Urol*. 2024;24(1):142. DOI: 10.1186/s12894-024-01526-4.
4. Thomas G., Sehgal A.R., Kashyap S.R., Srinivas T.R., Kirwan J.P., Navaneethan S.D. Metabolic syndrome and kidney disease: a systematic review and meta-analysis. *Clin. J. Am. Soc. Nephrol*. 2011;6(10):2364–2373. DOI: 10.2215/CJN.02180311.
5. Bespalova I.D., Bychkov V.A., Kalyuzhin V.V., Ryazantseva N.V., Medyantsev Yu.A., Osikhov I.A. et al. Quality of Life in Patients with Hypertension and Metabolic Syndrome: Relationship with Systemic Inflammation Markers. *Bulletin of Siberian Medicine*. 2013;12(6):5–15. (In Russ.).
6. Bespalova I.D., Kalyuzhin V.V., Medyantsev Yu.A. Quality of Life in Patients with Coronary Heart Disease: Relationship with Metabolic Syndrome Components and Systemic Inflammation Markers. *Bulletin of Siberian Medicine*. 2012;11(6):17–20. (In Russ.).
7. Gurgenyan S.V., Vatinyan S.Kh., Zelveyan P.A. Metabolic Syndrome and Coronary Heart Disease. *Terapevticheskii Arkhiv*. 2014;86(3):106–110. (In Russ.).
8. Amosova M.V., Gurova O.Yu. Kidney Damage in Type 2 Diabetes Mellitus: is it only Diabetic Nephropathy? *Klinicheskaya Nefrologiya*. 2017;(4):72–76. (In Russ.).
9. Prosiannikov M.Yu., Anokhin N.V., Golovanov S.A., Kirpatovskii V.I., Sivkov A.V., Konstantinova O.V. et al. Urolithiasis and Cardiovascular Diseases: only Statistical Relationship or Common Pathogenetic Mechanisms? *Experimental and Clinical Urology*. 2018;(3):34–41. (In Russ.).
10. Shutov A.M. Consilium. *Nephrology/ Cardiology/ Oncology/ Dermatovenereology*. 2015;1(131):20–24. (In Russ.).
11. Wong Y.V., Cook P., Somani B.K. The association of metabolic syndrome and urolithiasis. *Int. J. Endocrinol*. 2015;2015:570674. DOI: 10.1155/2015/570674.
12. Cupisti A., Meola M., D'Alessandro C., Bernabini G., Pasquali E., Carpi A. et al. Insulin resistance and low urinary citrate excretion in calcium stone formers. *Biomed. Pharmacother.* 2007;61(1):86–90. DOI: 10.1016/j.biopha.2006.09.012.
13. Rule A.D., Roger V.L., Melton L.J. 3rd, Bergstralh E.J., Li X., Peyser P.A. et al. Kidney stones associate with increased risk for chronic kidney disease. *Clin. J. Am. Soc. Nephrol*. 2009;4(4):804–808. DOI: 10.2215/CJN.05811108.
14. Ferraro P.M., Taylor E.N., Eisner B.H., Gambaro G., Rimm E.B., Mukamal K.J. et al. Metabolic syndrome and the risk of recurrent nephrolithiasis: results from the Nurses' Health Study II. *Clin. J. Am. Soc. Nephrol*. 2013;8(8):1330–1336. DOI: 10.2215/CJN.04510413.
15. Jung S.W., Kim S.M., Kim Y.G., Lee S.H., Moon J.Y. Uric acid and inflammation in kidney disease. *Am. J. Physiol. Renal. Physiol*. 2020;318(6):F1327–F1340. DOI: 10.1152/ajprenal.00272.2019.
16. Mironova O.Yu. Hyperuricemia and Kidney Damage in Patients with Cardiovascular Diseases. *Terapevticheskii Arkhiv.* 2022;94(12):1426–1430. (In Russ.). DOI: 10.26442/00403660.2022.12.201999.
17. Maksudova A.N., Khalfina T.N., Almukhametova A.I. A Patient with Hyperuricemia and Chronic Kidney Disease at the Appointment: Management Features according to Russian Recommendations. *Therapy*. 2023;(3):129–135. (In Russ.). DOI: 10.18565/therapy.2023.3.129-135.
18. Sadulaeva I.A., Vasyuk Yu.A., Trofimenko O.S., Yushchuk E.N., Ivanova S.V., Shupenina E.Yu. et al. Obesity in Metabolic Syndrome and Renal Dysfunction. *Terapevticheskii Arkhiv*. 2016;88(6):93–98. (In Russ.). DOI: 10.17116/terarkh201688693-98.
19. Bespalova I.D., Boshchenko V.S., Koshchavtseva Yu.I., Tsoi A.V., Teteneva A.V., Mesko P.E. et al. Gender Aspects of Urolithiasis Development in Patients with Metabolic Syndrome. *Bulletin of Siberian Medicine*. 2021;20(4):123–130. (In Russ.). DOI: 10.20538/1682-0363-2021-4-123-130.
20. Keddis M.T., Rule A.D. Nephrolithiasis and loss of kidney function. C*urr. Opin. Nephrol. Hypertens*. 2013;22(4):390–396. DOI: 10.1097/MNH.0b013e32836214b9.
21. Royuk R.V., Yarovoi S.K. Chronic Kidney Disease in Patients

with Recurrent Nephrolithiasis and Concomitant Cardiovascular Disease. *Klinicheskaya Nefrologiya*. 2021;9(3):52–61. (In Russ.). DOI: 10.21886/2308-6424-2021-9-3-52-61.
22. Alexander R.T., Hemmelgarn B.R., Wiebe N., Bello A., Morgan C., Samuel S. et al. Progression to ESRD in stone formers. *Kidney Int*. 2016;89(5):1177–1185. DOI: 10.1016/j.kint.2016.01.015.
23. Shaderkina V.A., Dzeranov N.K., Prosiannikov M.Yu. Renal Dysfunction in the Combination of Urolithiasis and Metabolic Syndrome. *Nephrology*. 2023;27(1):56–64. (In Russ.). DOI: 10.36485/1561-6274-2023-27-1-56-64.
24. Karagöz M.A., Güven S., Tefik T., Gökçe M.İ., Kiremit M.C., Atar F.A. et al. Attitudes of urologists on metabolic evaluation for urolithiasis: outcomes of a global survey from 57 countries. *Urolithiasis*. 2022;50(6):711–720. DOI: 10.1007/s00240-022-01362-x.
25. Desai M., Jain P., Ganpule A., Sabnis R., Patel S., Shrivastav P. Developments in technique and technology: the effect on the results of percutaneous nephrolithotomy for staghorn calculi. *BJU Int.* 2009;104(4):542–548. DOI: 10.1111/j.1464-410X.2009.08472.x.
26. Abouelgreed T.A., Ismail H., Ali S.S., Koritenah A.K., Badran Y., Ali M. et al. Safety and efficacy of percutaneous nephrolithotripsy in comorbid patients: a 3-year prospective observational study. *Archivio Italiano di Urologia e Andrologia*. 2023;95(3):11581. DOI: 10.4081/aiua.2023.11581.
27. Russian Society of Urology. Clinical Guidelines on Urolithiasis. M: RSU, 2023:56. (In Russ.).

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