DOI: https://doi.org/10.17816/uroved643098

Simple renal cyst and glomerulopathy: is there a connection?

Mikhail A. Firsov[1,2], Pavel A. Simonov[1,2], Tatyana A. Garkusha[1,3], Evgenii A. Bezrukov[1,4], Dmitrii I. Laletin[1,2], Semen E. Nagaev[1], Valeriya D. Moiseeva[1]

[1] Professor V.F. Voino-Yasenetsky Krasnoyarsk State Medical University, Krasnoyarsk, Russia;
[2] Regional Clinical Hospital, Krasnoyarsk, Russia;
[3] Krasnoyarsk Regional Pathological Bureau, Krasnoyarsk, Russia;
[4] The First Sechenov Moscow State Medical University (Sechenov University), Moscow, Russia

ABSTRACT

BACKGROUND: Simple renal cysts are quite common in the general population and are often accompanied by a decrease in the glomerular filtration rate, which may be linked to latent glomerulopathies. Delayed diagnosis of glomerular damage inevitably leads to the progression of chronic kidney disease.

AIM: To assess the likelihood of hidden glomerular lesions in patients with simple renal cysts.

MATERIALS AND METHODS: The study involved a group of 78 patients, including 29 men (37%) and 49 women (63%), with renal cysts of classes I and II according to the Bosniak classification (2019). An exclusion criterion for the study was a history of nephrological diseases. The mean age of the patients was 59.11±1.47 years, and the mean cyst size was 7.19±1.98 cm. All patients underwent laparoscopic excision of the renal cyst walls. Intraoperatively, a renal parenchymal biopsy was performed under visual control, and the nephrobiopsy specimens were examined using light microscopy, immunofluorescence analysis, and electron microscopy.

RESULTS: Based on the analysis of 234 nephrobiopsy specimens, morphological signs of glomerulopathy were identified in 20.5% of patients, including diabetic nephropathy (37.5%), focal segmental glomerulosclerosis (31.3%), mesangioproliferative IgA glomerulonephritis and hypertensive nephropathy (12.5% each), and thin basement membrane disease (6.2%). Patients with glomerulopathy exhibited reduced glomerular filtration rate and increased creatinine and uric acid levels in the serum. Moreover, proteinuria and leukocyturia were more frequently observed in the common urinalysis of this group of patients.

CONCLUSIONS: The combination of a renal cyst, changes in urinalysis, and biochemical blood analysis may indicate hidden glomerular injury. Focusing on the markers of renal damage in the preoperative period allows for determining the indications for intraoperative nephrobiopsy during surgical treatment of renal cysts. This facilitates early morphological identification of glomerulopathy and timely initiation of nephroprotective therapy to reduce the risk of chronic kidney disease progression.

Keywords: renal cyst; glomerulopathy; nephrobiopsy; chronic kidney disease; glomerular filtration rate.

To cite this article
Firsov MA, Simonov PA, Garkusha TA, Bezrukov EA, Laletin DI, Nagaev SE, Moiseeva VD. Simple renal cyst and glomerulopathy: is there a connection? *Urology reports (St. Petersburg)*. 2025;15(1):25–32. DOI: https://doi.org/10.17816/uroved643098

Received: 17.12.2024 **Accepted:** 22.01.2025 **Published online:** 31.03.2025

DOI: https://doi.org/10.17816/uroved643098

Неосложненная киста почки и гломерулопатии. Есть ли связь?

М.А. Фирсов[1,2], П.А. Симонов[1,2], Т.А. Гаркуша[1,3], Е.А. Безруков[1,4], Д.И. Лалетин[1,2], С.Е. Нагаев[1], В.Д. Моисеева[1]

[1] Красноярский государственный медицинский университет им. проф. В.Ф. Войно-Ясенецкого, Красноярск, Россия;
[2] Краевая клиническая больница, Красноярск, Россия;
[3] Красноярское краевое патолого-анатомическое бюро, Красноярск, Россия;
[4] Первый Московский государственный медицинский университет им. И.М. Сеченова (Сеченовский Университет), Москва, Россия

АННОТАЦИЯ

Актуальность. Простые кисты почек весьма распространены в общей популяции и часто сопровождаются снижением скорости клубочковой фильтрации, что может быть связано с латентными гломерулопатиями. Несвоевременная диагностика поражения клубочков почки неизбежно приводит к прогрессии хронической болезни почек.

Цель — оценить вероятность наличия скрытых гломерулярных поражений у пациентов с простыми кистами почек.

Материалы и методы. Исследование проведено в группе из 78 пациентов, из них — 29 мужчин (37%) и 49 женщин (63%) с кистами почек 1-й и 2-й категории по классификации Bosniak (2019 г.). Критерием исключения из исследования были нефрологические заболевания в анамнезе. Средний возраст пациентов составил 59,11±1,47 года, средний размер кисты — 7,19±1,98 см. Всем пациентам проведено иссечение стенок кист почек лапароскопическим доступом. Интраоперационно выполнялась биопсия почечной паренхимы под визуальным контролем с исследованием нефробиоптата путем светооптической микроскопии, реакции иммуннофлуоресценции и электронной микроскопии.

Результаты. По результатам анализа 234 нефробиоптатов у 20,5% пациентов выявлены морфологические признаки гломерулопатии: диабетическое поражение — 37,5%, фокально-сегментарный гломерулосклероз — 31,3%, мезангиопролиферативный IgA-гломерулонефрит и гипертоническая нефропатия — по 12,5%, болезнь тонких базальных мембран — 6,2%. Пациенты с гломерулопатией характеризовались снижением скорости клубочковой фильтрации и повышением концентрации креатинина и мочевой кислоты в сыворотке крови. При этом в общем анализе мочи у пациентов этой группы чаще встречались протеинурия и лейкоцитурия.

Выводы. Сочетание кисты почки, изменений анализа мочи, биохимического анализа крови может быть признаками скрытого гломерулярного поражения. Акцентирование на наличие маркеров почечного повреждения в дооперационном периоде позволяет определить показания для проведения интраоперационной нефробиопсии в ходе оперативного лечения по поводу кист почек с целью ранней морфологической идентификации гломерулопатии и своевременного назначения нефропротективной терапии для снижения риска прогрессии хронической болезни почек.

Ключевые слова: киста почки; гломерулопатия; нефробиопсия; хроническая болезнь почек; скорость клубочковой фильтрации.

Как цитировать

Фирсов М.А., Симонов П.А., Гаркуша Т.А., Безруков Е.А., Лалетин Д.И., Нагаев С.Е., Моисеева В.Д. Неосложненная киста почки и гломерулопатии. Есть ли связь? // Урологические ведомости. 2025. Т. 15, № 1. С. 25–32. DOI: https://doi.org/10.17816/uroved643098

Рукопись получена: 17.12.2024 **Рукопись одобрена:** 22.01.2025 **Опубликована online:** 31.03.2025

BACKGROUND

Renal cysts are structural lesions of the kidneys that appear as one or several closed pouches inside the kidneys. These pouches are surrounded by a connective tissue capsule and filled with serous fluid. The prevalence of renal cysts in the general population varies between 20% and 50% [1, 2]. Simple renal cysts tend to increase in size at an average rate of 3.9% to 5% per year, with the potential to grow to twice their original size within a decade [3–5].

The available evidence suggests that the risk factors for this disease include elderly age, male sex, smoking, nephrolithiasis, and elevated creatinine levels [6–8]. Russian studies have demonstrated a negative correlation between fluid-filled renal lesions and glomerular filtration rate (GFR) associated with parenchymal atrophy [9]. Simple renal cysts are common findings in nephrological patients with chronic kidney disease (CKD) [10]. The most significant etiological factors of CKD are arterial hypertension, diabetes mellitus, and glomerulopathy [11, 12]. The latter refers to a disease of the renal glomerulus of any pathogenesis, including glomerulonephritis. *Glomerulonephritis* comprises a subgroup of renal diseases in which immune-mediated damage to the capillary basement membrane, mesangial and endothelial cells contribute to hematuria, proteinuria, and azotemia [13]. The most prevalent forms of glomerulonephritis include IgA nephropathy, membranous nephropathy, nephrotic syndrome, minimal change disease, focal segmental glomerulosclerosis, and postinfectious glomerulonephritis.

Glomerulopathies, whether primary or secondary, i.e., those associated with systemic autoimmune diseases, drug therapy, or malignant tumors, may affect patients of any age. Most registries of patients with terminal CKD report that glomerular diseases account for 20%–25% of cases, with progression often occurring within weeks to months following the onset of acute nephritic syndrome [14].

The population analysis, including patients receiving renal replacement therapy in the Krasnoyarsk Territory also suggests a high prevalence of glomerular diseases, with a proportion of more than 30% [15]. Notably, glomerulopathies are consistently associated with a bilateral lesion, which invariably causes a substantial decline in renal function in some patients [16–18]. Many glomerular lesions, regardless of their etiology, are asymptomatic or accompanied by mild symptoms, thereby challenging diagnosis. A delayed diagnosis of renal glomerular lesions inevitably contributes to a decrease in glomerular filtration rate [11].

The study aimed to assess the probability of latent glomerular lesions in patients with simple renal cysts.

METHODS

The study population comprised 78 patients who were admitted to the urology department at the Krasnoyarsk Regional Clinical Hospital for surgical treatment of symptomatic renal cysts between 2023 and 2024. The preoperative assessment included medical history and standard laboratory tests, such as hematology, blood chemistry, and urinalysis. The GFR was calculated using the standardized equations of MDRD (Modification of Diet in Renal Disease Study), CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration), and Cockcroft–Gault. The instrumental assessment included renal ultrasound and contrast-enhanced multi-detector computed tomography. The Bosniak Classification (2019) was used to stratify the risk of malignancy in cystic renal masses. The study included 29 males (37%) and 49 females (63%) with Bosniak I and II renal cysts. An exclusion criterion was a history of nephrological diseases.

The mean age of the patients was 59.11±1.47 years, and the mean cyst size was 7.19±1.98 cm. All patients underwent laparoscopic excision of renal cyst walls. Intraoperative renal parenchymal biopsy was performed under visual control, and biopsy specimens were examined by light microscopy, immunofluorescence staining, and electron microscopy [19]. The study procedures were approved by the Local Ethics Committee of the Krasnoyarsk Regional Clinical Hospital (Protocol No. 189/6, September 29, 2002) and the Krasnoyarsk State Medical University (Protocol No. 119/2023, July 6, 2002) and were in compliance with the standards of the Ethics Committee and the Declaration of Helsinki (1964) as amended. All participating patients provided their voluntary, informed consent to participate in the study. In addition to standard histology of the cyst wall specimens, morphology of the renal parenchymal specimens evaluated the number of glomeruli, the number and percentage of globally sclerotic glomeruli, the number and percentage of segmentally sclerotic glomeruli, signs of mesangial cell proliferation, the number of abnormal glomeruli, tubular atrophy, vascular sclerosis and hyalinosis, and the presence of lymphoid follicles.

The statistical data analysis was performed using Statistica v. 10.0. The non-parametric Mann–Whitney test (*U* test) was used to calculate the significance. The percentage frequency of the parameters was calculated using Fisher's test.

RESULTS

The analysis of 234 intraoperative renal biopsy specimens obtained from 78 patients demonstrated morphological patterns of glomerular lesions in 19 (24.4%) cases. Specifically, 4 (21.1%) cases of diabetic nephropathy (Fig. 1) and 5 (26.3%) cases of focal segmental glomerulosclerosis (Fig. 2) were identified.

Glomerulopathy was suspected in 10 (52.6%) patients, including 7 patients having glomerular lesions confirmed by immunohistochemistry. Specifically, mesangial proliferative glomerulonephritis with suspected IgA nephropathy was diagnosed in two cases (positive staining for both kappa and lambda light chain subtypes) (Fig. 3, Fig. 4). One case of thin basement membrane nephropathy, two cases of diabetic nephropathy and two cases of hypertensive nephropathy with focal segmental glomerulosclerosis were also reported.

Based on the analysis of the obtained renal biopsy specimens, a group of 62 (79.5%) patients without glomerular lesions was identified. This group was taken as controls (group 1). Sixteen (20.5%) patients with morphologically confirmed glomerulopathy were assigned to the study group (group 2). The ultrasound comparison showed no differences in the renal size between the groups (Table 1).

Group 1 (controls) consisted of 36 (58.1%) females and 26 (41.9%) males. Thirty-six (58.1%) cases of hypertension, 1 (1.6%) case of nodular goiter, and 1 (6.1%) hyperuricemia were reported. Twenty-four (38.7%) patients had no concomitant diseases. Group 2 (the study patients) was mostly composed of females with 12 (75%) patients. This group included 4 (25%) patients with diabetes mellitus and 8 (50%) with hypertension. The patients in group 2 had higher mean creatinine and uric acid levels and lower GFR, calculated using all standardized equations, compared with those in group 1. The differences in these parameters between patients in the two groups were statistically significant (Table 2).

Although there were no significant differences between the groups in urine specific gravity and the number of red blood cells found by urinalysis, more severe proteinuria was reported for group 2. The patients in this group also had more white blood cells in the field of vision compared with the control group (Table 3).

The renal parenchyma morphology showed a significantly lower number of glomeruli in the renal biopsy specimens from patients in group 1 as compared with those in group 2. No significant differences were found in the number and frequency of globally

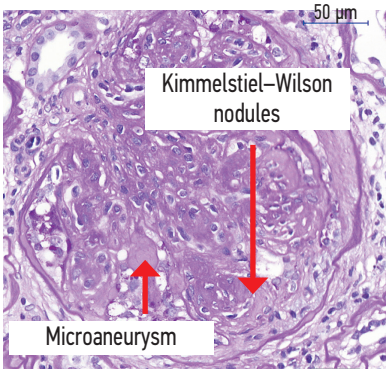

Fig. 1. Diabetic nephropathy, class III. Periodic acid-Schiff staining. Magnification ×400. Kimmelstiel–Wilson nodules are observed.
Рис. 1. Диабетическая нефропатия, класс III. Окраска Шифф-йодной кислотой. Увеличение ×400. Визуализируются узелки Киммелстила–Уилсона.

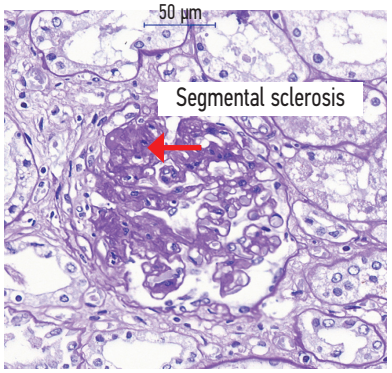

Fig. 2. Focal segmental glomerulosclerosis. Periodic acid-Schiff staining. Magnification ×400. Segmental glomerulosclerosis with adhesion of capillary loops to Bowman's capsule is observed.
Рис. 2. Фокально-сегментарный гломерулосклероз. Окраска Шифф-йодной кислотой. Увеличение ×400. Визуализируется сегментарный склероз клубочка с наличием спайки капиллярных петель с капсулой Боумена.

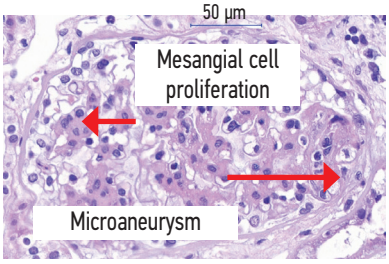

Fig. 3. IgA nephropathy. Hematoxylin and eosin staining. Magnification ×400. Mesangial proliferation and segmental sclerosis are observed.
Рис. 3. IgA-нефропатия. Окраска гематоксилином и эозином. Увеличение ×400. Визуализируется мезангиальная пролиферация и сегментарный склероз.

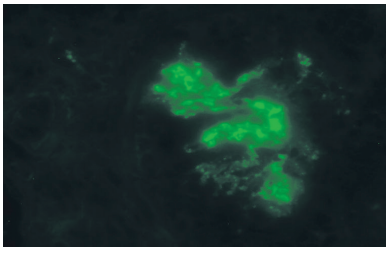

Fig. 4. Immunofluorescence reaction with anti-IgA antibodies in IgA nephropathy. Mesangial fluorescence. Magnification ×400.
Рис. 4. Реакция иммуннофлуоресценции с антителами к IgA при IgA-нефропатии. Мезангиальное свечение. Увеличение ×400.

DOI: https://doi.org/10.17816/uroved643098

Table 1. Kidney size of patients in Group 1 and Group 2 based on ultrasound findings

Таблица 1. Размеры почек пациентов 1-й и 2-й групп по результатам ультразвукового исследования

Parameters	Group 1 (*n*=62)	Group 2 (*n*=16)	*p*
Right kidney			
Length, cm	11.3 [10.5; 12.0]; 95% CI 10.5–11.8	10.9 [10.1; 11.1]; 95% CI 10.1–11.1	*U* 42.5; >0.05
Width, cm	5.0 [4.7; 5.4]; 95% CI 4.7–5.3	5.5 [4.5; 5.7]; 95% CI 4.5–5.7	*U* 49.0; >0.05
Parenchymal thickness, cm	1.6 [1.5; 1.9]; 95% CI 1.5–1.9	1.7 [1.6; 1.9]; 95% CI 1.6–1.9	*U* 56.0; >0.05
Left kidney			
Length, cm	11.2 [10.8; 12.1]; 95% CI 10.9–12.0	11.0 [10.3; 12.1]; 95% CI 10.3–12.1	*U* 57.0; >0.05
Width, cm	5.3 [5.0; 5.5]; 95% CI 5.1–5.4	5.5 [5.0; 5.7]; 95% CI 5.0–5.7	*U* 63.4; >0.05
Parenchymal thickness, cm	1.6 [1.4; 1.8]; 95% CI 1.4–1.8	1.7 [1.6; 1.8]; 95% CI 1.6–1.8	*U* 46.0; >0.05

Table 2. Parameters of kidney functional status in patients before surgery, *M*±σ

Таблица 2. Показатели функционального состояния почек пациентов до оперативного вмешательства, *M*±σ

Parameters	Group 1 (*n*=62)	Group 2 (*n*=16)	Difference, %	*p*
Creatinine, mmol/L	89.95±2.19	98.38±9.90	9.37	<0.05
Glomerular filtration rate (CKD-EPI equation), mL/min × 1.73 m^2	73.25±2.11	62.88±5.05	–14.15	<0.0001
Glomerular filtration rate (MDRD equation), mL/min × 1.73 m^2	69.36±2.1	59.43±4.62	–14.31	<0.0001
Glomerular filtration rate (Cockcroft–Gault equation), mL/min	87.11±3.18	82.12±8.28	–5.72	<0.005
Uric acid, mmol/L	334.35±12.17	356.86±23.59	6.73	<0.05

Table 3. Parameters of common urinalysis in patients, *M*±σ

Таблица 3. Показатели общего анализа мочи у пациентов, *M*±σ

Parameters	Group 1 (*n*=62)	Group 2 (*n*=16)	*p*
Protein, g/L	0.07±0.01	0.18±0.04	<0.05
Specific gravity, g/L	1014.0±0.71	1014.4±1.69	0.45
Red blood cells (field of vision)	6.11±1.18	6.1±3.03	0.31
White blood cells (field of vision)	6.54±1.29	7.75±2.43	<0.05

sclerotic glomeruli and the frequency of mesangial cell proliferation and vascular sclerosis between the groups. The patients in group 2 had a significantly higher frequency of segmentally sclerotic glomeruli than those in the control group (50% vs. 1.6%; p <0.05). The percentage of abnormal glomeruli and the frequency of glomerular vascular hyalinosis were significantly higher in group 2 (Table 4).

DISCUSSION

The study findings demonstrated that the patients in group 2, i.e., those with morphologically confirmed glomerulopathies, had lower GFRs and higher serum creatinine and uric acid levels. Moreover, this group experienced more severe proteinuria and leukocyturia compared with the control group. Along with the decreased renal function, the patients in the study group

Table 4. Histopathological findings of kidney biopsies in patients
Таблица 4. Результаты гистологического исследования биоптатов почек пациентов

Parameters	Group 1 (*n*=62)	Group 2 (*n*=16)	*p*
Average number of glomeruli in the biopsy specimen, *M*±σ	15.77±1.73	13.19±1.53	<0.05
Average number of globally sclerotic glomeruli, *M*±σ	2.36±0.45	2.29±0.84	0.62
Parameter frequency, %			
Globally sclerotic glomeruli	41.94	50	0.58*
Segmentally sclerotic glomeruli	1.6	50	<0.05*
Mesangial cell proliferation	17.74	31.25	0.28*
Abnormal glomeruli	54.83	87.5	<0.05*
Tubular atrophy	80.64	87.5	0.72*
Vascular sclerosis	58.06	81.5	0.08*
Vascular hyalinosis	0	12.5	<0.05*

*Fisher's Criterion / *Критерий Фишера

had higher frequencies of abnormal glomeruli, segmental sclerotic glomeruli, and vascular hyalinosis. Morphologically confirmed glomerulopathy was more common among female patients. It is worth noting that renal biopsy demonstrated signs of diabetic glomerular lesions in 6 patients, whereas type 2 diabetes mellitus was diagnosed in 4 patients. Although 2 patients presented with signs of diabetic nephropathy, the diagnosis of diabetes mellitus was not established. Subsequent assessment of these patients showed fasting hyperglycemia in one and impaired glucose tolerance in the other, suggesting prediabetes associated with carbohydrate metabolism disorders and characterized by blood glucose levels below the diagnostic threshold for diabetes mellitus but above normal values [20, 21]. The early detection of glycemia is clinically significant, as up to 50% of dysglycemic patients may progress to diabetes mellitus [21]. In patients with prediabetes, the risk of the CKD onset and progression is higher [22, 23].

In the Russian population, the prevalence of glomerular and tubulointerstitial diseases is approximately 1,500 cases per 100,000 people [24]. No official statistics or population-based studies on the prevalence, morbidity, and mortality due to glomerular diseases, including IdA nephropathy, were found. The prevalence of immune-mediated glomerulopathies, including IgA nephropathy, is estimated at 300–450 cases per 100,000 patients with glomerular and interstitial renal diseases. The findings of this study suggest that morphological patterns of glomerulopathies are seen in 20.5% of patients after renal cyst surgery. The most common glomerular diseases are diabetic lesions and focal segmental glomerulosclerosis (37.5% and 31.3%, respectively), followed by mesangial proliferative IgA glomerulonephritis and hypertensive nephropathy (12.5% each), and thin basement membrane nephropathy (6.2%). The obtained findings suggest that a combination of renal cysts, abnormal urinalysis and blood chemistry may be indicative of a latent glomerular lesion. Preoperative biomarkers of renal injury may help assess the indications for intraoperative renal biopsy in patients with renal cysts. This will facilitate the early morphologic identification of glomerulopathies and the prompt initiation of nephroprotective therapy to reduce the risk of progression to CKD.

CONCLUSION

The study findings demonstrated that biomarkers of renal injury associated with renal cysts may be used to support the intraoperative renal biopsy at the time of renal cyst wall excision for the diagnosis of latent glomerular disease.

ADDITIONAL INFO

Authors' contribution. M.A. Firsov, concept and design of the study, performing surgical operations, analysis of the obtained data, editing the text of the manuscript; P.A. Simonov, performing surgical operations, analysis of the obtained data, writing the text of the manuscript; T.A. Garkusha, performing morphological studies, analysis of the obtained data; E.A. Bezrukov, concept and design of the study, analysis of the obtained data, editing the text of the manuscript; D.I. Laletin, performing surgical operations; S.E. Nagaev, V.D. Moiseeva, analysis of the obtained data.

Ethics approval. The study was approved by the Ethical Committees of Regional Clinical Hospital (protocol No. 189/6 dated 2022 Sept 29) and Professor V.F. Voino-Yasenetsky Krasnoyarsk State Medical University of the Ministry of Health of Russia (protocol No. 119/2023 dated 2023 Jun 07).

Consent for publication. The authors received written informed voluntary consent from patients to publish personal data in a scientific journal, including its electronic version. The scope of published data was agreed with the patients.
Funding sources. No funding.
Disclosure of interests. The authors have no relationships, activities or interests for the last three years related with for-profit or not-for-profit third parties whose interests may be affected by the content of the article.
Generative AI. Generative AI technologies were not used for this article creation.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. М.А. Фирсов — концепция и дизайн исследования, выполнение хирургических вмешательств, анализ полученных данных, редактирование текста рукописи; П.А. Симонов — выполнение хирургических вмешательств, анализ полученных данных, написание текста рукописи; Т.А. Гаркуша — выполнение морфологических исследований, анализ полученных данных; Е.А. Безруков — концепция и дизайн исследования, анализ полученных данных, редактирование текста рукописи; Д.И. Лалетин — выполнение хирургических вмешательств; С.Е. Нагаев, В.Д. Моисеева — анализ полученных данных.
Этическая экспертиза. Исследование одобрено Этическими комитетами КГБУЗ «Краевая клиническая больница» (протокол № 189/6 от 29.09.2022) и ФГБОУ ВО «Красноярский государственный медицинский университет им. проф. В.Ф. Войно-Ясенецкого» Минздрава России (протокол № 119/2023 от 07.06.2023).
Согласие на публикацию. Авторы получили письменное информированное добровольное согласие пациентов на публикацию персональных данных в научном журнале, включая его электронную версию. Объем публикуемых данных с пациентами согласован.
Источники финансирования. Отсутствуют.
Раскрытие интересов. Авторы заявляют об отсутствии отношений, деятельности и интересов за последние три года, связанных с третьими лицами (коммерческими и некоммерческими), интересы которых могут быть затронуты содержанием статьи.
Генеративный искусственный интеллект. При создании настоящей статьи технологии генеративного искусственного интеллекта не использовали.

REFERENCES | СПИСОК ЛИТЕРАТУРЫ

1. Bas O, Nalbant I, Sener C, et al. Management of renal cysts. *JSLS*. 2015;19(1): e2014.00097. doi: 10.4293/JSLS.2014.00097
2. Skolarikos A, Laguna MP, de la Rosette JJ. Conservative and radiological management of simple renal cysts: a comprehensive review. *BJU Int*. 2012;110(2):170–178. doi: 10.1111/j.1464-410X.2011
3. Ryu DS, Oh TH. Laparoscopic decortication of large renal cysts: a comparison between the transperitoneal and retroperitoneal approaches. *J Laparoendosc Adv Surg Tech*. 2009;9(5):629–632. doi: 10.1089/lap.2009.0008
4. Mao X, Xu G, Wu H, Xiao J. Ureteroscopic management of asymptomatic and symptomatic simple parapelvic renal cysts. *BMC Urol*. 2015;15:48. doi: 10.1186/s12894-015-0042-5 EDN: QCXMKX
5. Floege J, Johnson RJ, Feehally J. *Comprehensive clinical nephrology. 4th edit*. London: Elsevier Mosby; 2010. 1200 p.
6. Terada N, Arai Y, Kinukawa N, Terai A. The 10-year natural history of simple renal cysts. *Urology*. 2008;71(1):7–11. doi: 10.1016/j.urology.2007.07.075
7. Hommos MS, Glassock RJ, Rule AD. Structural and functional changes in human kidneys with healthy aging. *J Am Soc Nephrol*. 2017;28(10):2838–2844. doi: 10.1681/ASN.2017040421
8. Chang C-C, Kuo J-Y, Chan W-L, et al. Prevalence and clinical characteristics of simple renal cyst. *J Chinese Med Assoc*. 2007;70(11):486–491. doi: 10.1016/S1726-4901(08)70046-7
9. Malkhasyan VA, Makhmudov TB, Gilfanov YuSh, et al. Effect of a simple kidney cyst on renal function. *Urologiia*. 2023;(4):75–81. doi: 10.18565/urology.2023.4.75-81 EDN: WNGWKE
10. Marumo K, Horiguchi Y, Nakagawa K, et al. Incidence and growth pattern of simple cysts of the kidney in patients with asymptomatic microscopic hematuria. *Int J Urol*. 2003;10(2):63–67. doi: 10.1046/j.1442-2042.2003.00577.x EDN: BEXFHT
11. Colvin RB, Chang A, Cornell LD. *Diagnostic pathology: Kidney diseases. 4th edit*. Amsterdam: Elsevier; 2023. 1176 p.
12. Zharikov AYu, Shchekochikhina RO. Diabetic nephropaty. Modern view of the problem. *Bulletin of Medical Science*. 2018;(2):22–28. EDN: XPTVET
13. Coresh J, Astor BC, Greene T, et al. Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. *Am J Kidney Dis*. 2003;41(1):1–12. doi: 10.1053/ajkd.2003.50007
14. Bobkova IN, Bulanov NM, Zakharova EV, et al. KDIGO 2021 clinical practice guideline for the management of GLOMERULAR diseases. *Nephrology and Dialysis*. 2022;24(4):577–874. doi: 10.28996/2618-9801-2022-4-577-874 EDN: TSNIXO
15. Simonov PA, Firsov MA, Dunc DA, et al. The role of urological pathology in the development of terminal renal failure. *Consilium Medicum*. 2022;24(10):759–762. doi: 10.26442/20751753.2022.10.201927 EDN: NGTOIS
16. Melnikova LV, Osipova EV. Kidney damage in essential arterial hypertension: pathogenetic issues for early diagnostics. *Arterial Hypertension*. 2019;25(1):6–13. doi: 10.18705/1607-419X-2019-25-1-6-13 EDN: EOOICK
17. Dobronravov VA, Smirnov AV. Etiology and clinic-morphological presentation of membranoproliferative glomerulonephritis in Russian population. *Nephrology (Saint-Petersburg)*. 2018;22(4):9–17. doi: 10.24884/1561-6274-2018-22-4-9-17 EDN: XWBYVF
18. Ismailov IYa, Skvortsov VV Chronic glomerulone-phritis. *The Nurse*. 2018;20(6):17–20. doi: 10.29296/25879979-2018-06-04 EDN: XWPBZZ
19. Firsov MA, Garkusha TA, Simonov PA, et al. Possibilities of intraoperative laparoscopic nephrobiopsy for diagnostics of latent kidney diseases. *Experimental and Clinical Urology*. 2023;16(1):188–194. doi: 10.29188/2222-8543-2023-16-1-188-194 EDN: CLHYOC
20. Bansal N. Prediabetes diagnosis and treatment: A review. *World J Diabetes*. 2015;6(2):296–303. doi: 10.4239/wjd.v6.i2.296

21. Zilov AV. Prediabetes: current state of the problem and clinical guidelines. *Effective pharmacotherapy*. 2022;18(30):20–26. doi: 10.33978/2307-3586-2022-18-30-20-26 EDN: NLGMFA

22. Colvin RB, Chang A. *Diagnostic pathology: Kidney diseases e-book. 3rd edit*. Elsevier Health Sciences; 2019.

23. Chen C, Liu G, Yu X, et al. Association between prediabetes and renal dysfunction from a community-based prospective study. *Int J Med Sci*. 2020;17(11):1515–1521. doi: 10.7150/ijms.46477 EDN: KJQUKC

24. Sursyakova KI, Saf'yanova TV. Several epidemiological peculiarities of incidence of glomerular and tubulointerstitial kidney diseases and urinary tract infections among the population of the Altai Krai. *Experimental and Clinical Urology*. 2017;(4):6–11. EDN: YNJPFJ

AUTHORS' INFO

Mikhail A. Firsov, MD, Cand. Sci. (Medicine);
ORCID: 0000-0002-0887-0081; eLibrary SPIN: 6308-6260;
e-mail: firsma@mail.ru

***Pavel A. Simonov,** MD; address: 1 Partizana Zheleznyaka st., Krasnoyarsk, 660022, Russia; ORCID: 0000-0002-9114-3052; eLibrary SPIN: 4765-6498; e-mail: doctorsimonov@mail.ru

Tatyana A. Garkusha, MD; ORCID: 0000-0002-3343-6973; eLibrary SPIN: 6178-8049; e-mail: sapfiradracula@yandex.ru

Evgenii A. Bezrukov, MD, Dr. Sci. (Medicine), Professor; ORCID: 0000-0002-2746-5962; eLibrary SPIN: 2208-2676; e-mail: eabezrukov@rambler.ru

Dmitrii I. Laletin, MD; ORCID: 0000-0002-1720-075X; eLibrary SPIN: 5702-2065; e-mail: sloth-doc@yandex.ru

Semen E. Nagaev; e-mail: samnagaev@gmail.com

Valeriya D. Moiseeva; e-mail: leramoiseeva814@gmail.com

ОБ АВТОРАХ

Михаил Анатольевич Фирсов, канд. мед. наук;
ORCID: 0000-0002-0887-0081; eLibrary SPIN: 6308-6260;
e-mail: firsma@mail.ru

***Павел Андреевич Симонов;** адрес: Россия, 660022, Красноярск, ул. Партизана Железняка, д. 1; ORCID: 0000-0002-9114-3052; eLibrary SPIN: 4765-6498; e-mail: doctorsimonov@mail.ru

Татьяна Андреевна Гаркуша; ORCID: 0000-0002-3343-6973; eLibrary SPIN: 6178-8049; e-mail: sapfiradracula@yandex.ru

Евгений Алексеевич Безруков, д-р мед. наук, профессор; ORCID: 0000-0002-2746-5962; eLibrary SPIN: 2208-2676; e-mail: eabezrukov@rambler.ru

Дмитрий Иванович Лалетин; ORCID: 0000-0002-1720-075X; eLibrary SPIN: 5702-2065; e-mail: sloth-doc@yandex.ru

Семен Евгеньевич Нагаев; e-mail: samnagaev@gmail.com

Валерия Денисовна Моисеева; e-mail: leramoiseeva814@gmail.com

* Corresponding author / Автор, ответственный за переписку