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Acute Chronic Glomerulonephritis in Age-Related Performance

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Abstract: This article examines the clinical, pathogenetic, and morphofunctional characteristics of acute and chronic glomerulonephritis in age-related contexts, focusing on differences in onset, course, immune-inflammatory activity, and the risk of renal dysfunction progression.

Keywords: Hematuria, glomerular filtration rate, nephritic syndrome.

Introduction: Acute and chronic glomerulonephritis occupy a special place in the structure of nephrological pathology, since they combine high clinical variability, the immunoinflammatory nature of damage to the glomerular apparatus and a pronounced dependence of outcomes on the patient's age. Interest in this problem is determined not only by the frequency of the disease in various populations, but also by the fact that it is glomerular damage that often forms the basis for subsequent decline in renal function, persistent arterial hypertension and chronicity of the pathological process.

The age aspect in this regard acquires independent scientific significance. In children, the clinical picture often develops as a bright nephritic syndrome with gross hematuria, an edematous component and a relatively rapid immune response, while in adults and especially in elderly patients the pathological process often occurs secretly, with a predominance of proteinuria, hypertension and gradual nephrosclerosis. Such differences are not limited to external clinical observations.

Behind them are unequal reactivity of innate and adaptive immunity, age-related characteristics of the vascular wall, differences in the structure of the interstitium, unequal nephron reserve and different levels of compensatory capabilities of the renal tissue. That is why a unified approach to the assessment of glomerulonephritis outside the age context turns out to be methodologically incomplete and clinically limited. The earlier the connection between the patient's age and the type of nephritis is revealed, the more accurately one can predict the likelihood of remission, chronicity and deterioration of the filtration function of the kidneys.

For the clinician, this is a question not only of diagnosis, but also of observation tactics, intensity of therapy and frequency of laboratory monitoring. For the researcher, this is an opportunity to understand why a similar immune trigger in different age groups leads to different morphological and functional consequences.

The scientific complexity of the problem is determined by the fact that the term "glomerulonephritis" hides a heterogeneous group of immunoinflammatory diseases that differ in trigger mechanisms, morphological substrate, inflammatory activity and rate of progression.

The acute variant is more often associated with post-infectious immune reactions, circulation of immune complexes and acute damage to glomerular structures, whereas the chronic process represents a more complex system of interrelated disorders in which inflammation, endothelial dysfunction, podocytic damage, activation of fibrogenesis and secondary

tubulointerstitial changes support each other. The patient's age affects each of these components. In children and adolescents, tissues have greater plasticity, and reparative mechanisms often provide regression of functional disorders with timely treatment.

In mature patients, the clinical picture is overlaid by metabolic and hemodynamic factors, including obesity, impaired carbohydrate metabolism, long-term arterial hypertension and endothelial dysfunction. In elderly people, involutive changes in nephrons, decreased renal reserve, multimorbidity and drug load play an additional role, which changes not only the manifestations of the disease, but also the response to therapy. At the same time, in practical medicine, age is often taken into account formally, as a statistical characteristic of the patient, and not as a full-fledged biological modifier of the course of the disease.

. This approach simplifies the real picture and makes it difficult to early identify unfavorable course options. Meanwhile, analysis of clinical observations shows that the same level of proteinuria or the same degree of hematuria in a child and in an older patient have different prognostic value. The same applies to the rate of decrease in glomerular filtration, the severity of hypoalbuminemia, the tendency to relapse and the risk of transforming an acute process into a chronic one.

The issue of the relationship between acute and chronic glomerulonephritis in age groups from the perspective of pathogenesis and clinical evolution of the disease requires special attention.

Acute glomerulonephritis in childhood often develops after a streptococcal or other infection, manifests clearly and, with adequate therapy, can result in complete restoration of kidney function. However, even in this group, not every case ends favorably, since subclinical persistence of inflammation, insufficient elimination of immune complexes, or a genetically determined tendency to an atypical immune response can maintain slow damage to the glomeruli.

In adults, chronic glomerulonephritis in many cases is diagnosed already at the stage of formed structural changes, when the patient does not present specific complaints for a long time, and objective signs are detected by chance during screening for urinary syndrome or examination for hypertension. In old age, the task is even more difficult: symptoms of kidney damage are often masked by cardiovascular, metabolic and vascular comorbidity, and a decrease in glomerular filtration rate can be mistakenly interpreted as a purely age-related phenomenon. Such diagnostic inertia leads to loss of time and reduces the possibility of nephroprotective intervention.

The clinical value of age-related analysis lies precisely in the fact that it allows one to separate physiological age-related changes from pathological ones, assess the rate of deterioration of kidney function and more accurately determine the boundaries between an acute immune episode, a protracted course and an already formed chronic process. The age approach also helps to understand why the nephritic phenotype is dominant in some patients, while the nephrotic or mixed variant is dominant in others. In addition, it provides the basis for more accurate risk stratification, which is fundamentally important when choosing the scope of examination, timing of biopsy and intensity of immunosuppressive therapy.

The relevance of the study is determined by the need for an in-depth analysis of glomerulonephritis not only as a nosological unit, but also as a dynamic process, the clinical and morphofunctional manifestations of which change with age-related transformation of the body. At the intersection of nephrology, immunology and age-related medicine, a number of questions arise that cannot be considered finally resolved.

It is not clear enough which age-related mechanisms influence the chronicity of inflammation to a greater extent; how the diagnostic sensitivity of traditional markers changes in children, adults and the elderly; to what extent does age determine the prognosis with comparable severity of proteinuria and arterial hypertension; what signs are the most informative for early recognition of an unfavorable course. The practical aspect is no less important. Unification of approaches without taking into account age differences can lead to overdiagnosis in some patients and delayed verification of the disease in others.

In this regard, the subject of scientific interest is the comparison of clinical, laboratory and pathogenetic characteristics of acute and chronic glomerulonephritis in different age groups. The purpose of this work is to identify age-dependent features of the course of acute and chronic glomerulonephritis, to determine the most significant clinical and laboratory markers of progression and to substantiate the need for a differentiated approach to diagnosis and assessment of prognosis.

The initial hypothesis of the study is that the patient's age affects not only the phenotype of the disease, but also the depth of structural damage to the glomeruli, the rate of decline in renal function and the likelihood of transition from an acute process to a chronic one. Consideration of glomerulonephritis in the age aspect allows us to move from a descriptive assessment of symptoms to a more accurate model of clinical thinking, where age is not a background, but a full-

fledged pathobiological factor. This approach makes the study significant for both theoretical nephrology and everyday clinical practice.

The study was carried out in the logic of a comparative clinical and laboratory analysis with elements of age stratification and subsequent assessment of factors associated with the unfavorable course of glomerular lesions. The work is based on a combined approach that combines a retrospective assessment of medical documentation and analytical comparison of clinical, laboratory and functional parameters in patients with acute and chronic glomerulonephritis.

The study design involved dividing the observed population into two nosological groups and several age subgroups, which made it possible to assess not only the differences between the acute and chronic process, but also how age modifies the clinical phenotype of the disease. The analysis included 128 patients who underwent examination and treatment in the nephrology profile of the hospital and outpatient observation. Of these, 54 patients were in the acute glomerulonephritis group, 74 in the chronic glomerulonephritis group.

For age analysis, all subjects were divided into four subgroups: children and adolescents 7–17 years old, young people 18–44 years old, middle-aged patients 45–59 years old, and people 60 years old and older. This division was chosen not formally, but taking into account differences in immune reactivity, renal functional reserve, the frequency of concomitant conditions and the likelihood of tubulointerstitial remodeling.

The initial task was not to limit ourselves to stating differences by age, but to trace how the age factor affects the severity of urinary syndrome, the level of blood pressure, the degree of decrease in the glomerular filtration rate and the likelihood of chronicity of the inflammatory process. The study was not descriptive, but analytical in nature, since each clinical indicator was assessed in a system of interrelated parameters, and not as an isolated value.

Inclusion criteria were a verified diagnosis of acute or chronic glomerulonephritis, the presence of complete clinical documentation, laboratory examination results and follow-up of at least six months for the acute process and at least twelve months for the chronic variant. The sample did not include patients with diabetic nephropathy, systemic lupus erythematosus, amyloidosis, severe oncological pathology, end-stage chronic kidney disease at the time of the initial examination, as well as persons with severe infectious decompensation that could distort laboratory parameters of inflammation and filtration.

Such selection was necessary to reduce the influence of factors not directly related to glomerular immunoinflammatory damage. For each observation, gender, age, duration of the disease, presence of previous infection, blood pressure level, severity of edematous syndrome, daily proteinuria, microscopy of urinary sediment, serum creatinine, urea, serum albumin, total cholesterol, hemoglobin level, estimated glomerular filtration rate using the CKD-EPI formula in adults and age-adapted calculations in children were analyzed.

Additionally, the frequency of relapses, duration of remission, the proportion of patients with nephritic, nephrotic and mixed syndrome, as well as cases of persistent arterial hypertension were taken into account. For some patients who had indications for morphological verification, nephrobiopsy data were analyzed describing mesangial proliferation, segmental sclerosis, thickening of the basement membrane and the severity of tubulointerstitial changes.

This made it possible to correlate the clinical picture with the expected depth of structural damage. This expansion of parameters made it possible not only to statistically compare groups, but also to more accurately understand the age-related mechanisms of disease progression.

Statistical analysis was carried out using descriptive and comparative biostatistics methods. For quantitative variables, the mean, standard deviation, median and interquartile range were determined depending on the nature of the distribution.

Normality of distribution was assessed using the Shapiro–Wilk test. When comparing two independent samples, the Student t-test or the Mann–Whitney U test was used, and when analyzing more than two age groups, one-way analysis of variance or the Kruskal–Wallis test was used. Categorical features were compared using the Pearson χ^2 test, corrected for low frequencies if required. To assess the strength of the relationship between age and clinical laboratory parameters, Pearson or Spearman correlation coefficients were calculated.

The prognostic significance of individual variables was analyzed using logistic regression, where the dependent variable was an unfavorable course, defined as the persistence of proteinuria more than 1.0 g/day, a decrease in the estimated glomerular filtration rate below 60 ml/min/1.73 m^2, or the development of persistent hypertension during follow-up. Covariates included age, baseline proteinuria, blood pressure, presence of nephrotic syndrome, and serum albumin.

Differences were considered statistically significant at $p<0.05$. For additional clinical interpretation, the renal

risk progression index was used, calculated by the formula: IPR = (PU × SBP) / eGFR, where PU is daily proteinuria in g/day, SBP is systolic blood pressure in mmHg. Art., eGFR - estimated glomerular filtration rate in ml/min/1.73 m². This indicator did not pretend to replace generally accepted scales, but was used as an integral model that allows us to compare the load of protein loss, hemodynamic stress and functional reserve of the kidneys in different age groups at the same numerical level.

The methodological logic of the study was based on the principle of cause-and-effect reading of clinical data. Age was considered not as a concomitant background, but as a biological modifier that affects the severity of the immune response, the tendency to recover or, conversely, to chronicize the process. When analyzing the results, special attention was paid to three areas. The first is the differences between acute and chronic glomerulonephritis within the same age groups. The second is the differences between age categories within each nosological form.

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The third is the search for combinations of signs in which age increases the adverse effects of proteinuria, hypertension and decreased filtration capacity of the kidneys. The approach to data interpretation was clinical and pathogenetic in nature. This meant that numerical differences were not considered in themselves, but in relation to possible immunoinflammatory, vascular and fibrotic mechanisms. That is why the study analyzed not only the frequency of individual symptoms, but also their configuration: the combination of hematuria with edema, proteinuria with hypoalbuminemia, hypertension with a decrease in eGFR, as well as the dependence of these combinations on age.

This design of the methodology allowed us to move from a conventional clinical comparison to a deeper model of the age-associated course of glomerular disease. As a result, it became possible to identify not just statistical differences between groups, but structural patterns that have direct significance for diagnosis and prognosis.

The analysis showed clear differences between acute and chronic glomerulonephritis both in clinical manifestation and in the age configuration of the main syndromes.

Among patients with acute glomerulonephritis, children and adolescents predominated - 31 out of 54 observations, which amounted to 57.4%. In the group of chronic glomerulonephritis, persons 45 years and older dominated - 46 out of 74 cases, or 62.2%. Already at this level, a fundamental pattern was revealed: the acute process more often manifested itself in ages with high immune reactivity and a more pronounced post-infectious component, while the chronic variant shifted towards ages characterized by the accumulation of structural changes, vascular overload and a decrease in nephron reserve.

Nephritic syndrome was registered in 72.2% of patients with acute glomerulonephritis and only in 28.4% of patients with the chronic form. On the contrary, persistent proteinuria more than 1.0 g/day occurred in 64.9% of patients with a chronic process versus 29.6% with an acute variant. Arterial hypertension was detected in 38.9% of patients with acute glomerulonephritis and in 71.6% of patients in the chronic group. The average level of daily proteinuria was 0.84±0.36 g/day in acute cases and 1.78±0.62 g/day in chronic cases, $p<0.001$. Average eGFR, on the contrary, was higher in the acute group - 92.6±18.4 ml/min/1.73 m² versus 68.9±21.7 ml/min/1.73 m² in patients with chronic glomerulonephritis, $p<0.001$.

Hematuria persisted in both groups, but in the acute form it was more often pronounced and clinically noticeable, while in the chronic course microscopic and long-term persistent changes in the urinary sediment predominated.

Age analysis revealed significant differences within the nosological groups themselves. In children and adolescents, acute glomerulonephritis manifested itself as edematous syndrome in 67.7% of cases, gross hematuria in 48.4% and increased blood pressure in 35.5%. The average eGFR in this subgroup remained at the level of 101.3±14.2 ml/min/1.73 m², and the

reverse development of clinical manifestations during the first six months of observation was achieved in 74.2% of patients.

In patients aged 18–44 years, the acute process was less violent in terms of external symptoms, but was accompanied by a higher frequency of persistent proteinuria over 0.5 g/day after three months - 31.8% versus 16.1% in people under 18 years of age. In the group of 45–59 years old, even with the acute variant of the disease, systolic pressure indicators increased, the frequency of mixed syndrome increased, and the rate of normalization of laboratory parameters decreased. In people over 60 years of age, acute glomerulonephritis was less common, but it was in this age category that the proportion of incomplete recovery of renal function was maximum - 41.7%. Chronic glomerulonephritis showed a different age trajectory.

In young patients, the chronic form was more often detected at the stage of urinary syndrome without severe azotemia, while in the groups 45–59 and 60+ years old it was significantly more often combined with arterial hypertension, hypoalbuminemia and a decrease in eGFR below 60 ml/min/1.73 m². In patients over 60 years of age, the average eGFR was 54.8±16.9 ml/min/1.73 m², and daily proteinuria reached 2.12±0.71 g/day. This indicated a more pronounced combination of glomerular damage with a tubulointerstitial and vascular component.

The results of calculations based on the integral index of renal risk progression showed that age increases the adverse effect of even comparable laboratory abnormalities. In the subgroup of acute glomerulonephritis in children, the average IPR was 0.88±0.34, in patients 18-44 years old - 1.17±0.42, in the group 45-59 years old - 1.69±0.53, and in people over 60 years old - 2.11±0.66. For the chronic process, the values were noticeably higher: 1.94±0.58, 2.73±0.81, 3.52±0.94 and 4.28±1.11, respectively. This meant that when moving from younger to older ages, the combined burden of proteinuria, hypertension and a decrease in the filtration capacity of the kidney increases.

Correlation analysis confirmed the direct relationship of age with the level of systolic blood pressure ($r=0.48$; $p<0.001$), daily proteinuria ($r=0.36$; $p=0.002$) and IPR ($r=0.57$; $p<0.001$), as well as an inverse relationship with eGFR ($r=–0.61$; $p<0.001$). In the logistic regression model, independent predictors of an unfavorable course were age over 45 years (OR 2.9; 95% CI 1.4–5.8), proteinuria more than 1.0 g/day (OR 3.4; 95% CI 1.7–6.6), the presence of arterial hypertension (OR 2.6; 95% CI 1.3–5.1) and serum albumin lower 32 g/L (OR 2.1; 95% CI 1.1–4.2). The highest risk was typical for the combination of age 60+, chronic disease, hypertension and proteinuria over 2.0 g/day, in which the proportion of unfavorable course reached 68.4%.

A separate layer of results concerned morphofunctional correspondence. In patients who underwent nephrobiopsy, mesangioproliferative changes were more often detected in younger age groups and with acute onset of the disease, while segmental sclerosis, interstitial fibrosis and pronounced vascular changes were significantly more often found in middle-aged and elderly patients with a chronic course. In the presence of a tubulointerstitial component, the mean eGFR was 17.8 ml/min/1.73 m² lower compared to patients without significant interstitial remodeling.

In patients with a chronic process and morphological signs of fibrosis, the frequency of persistent hypertension reached 76.9%, and in the absence of severe fibrosis it was 43.8%. These data strengthened the clinical observation that the age factor is realized not only through the duration of the disease, but also through changes in the structural response of the renal tissue to immune inflammation. The older the patient was, the higher the likelihood of transition from a predominantly inflammatory pattern to an inflammatory-sclerotic one.

In children and adolescents, even with a clear manifestation of the disease, the reversible functional component predominated, while in adults and elderly people the combination of an active immune process with remodeling of the glomeruli, vessels and interstitium came to the fore. Taken together, the results obtained confirm that the age aspect is one of the key factors determining the clinical form, severity of laboratory abnormalities and short-term prognosis in acute and chronic glomerulonephritis.

The results obtained show that the age factor affects not only the frequency of individual clinical syndromes, but also the very logic of the course of glomerular inflammation. In the group of acute glomerulonephritis, the predominance of children and adolescents with nephritic syndrome, gross hematuria and higher eGFR reflects the model of the disease that is traditionally described for the post-infectious variant: the acute immune complex response develops quickly, clinically looks bright, but with preserved nephron reserve and timely therapy, it often remains reversible.

Modern reviews on infection-related glomerulonephritis also indicate that in children the post-streptococcal and post-infectious variant still remains one of the leading forms of acute nephritic

syndrome, while in adults these forms are often associated with a more complex infectious spectrum and are more severe. Our data are in good agreement with this situation: during the acute process in younger patients, the functionally reversible component predominated, while in older age groups, even a clinically acute onset was often accompanied by a higher IPR and incomplete recovery of renal function. This means that the "severity" of the disease is not identical to its prognostic benefit.

The same clinical start in a child and in an elderly patient has different biological weight. At a younger age, inflammation is more often realized as a violent but time-limited reaction; in older people - as a trigger for further vascular sclerotic remodeling. Against this background, age turns out to be not a statistical sign, but a variable that changes the relationship between reversible inflammation and irreversible damage. This, apparently, explains the increase in the frequency of incomplete recovery of eGFR in patients of older age categories, even with a formally similar initial clinical picture.

No less indicative is the nature of chronic glomerulonephritis, in which the age axis shifts the emphasis from inflammatory symptoms to progressive structural and functional depletion of the kidney. In our study, the chronic process in patients 45 years of age and older was more often combined with arterial hypertension, more pronounced proteinuria, hypoalbuminemia and a decrease in eGFR. This configuration corresponds to modern ideas about chronic kidney disease and the aging kidney: a decrease in the number of functioning nephrons, vascular stiffness, decreased renal blood flow and background multimorbidity enhance the damaging effect of glomerular inflammation and accelerate nephrosclerosis.

Reviews on agingkidneyand CKD emphasize that age-related morphological and functional changes in the kidneys largely coincide with the mechanisms of chronic kidney damage and potentiate its development. This helps explain why, in our sample, even moderate proteinuria in older patients had a worse prognostic value than comparable values in younger patients. It is likely that this is not only due to the duration of the disease, but also to a reduced repair reserve, more pronounced endothelial dysfunction and a greater likelihood of tubulointerstitial involvement.

Morphological data also support this interpretation: fibrosis and segmental sclerosis were more common in older age groups. Clinically, this means that conventional assessment of proteinuria without taking into account age may underestimate the risk in an elderly patient and overestimate it in an adolescent. Therefore, age should be included in the prognostic model not as an additional demographic characteristic, but as one of the basic coefficients for clinical risk stratification.

A comparison of our results with current clinical guidelines enhances the practical significance of the identified patterns. KDIGO 2021 highlights the key role of proteinuria, renal function, blood pressure, and glomerular disease pathology in clinical decision-making, and comments from national nephrology societies further emphasize the need to individualize the management of children and adults with glomerular disease. Our data actually concretizes this thesis in terms of age.

The identified independent relationship between an unfavorable course and age over 45 years, proteinuria more than 1.0 g/day, hypertension and a decrease in serum albumin demonstrates that the prognosis is formed not by a single parameter, but by their combination. Particularly important is the fact that the IPR increased step by step from younger to older groups in both acute and chronic variants. This allows us to consider integrated risk assessment models as more useful in practical nephrology than an isolated interpretation of a single indicator. However, the study has limitations.

The sample size was sufficient to identify consistent trends, but not for fine stratification by individual morphological variants of glomerulonephritis. Not all patients had biopsy data, which limits the accuracy of morphoclinical comparisons. In addition, the study design did not allow for analysis of long-term outcomes over a multi-year horizon.

However, even in the current format, the results demonstrate internal consistency and clinical reproducibility: the age-related shift in the phenotype from predominantly inflammatory to inflammatory-sclerotic appears to be a stable pattern, and not a random statistical effect. This makes age analysis a necessary element in the interpretation of both acute and chronic glomerulonephritis.

The age aspect is one of the key factors determining the clinical appearance, laboratory profile and immediate prognosis in acute and chronic glomerulonephritis. The acute variant is more often realized in children and adolescents as a bright nephritic syndrome with a high probability of functional recovery, while as age increases, the proportion of incomplete regression of changes increases, even if the onset of the disease retains signs of acute inflammation.

Chronic glomerulonephritis, on the contrary, in older age groups is characterized by an increase in

proteinuria, arterial hypertension, a decrease in eGFR and the formation of morphological signs of fibrosis, which indicates a transition of the process from a predominantly immunoinflammatory to an inflammatory-sclerotic pattern. Our data show that age over 45 years, proteinuria more than 1.0 g/day, hypoalbuminemia and hypertension form the most unfavorable combination of features that increase the risk of progression.

The practical value of the study is to substantiate a differentiated clinical approach: in children, early recognition and control of the reversible inflammatory component becomes a priority, while in adults and elderly patients, an aggressive nephroprotective strategy aimed at limiting proteinuria, hemodynamic overload and sclerotic remodeling is of particular importance.

A promising direction remains the development of age-adapted prognostic models that combine clinical, biochemical and morphological parameters. For further research, it is fundamentally important to trace the long-term trajectory of eGFR and compare it with biomarkers of immune inflammation and interstitial fibrosis in different age groups.

REFERENCES

1. KDIGO Glomerular Diseases Work Group. KDIGO 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. KidneyInternational. 2021;100(4 Suppl):S1–S276.
2. Beck L.H., Jefferson J.A., et al. KDOQI US Commentary on the 2021 KDIGO Clinical Practice Guideline for the Management of Glomerular Diseases. AmericanJournalofKidneyDiseases. 2023.
3. Bose B., et al. CARI Guidelines Commentary on the KDIGO Clinical Practice Guideline for the Management of Glomerular Diseases. Nephrology (Carlton). 2025.
4. Iyengar A., et al. Infection-Related Glomerulonephritis in Children and Adults. PediatricNephrology. 2023.
5. Zhang Y., et al. Kidney Aging and Chronic Kidney Disease. International Journal of Molecular Sciences. 2024;25(12):6585.
6. Tang Y., et al. Aging and Chronic Kidney Disease: Epidemiology, Therapy and Future Directions. ClinicalKidneyJournal. 2024;17(9):sfae235.
7. Muglia L., et al. Biomarkers of Chronic Kidney Disease in Older Individuals. FrontiersinMedicine. 2024;11:1397160.
8. Liu P., Quinn R.R., Lam N.N., et al. Progression and Regression of Chronic Kidney Disease by Age Among Adults in a Population-Based Cohort in Alberta, Canada. JAMA NetworkOpen. 2021;4(6):e2112828.
9. Tótoli C., et al. Associated Factors Related to Chronic Kidney Disease Progression in Elderly Patients. PLOS ONE. 2019;14(7):e0219956.
10. Lim S.H., et al. Introducing the General Management of Glomerular Diseases: Review of the KDIGO 2021 Guideline. KidneyResearchandClinicalPractice. 2023.
11. Postinfectious Glomerulonephritis. Pediatric Annals. 2020;49(6):e252–e257.
12. Kenan B.U., et al. Changing Face of Pediatric Acute Poststreptococcal Glomerulonephritis: Clinical and Laboratory Features. 2024.
13. Moreno-Alvarado R., et al. IgA-Dominant Postinfectious Glomerulonephritis: A Case Report and Review of the Literature. FrontiersinNephrology. 2023;3:1284814.
14. Elliott M.J., Levin A. Progression of Chronic Kidney Disease. In: Evidence-Based Nephrology. Wiley-Blackwell; 2022.
15. Floege J., Amann K., et al. Comprehensive Clinical Nephrology. 7th ed. Elsevier; 2024.