

**DIAGNOSTIC VALUE OF SERUM URIC ACID, CIAS1 AND CD354
IN CHRONIC GLOMERULONEPHRITIS****DIJAGNOSTIČKA VREDNOST SERUMSKE MOKRAĆNE KISELINE, CIAS1 I CD354
KOD HRONIČNOG GLOMERULONEFRITISA**Yuyan Hu¹, Duoshuai Tang¹, Jiaying Sheng¹, Ming Jin², Yueyue Gu², Xiaohui Ren³¹Department of Clinical Laboratory, Shaoxing Central Hospital (Head Hospital of Shaoxing Central Hospital Medical Community), No. 1 Huayu Road, Keqiao District, Shaoxing City 312030, Zhejiang Province, China²Laboratory Department, Guangdong Provincial People's Hospital, No. 106, Zhongshan Second Road, Yuexiu District, Guangzhou City 510080, China³Department of Critical Care Medicine, Shaoxing Central Hospital (Head Hospital of Shaoxing Central Hospital Medical Community), No. 1, Huayu Road, Keqiao District, Shaoxing City 312030, China**Summary**

Background: To examine the levels and significance of soluble triggering receptor expressed on myeloid cells-1 (sTREM-1), Cold-induced autoinflammatory syndrome 1 (CIAS1), and serum uric acid (UA) in individuals with chronic glomerulonephritis (CGN).

Methods: The CGN group consisted of 192 patients admitted to the hospital between January 2023 and August 2025, whereas the control group consisted of 192 volunteers who underwent physical tests at the same hospital during the same period. Based on their glomerular filtration rate (GFR), patients with CGN were classified into three stages: I, II, and III. The serum uric acid, CIAS1, and TREM-1 levels in the two groups and patients with different stages of CGN were compared; Multivariate logistic regression analysis was used to look at the factors influencing the occurrence of CGN, and receiver operating characteristic (ROC) curves were used to look at the diagnostic value of serum uric acid, CIAS1, and TREM-1 for CGN.

Results: In addition to having higher levels of blood urea nitrogen (BUN), blood creatinine (Scr), 24-hour urine protein (24 h Upro), serum uric acid, CIAS1, and TREM-1, the CGN group exhibited a worse glomerular filtration rate (GFR) than the control group ($P < 0.05$). 52 patients were in stage I, 96 were in stage II, and 44 were in stage III. Blood uric acid, CIAS1, and TREM-1 levels were higher

Kratak sadržaj

Uvod: Ispitati koncentracije i značaj solubilnog receptora okidača eksprimiranog na mijeloidnim ćelijama 1 (sTREM-1), proteina povezanog sa hladno indukovanim autoinflamatornim sindromom 1 (CIAS1) i mokraćne kiseline u serumu (UA) kod osoba sa hroničnim glomerulonefritsom (CGN).

Metode: Grupu sa CGN su činila 192 pacijenta hospitalizovana u periodu od januara 2023. do avgusta 2025. godine, dok su kontrolnu grupu činila 192 dobrovoljca podvrgnuta sistematskom pregledu u istoj bolnici tokom istog perioda. Na osnovu brzine glomerularne filtracije (GFR), pacijenti sa CGN razvrstani su u tri stadijuma: I, II i III. Upoređene su koncentracije mokraćne kiseline u serumu, CIAS1 i TREM-1 između dve grupe, kao i među pacijentima u različitim stadijumima CGN. Multivarijantna logistička regresiona analiza primenjena je radi ispitivanja faktora povezanih sa pojavom CGN, dok su krive operativnih karakteristika primaoca (ROC) korišćene za procenu dijagnostičke vrednosti mokraćne kiseline u serumu, CIAS1 i TREM-1 za CGN.

Rezultati: U poređenju sa kontrolnom grupom, grupa sa CGN je imala više koncentracije azota uree u krvi (BUN), kreatinina u serumu (Scr), proteina u 24-časovnom urinu (24 h Upro), mokraćne kiseline u serumu, CIAS1 i TREM-1, kao i nižu GFR ($P < 0,05$). U stadijumu I bila su 52 pacijenta, u stadijumu II njih 96, a u stadijumu III njih

Address for correspondence:

Xiaohui Ren

Department of Critical Care Medicine, Shaoxing Central Hospital (Head Hospital of Shaoxing Central Hospital Medical Community)

No.1, Huayu Road, Keqiao District, Shaoxing City 312030, China

e-mail: m15257596897@163.com

in stage III patients compared to both stage I and stage II patients ($P<0.05$). Still, these substances were higher in stage II patients compared to stage I patients ($P<0.05$). The findings of the correlation study showed a positive association between the stage of CGN and the levels of serum uric acid, CIAS1, and TREM-1 ($r_s=0.281, 0.275$, and 0.658 , respectively; all $P<0.05$). A multivariate logistic regression study revealed that elevated blood uric acid, CIAS1, TREM-1, and BUN levels were risk factors for the development of CGN ($P<0.05$). The ROC curve analysis results showed that the AUCs for the single diagnosis of CGN by serum uric acid, CIAS1, and sTREM-1, and their combination, were $0.809, 0.794, 0.840$, and 0.948 , respectively. The AUC of the combination diagnosis was higher than that of the single diagnosis of serum uric acid, CIAS1, and sTREM-1 ($Z=4.577, 3.966$, and 3.727 , respectively; all $P<0.001$).

Conclusion: The serum uric acid, CIAS1 and TREM-1 levels were elevated in patients with CGN and closely related to the stage of CGN. Moreover, the combination of these three indicators had relatively high diagnostic value for CGN.

Keywords: uric acid, soluble myeloid cell triggering receptor-1, cold-induced autoinflammatory syndrome 1, chronic glomerulonephritis, diagnostic value

Introduction

A primary glomerular illness, chronic glomerulonephritis (CGN) has a wide range of complicated causes, including bacterial or viral infections, severe tiredness, an imbalance in the metabolism of water and electrolytes, and medication toxicity. Typical symptoms include hematuria, proteinuria, and hypertension (1). The development of CGN is gradual and often concealed, and it is often accompanied by varying degrees of decline in renal function and a trend toward deterioration. If appropriate treatment measures are not taken in time, this disease is highly likely to lead to renal failure, which can seriously threaten a patient's life and health (2–3). Therefore, identifying biomarkers that enable early and effective diagnosis of CGN is particularly important. The result of purine metabolism is uric acid. When the blood uric acid level increases, vascular endothelial cells can quickly take up uric acid, causing a temporary increase in its intracellular level and inducing oxidative stress, whereas the overall change in the concentration of blood uric acid depends mainly on renal excretion function. According to studies, aberrant elevations in uric acid levels can accelerate the progression of kidney disorders by promoting glomerular basement membrane thickening and glomerular microvascular wall thickening (4). A crucial inflammatory regulatory factor, Cold-induced autoinflammatory syndrome 1 (CIAS1), can release proinflammatory cytokines and encourage inflammatory cells to gather at inflammatory sites, hastening disease progression. CIAS1 may be impli-

44. Koncentracije mokraćne kiseline u serumu, CIAS1 i TREM-1 bile su više kod pacijenata u stadijumu III nego kod pacijenata u stadijumima I i II ($P<0,05$), dok su kod pacijenata u stadijumu II bile više nego kod pacijenata u stadijumu I ($P<0,05$). Rezultati korelacione analize pokazali su pozitivnu povezanost između stadijuma CGN i koncentracija mokraćne kiseline u serumu, CIAS1 i TREM-1 ($r_s=0,281, 0,275$ i $0,658$; za sve $P<0,05$). Multivarijantna logistička regresiona analiza pokazala je da su povišene koncentracije mokraćne kiseline u serumu, CIAS1, TREM-1 i BUN predstavljale faktore rizika za razvoj CGN ($P<0,05$). Analiza ROC krivih je pokazala da su površine ispod krive (AUC) za pojedinačnu dijagnostičku primenu mokraćne kiseline u serumu, CIAS1 i sTREM-1, kao i za njihovu kombinovanu primenu, iznosile $0,809, 0,794, 0,840$ i $0,948$. AUC kombinovane dijagnostičke primene bio je veći od AUC pojedinačne primene mokraćne kiseline u serumu, CIAS1 i sTREM-1 ($Z=4,577, 3,966$ i $3,727$; za sve $P<0,001$).

Zaključak: Koncentracije mokraćne kiseline u serumu, CIAS1 i TREM-1 bile su povišene kod pacijenata sa CGN i blisko povezane sa stadijumom bolesti. Pored toga, kombinacija ova tri pokazatelja imala je relativno visoku dijagnostičku vrednost za CGN.

Ključne reči: mokraćna kiselina, solubilni receptor okidač eksprimiran na mijeloidnim ćelijama 1, hladno indukovani autoinflamatorni sindrom 1, hronični glomerulonefritis, dijagnostička vrednost

cated in the pathological process of CGN because it is highly expressed in several kidney illnesses and is strongly associated with impaired renal function (5). A member of the immunoglobulin superfamily, soluble myeloid cell triggering receptor-1 (CD354) is mostly expressed on the surface of myeloid cells and possesses immunomodulatory properties. Patients with nephrotic syndrome have a markedly higher level of it (6–8).

It is still unknown how useful CD354, CIAS1, and uric acid are for diagnosing CGN. In order to provide a reference for the clinical diagnosis of CGN, this study examined the blood levels of uric acid, CIAS1, and CD354 in CGN patients.

Materials and Methods

Research subjects and grouping

CGN group: 192 patients with chronic glomerulonephritis (CGN) who were admitted to a certain hospital from January 2023 to August 2025 were selected.

Control group: 192 volunteers who underwent health check-ups at the same hospital during the same period were selected.

CGN staging criteria: Based on the glomerular filtration rate (GFR) level, the CGN patients were divided into three stages: Stage I (52 cases); Stage II (96 cases); Stage III (44 cases).

Specimen collection and processing

Blood collection requirements: All subjects should collect about 5 mL of blood from the elbow vein on the morning after admission (or on the day of the physical examination), while fasting.

After collection, leave the blood at room temperature for 30 minutes, then centrifuge at 3000 r/min for 15 minutes to separate the serum. The serum should be aliquoted and stored in a -80 °C refrigerator for testing, avoiding repeated freezing and thawing.

Urine sample: Collect 24-hour urine, record the total urine volume, mix well, and take 10 mL for submission for testing to detect 24-hour urine protein quantification (24 h Upro).

Renal function indicators and detection of serum CIAS1 and CD354

Five millilitres of fasting venous blood were drawn on the day of the patient's visit to the CGN group and the day of the physical examination for the control group. Following natural coagulation, the sample was centrifuged for 10 minutes at 3,500 r/min (radius 8 cm) to extract the serum, which was then refrigerated at -80 °C for testing. Every participant's 24-hour urine was gathered, and the volume of all the urine was noted. After centrifuging three millilitres of urine for ten minutes at 3,500 r/min (radius 8 cm), the supernatant was kept at a low temperature for analysis.

Serum and urine samples were removed and stored at room temperature. Renal function indicators [serum uric acid, blood urea nitrogen (BUN), serum creatinine (Scr), 24-hour urine protein quantification (24 h Upro), and glomerular filtration rate (GFR)] were detected using a BS-1000 M automatic biochemical analyser (Shenzhen Meirui Biomedical Electronics Co., Ltd.). Serum levels of CIAS1 and CD354 were measured by enzyme-linked immunosorbent assay (ELISA). ELISA kits for CIAS1 and CD354 were purchased from Shanghai Keai-bo Biotechnology Co., Ltd., with the item numbers CB20041-Hu and CB10949-Hu, respectively. First, 50 µL of each sample and standard were put into a 96-well microplate. The mixture was then incubated at 37 °C for 60 minutes after 100 µL of detection antibody labelled with horseradish peroxidase was added. The sample was washed five times, and 50 µL of substrate was added after the solvent was discarded. For fifteen minutes, the samples were stored at 37 °C in the dark. The absorbance of each well was measured at 450 nm using an enzyme analyser after the stop solution was added. CIAS1 and CD354 serum levels were determined using a standard curve.

Stages of chronic glomerulonephritis

According to GFR, CGN patients were divided into stage I [GFR>90 mL/(min·1.73 m²)], stage II [GFR=60–90 mL/(min·1.73 m²)] and stage III [GFR<60 mL/(min·1.73 m²)].

Analysis of statistics

To analyse the data, SPSS 27.0 software was utilised. Measurement data with a normal distribution are expressed as $\bar{x} \pm s$, and the independent-samples t test was used to compare two groups. Count data are expressed as the number of cases and percentage, and the χ^2 test was used to compare multiple groups. The factors influencing the occurrence of CGN were examined using multivariate logistic regression analysis. The diagnostic value of serum uric acid, CIAS1, and CD354 for CGN was examined using receiver operating characteristic (ROC) curves, and the DeLong test was used to compare the area under the curve (AUC). The significance criterion α was set at 0.05, and differences were considered statistically significant if $P < 0.05$.

Results

Baseline information and markers of renal function were compared between the CGN and control groups

The CGN group and the control group did not differ statistically significantly ($P > 0.05$) in terms of sex, age, BMI, history of hypertension, alcohol use, or smoking; however, the CGN group had higher levels of serum uric acid, BUN, Scr, and 24-hour Upro ($P < 0.05$) and a lower glomerular filtration rate (GFR) ($P < 0.05$). Refer to *Table I*.

Comparison of serum CIAS1 and CD354 levels between the CGN group and the control group

Serum CIAS1 and CD354 levels were significantly higher in the CGN group than in the control group ($P < 0.05$). See *Table II*.

Comparison of serum uric acid, CIAS1 and CD354 levels in patients with different stages of CGN

Twenty-six patients had stage I disease, 48 had stage II disease, and 22 had stage III disease. Serum uric acid, CIAS1, and CD354 levels were higher in stage III patients than in stage I and II patients ($P < 0.05$); serum uric acid, CIAS1, and CD354 levels were higher in stage II patients than in stage I patients ($P < 0.05$); see *Table III*.

Table I Comparison of baseline data and renal function indicators between the CGN Group and the control group [n(%), or $\bar{x}\pm s$].

Group	N	Gender		Age (years)	BMI (kg/m ²)	Hypertension		Drinking history	
		Male	Female			Yes	No	Yes	No
CGN group	192	126 (65.62)	66 (34.38)	45.64±9.24	23.15±2.36	62 (32.29)	130 (67.71)	40 (20.83)	152 (79.17)
Control group	192	120 (62.50)	72 (37.50)	46.32±9.56	23.54±2.48	46 (23.96)	146 (76.04)	36 (18.75)	156 (81.25)
χ^2/t		0.207		-0.570	-1.133	1.642		0.134	
P		0.655		0.568	0.263	0.192		0.710	
Group	N	Smoking history		BUN (mmol/L)	24h Upro (mmol/L)	Scr (mg/24h)	GFR [mL/ (min ·1.73m ²)]	Uric acid (mmol/L)	
		Yes	No						
CGN group	192	68 (35.42)	124 (64.58)	4.60±2.31	129.06±13.33	316.46±37.65	82.97±10.60	346.35±38.89	
Control group	192	60 (31.25)	132 (68.75)	5.91±1.24	83.15±8.59	120.16±15.15	98.80±2.88	304.64±33.77	
χ^2/t		0.378		31.823	28.443	47.430	-9.348	7.944	
P		0.543		<0.001	<0.001	<0.001	<0.001	<0.001	

Table II Serum CIAS1 and CD354 levels were compared between the CGN and control group ($\bar{x}\pm s$).

Group	n	CIAS1 (pg/mL)	CD354 (ng/L)
CGN group	192	272.46±31.35	27.15±4.88
Control group	192	238.24±26.77	21.718±4.00
T		8.145	8.267
P		<0.001	<0.001

Correlation analysis of serum uric acid, CIAS1, and CD354 levels with the stage of CGN patients

Serum uric acid, CIAS1, and CD354 levels were favourably connected with the stage of CGN patients, according to the correlation analysis results ($r_s=0.282, 0.275, \text{ and } 0.658$, respectively; all $P<0.05$).

Table III Comparison of CD354, CIAS1, and serum uric acid levels in CGN patients at various phases ($\bar{x}\pm s$).

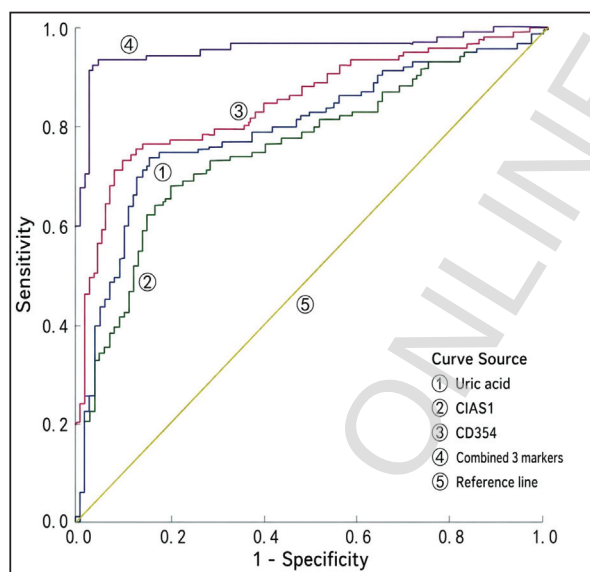
Instalment	n	Uric acid (mmol/L)	CIAS1 (pg/mL)	CD354 (ng/L)
Phase I	52	330.54±36.24	259.57±29.67	21.28±4.09
Phase II	96	347.11±38.35	273.43±31.30	27.34±4.67
Phase III	44	363.15±40.44	285.59±32.24	33.67±5.35
F		4.357	4.218	42.220
P		0.019	0.011	<0.001

Table IV Factors influencing CGN occurrence analysed by multiple logistic regression analysis.

Factor	β	SE	Wald χ^2	P	OR	OR 95%CI
Uric acid	0.980	0.298	11.204	0.001	2.686	1.508~4.788
CIAS1	0.756	0.279	7.442	0.006	2.126	1.230~3.641
CD354	0.631	0.224	8.328	0.004	1.896	1.220~2.911
BUN	1.161	0.191	34.809	<0.001	3.219	2.185~4.744

Table V Diagnostic efficacy of serum uric acid, CIAS1, CD354 alone and in combination for CGN.

Indicator	Sensitivity (%)	Specificity (%)	Optimal truncation value	AUC	AUC 95%CI	Youden index	P
Uric acid	73.99	84.30	325.19 mmol/L	0.809	0.745~0.862	0.586	<0.05
CIAS1	77.01	85.45	260.97 pg/mL	0.794	0.727~0.850	0.628	<0.05
CD354	75.03	89.51	24.46 ng/L	0.840	0.793~0.906	0.649	<0.05
3 Joint Projects	93.78	95.86	-	0.948	0.908~0.988	0.899	<0.05

**Figure 1** ROC curve of serum uric acid, CIAS1 and CD354 for the diagnosis of CGN.

of CGN; thus, they were not included as independent variables for analysis. Elevated serum uric acid, CIAS1, CD354, and BUN levels were risk factors for the occurrence of CGN ($P < 0.05$), see Table IV.

Diagnostic value of serum uric acid, CIAS1, and CD354 for CGN

A receiver operating characteristic (ROC) curve was created by using serum uric acid, CIAS1, and CD354 levels as the independent variables and whether CGN occurred (yes = 1, no = 0) as the dependent variable. The findings showed that the AUCs for the individual diagnosis of CGN by serum uric acid, CIAS1, and CD354 were 0.809, 0.794, and 0.840, respectively; the AUC for the three diagnoses taken together was 0.945. Serum uric acid, CIAS1, and CD354 single diagnoses had lower AUCs than the combination diagnosis ($Z = 4.577$, 3.966, and 3.727, respectively; all $P < 0.001$); refer to Figure 1 and Table V.

Investigation of the factors influencing the prevalence of CGN using multivariate logistic regression

To determine whether CGN occurred (no = 0, yes = 1) as the dependent variable and whether uric acid, CIAS1, CD354, and BUN were independent variables (all inputs as original values), a multivariate logistic regression analysis was conducted. Scr, 24 h Upro, and the GFR are diagnostic indicators

Discussion

In clinical practice, CGN is a prevalent chronic kidney disease. In recent years, owing to lifestyle changes and increased work pressure, its incidence has shown a year-on-year upward trend, especially in the middle-aged and young male population (9). Early screening and prompt care can slow disease progression and lower the risk of developing end-stage renal disease, according to studies (10). How-

ever, since the onset of CGN is relatively insidious, most patients already experience varying degrees of renal function impairment at the time of diagnosis, posing severe challenges for clinical treatment and patient prognosis (11). Currently, the main diagnostic method for CGN is routine urine testing, which assesses the degree of renal function impairment by detecting relevant clinical indicators (BUN, Scr, 24 h Upro, and GFR). This study revealed significant differences in renal function indicators between the CGN group and the control group, which is similar to previous findings (12). Although these indicators can provide certain reference values for clinical diagnosis, in the early stage of the disease, their specificity, sensitivity, and accuracy are lacking, making accurate diagnosis of CGN difficult and affecting patient treatment and prognosis (13). Therefore, exploring reliable and efficient biomarkers to achieve early and accurate diagnosis of CGN is highly important for improving patient prognosis.

Uric acid is not only an indicator of renal function but also among the abundant antioxidants in the human body. It plays a crucial role in maintaining blood pressure stability and protecting the nervous system (14). However, other research has demonstrated that excessively high uric acid levels lead to the accumulation of crystalline materials in tissues and activate the CIAS1 inflammasome, which in turn contributes to the onset and progression of several diseases, including kidney, liver, and cardiovascular disorders (15). Patients with diabetic nephropathy have greater serum uric acid levels than those with uncomplicated diabetes. CGN patients have significantly higher serum uric acid levels than healthy individuals, which is a risk factor for ineffective diabetic nephropathy treatment (16–17). Serum uric acid levels are much higher in CGN patients than in healthy individuals, and they are higher in patients with poor prognoses than in those with good prognoses. These findings imply that the development and occurrence of CGN may be related to serum uric acid (18). In line with the aforementioned findings, the study's findings showed that the CGN group's serum uric acid level was considerably higher than the control group's. Additionally, this study showed that serum uric acid levels steadily rise as the disease advances, which is positively connected with the stage of CGN patients and influences the occurrence of CGN. These findings further confirm that serum uric acid may play an important role in the occurrence and development of CGN.

CIAS1 is a pattern recognition receptor that was discovered earlier. It can create the CIAS1 inflammasome and trigger caspase-1 to regulate the production and release of inflammatory proteins, such as IL-1 β /IL-18, after recognising danger sig-

nals. It is intimately linked to the onset and development of certain kidney disorders. It plays significant regulatory functions in the occurrence and course of inflammatory reactions (19). CIAS1 levels were markedly elevated in animals with diabetic kidney disease in a mouse model of the condition. The body's inflammatory response and damage can be lessened by blocking the CIAS1 pathway, which also lessens the severity of kidney tissue damage and the quantity of fibrotic lesions (20–22). TREM-1 is a single transmembrane receptor protein located in the 21.1 region of human chromosome 6. It is a key trigger of the inflammatory response and directly participates in and exacerbates the inflammatory process (23). Current research has revealed that TREM-1 plays important roles in various inflammatory diseases, such as acute lung injury and sepsis. The soluble form of TREM-1 is called CD354. TREM-1 is released into the bloodstream as CD354 during the initial phase of the inflammatory reaction (24–26).

This study further explored the diagnostic efficacy of serum uric acid, CIAS1, and CD354 for CGN. The AUC values were 0.806, 0.791, 0.847, and 0.945 for the individual diagnosis of CGN using serum uric acid, CIAS1, and CD354 levels, as well as for the combination diagnosis of the three indications. The combined diagnosis of the three signs had a higher AUC than the individual diagnoses, indicating that it might be a helpful additional method for CGN diagnosis. In particular, when the serum uric acid, CIAS1, and CD354 concentrations exceeded the optimal cutoff values, that is, when serum uric acid >325.16 μ mol/L, CIAS1>260.94 pg/mL, and CD354>24.43 ng/L occurred simultaneously, this may indicate the occurrence of CGN.

Conclusion

Serum uric acid, CIAS1 and CD354 levels were elevated in patients with CGN and were related to the stage of CGN. Moreover, the combination of these three indicators was highly valuable for the diagnosis of CGN. It could be used as an auxiliary method for the clinical diagnosis of CGN.

Funding

None.

Conflict of interest statement

All the authors declare that they have no conflict of interest in this work.

References

1. Singh NA, Sen S. Molecular insights into chronic glomerulonephritis and dopamine interplay. *Chem Biol Interact* 2026 Jan 25; 424: 111880. doi: 10.1016/j.cbi.2025.111880. Epub 2025 Dec 12. PMID: 41390133.
2. Hanna RM, Ahdoot RS, Rhee CM, Tantisattamo E, Al Ammar F, Kalantar-Zadeh K. New and old approaches to nutritional management of acute and chronic glomerulonephritis. *Curr Opin Nephrol Hypertens* 2023 Jan 1; 32(1): 76–80. doi: 10.1097/MNH.0000000000000855. Epub 2022 Nov 21. PMID: 36444665.
3. Xu C, Fan J. Gene variant C3 glomerulonephritis with chronic urinary tract infection: A case report and literature review. *Medicine (Baltimore)* 2024 Dec 27; 103(52): e41001. doi: 10.1097/MD.00000000000041001. PMID: 39969315; PMCID: PMC11688077.
4. Wang X, Liu Z, Yi N, Li L, Ma L, Yuan L, Wang X. The global burden of chronic kidney disease due to glomerulonephritis: trends and predictions. *Int Urol Nephrol* 2025 Aug; 57(8): 2613–24. doi: 10.1007/s11255-025-04440-2. Epub 2025 Mar 5. PMID: 40045051; PMCID: PMC12259738.
5. Utari IALA, Adhi S, Hermawan K, Arguni E. Predictive factors of progression to chronic glomerulonephritis in pediatric patients with post streptococcal acute glomerulonephritis. *Pediatr Neonatol* 2024 Nov; 65(6): 571–5. doi: 10.1016/j.pedneo.2023.11.005. Epub 2024 Apr 16. PMID: 38649317.
6. Hattori T, Fujioka K, Nagai T, Kondo S, Kagami S, Hirayama M, Urushihara M. Intrarenal renin-angiotensin system activation and macrophage infiltrations in pediatric chronic glomerulonephritis. *Pediatr Nephrol* 2023 Nov; 38(11): 3711–9. doi: 10.1007/s00467-023-06026-5. Epub 2023 May 25. PMID: 37231123; PMCID: PMC10514104.
7. Gao YC, Jiang NN, Qin XJ, Jiang H, Wei LB, Gao JR. High-throughput data on circular RNA reveal novel insights into chronic glomerulonephritis. *Genes Genomics* 2023 Apr; 45(4): 475–90. doi: 10.1007/s13258-022-01320-2. Epub 2022 Oct 20. PMID: 36264417.
8. Gao W, Zhou Y, Li C, Liu T, Zhao H, Wang M, Wei X, Wang H, Yang J, Si N, Liang A, Bian B, Sato T. Studies on the metabolism and mechanism of acteoside in treating chronic glomerulonephritis. *J Ethnopharmacol* 2023 Feb 10; 302(Pt A): 115866. doi: 10.1016/j.jep.2022.115866. Epub 2022 Nov 1. PMID: 36332760.
9. Fan JH, Li XM. Mesangial cell-derived CircRNAs in chronic glomerulonephritis: RNA sequencing and bioinformatics analysis. *Ren Fail* 2024 Dec; 46(2): 2371059. doi: 10.1080/0886022X.2024.2371059. Epub 2024 Jul 1. PMID: 38946402; PMCID: PMC467094.
10. Atlas I, Wong C, Downe K, Vazquez M, Ruderman I. Querying ANCA-associated vasculitis in chronic Q fever-associated membranoproliferative glomerulonephritis. *Intern Med J* 2024 Apr; 54(4): 691–2. doi: 10.1111/imj.16376. PMID: 38637282.
11. Guo R, Shang JH, Ye RH, Zhao YL, Luo XD. Pharmacological investigation of indole alkaloids from *Alstonia scholaris* against chronic glomerulonephritis. *Phytomedicine* 2023 Sep; 118: 154958. doi: 10.1016/j.phymed.2023.154958. Epub 2023 Jul 8. PMID: 37453192.
12. Zhang H, Li XL, Lu JY, Ke Q, Li J, Du Y, Yu YF. Renal expression and clinical significance of Bach1-related oxidative stress indicators in patients with chronic glomerulonephritis. *Pathol Res Pract* 2025 May; 269: 155935. doi: 10.1016/j.prp.2025.155935. Epub 2025 Mar 29. PMID: 40186888.
13. Zhuang XX, Zheng WJ, Liu T, Tang YY, Guo MF, Ni SD, Gao JR. Qiteng Xiaozhuo Granules improve chronic glomerulonephritis through promoting glomerular mesangial cell autophagy via FTO-mediated SLC7A5 m6A modification. *J Ethnopharmacol* 2026 Feb 28; 357: 120948. doi: 10.1016/j.jep.2025.120948. Epub 2025 Nov 25. PMID: 41308711.
14. Markina MV, Milovanova LY, Lysenko LV, Milovanova SY, Volkov AV, Beketov VD, Lebedeva MV, Nezhdanov KS, Moiseev SV. Anemia of chronic diseases in the early stages of chronic kidney disease as a risk factor for cardiovascular complications in patients with glomerulonephritis. *Ter Arkh* 2024 Jul 7; 96(6): 606–13. Russian. doi: 10.26442/00403660.2024.06.202729. PMID: 39106502.
15. Idasiak-Piechocka I, Miedziaszczyk M, Woźniak A, Pawliczak E, Kaczmarek E, Oko A. Interleukin-6 and epidermal growth factor as noninvasive biomarkers of progression in chronic glomerulonephritis. *Am J Physiol Cell Physiol* 2023 Nov 1; 325(5): C1267–C75. doi: 10.1152/ajpcell.00058.2023. Epub 2023 Sep 18. PMID: 37721004.
16. Liu T, Zhuang XX, Qin XJ, Wei LB, Gao JR. The potential role of N6-methyladenosine modification of lncRNAs in contributing to the pathogenesis of chronic glomerulonephritis. *Inflamm Res* 2023 Mar; 72(3): 623–38. doi: 10.1007/s00011-023-01695-2. Epub 2023 Jan 26. PMID: 36700958.
17. Zhang K, Kan C, Han F, Fan Q, Wang Y, Li X, Pan R, Guo Z, Hou N, Sun X. Global Trends and Projections in Chronic Kidney Disease Burden from Diabetes, Hypertension, and Glomerulonephritis: A Population-Based Study. *Kidney Blood Press Res* 2026; 51(1): 72–90. doi: 10.1159/000550353. Epub 2026 Jan 8. PMID: 41505356; PMCID: PMC12890268.
18. Wang B, Kim K, Tian M, Kameishi S, Zhuang L, Okano T, Huang Y. Engineered Bone Marrow Stem Cell-Sheets Alleviate Renal Damage in a Rat Chronic Glomerulonephritis Model. *Int J Mol Sci* 2023 Feb 13; 24(4): 3711. doi: 10.3390/ijms24043711. PMID: 36835123; PMCID: PMC9959772.
19. Cai X, Li Y, Liu Q, Gao X, Li J. Exploration of the Shared Gene Signatures and Molecular Mechanisms

- between Chronic Bronchitis and Antineutrophil Cytoplasmic Antibody-associated Glomerulonephritis: Evidence from Transcriptome Data. *Curr Pharm Des* 2024; 30(25): 1966–84. doi: 10.2174/0113816128297623240521070426. PMID: 38847168.
20. Su H, Wu H, Wu S, Zhou M. Effects of electroacupuncture at KI3 and ST36 on the hypothalamic paraventricular nucleus in a rat model of chronic glomerulonephritis. *Acupunct Med* 2023 Oct; 41(5): 307–16. doi: 10.1177/09645284231166718. Epub 2023 May 11. PMID: 37166069.
 21. Sun L, Deng X, Yang B, Ren Q, Wang C, Yuan R, Liu S, Feng X, Liu X. Bioinformatics-based exploration of the role of copper metabolism in chronic glomerulonephritis. *Medicine (Baltimore)* 2025 Nov 7; 104(45): e45540. doi: 10.1097/MD.00000000000045540. PMID: 41204597; PMCID: PMC12599697.
 22. Nakai K, Sato K, Nohara N, Takagi M, Kihara M, Ueda S, Gohda T, Suzuki Y. Successful Pregnancy and Delivery in a Chronic Renal Failure Patient with Membranoproliferative Glomerulonephritis and Preeclampsia-related Nephrotic Syndrome. *Intern Med* 2024 Jul 15; 63(14): 2035–42. doi: 10.2169/internalmedicine.1972-23. Epub 2023 Nov 27. PMID: 38008458; PMCID: PMC11309875.
 23. Alekseeva MV, Kozlovskaya NL, Korotchaeva YV, Demyanova KA, Chegodaeva AG, Apresyan SV. Nephrotic syndrome during pregnancy. Is it chronic glomerulonephritis or preeclampsia? Case report. *Ter Arkh* 2023 Aug 17; 95(6): 500–4. Russian. doi: 10.26442/00403660.2023.06.202264. PMID: 38158970.
 24. Qin X, Chen H, Zheng W, Hu W, Xu X, Gao J. The role of METTL3-mediated CircStk4 modification in the treatment of chronic glomerulonephritis with Qi Teng Xiao Zhuo granule. *Phytomedicine* 2024 Dec; 135: 156183. doi: 10.1016/j.phymed.2024.156183. Epub 2024 Oct 26. PMID: 39488875.
 25. Wang M, Zhou Y, Jian Q, Ai Z, Zhou S. Mechanisms of *Rostellaria procumbens* (L.) Nees on treating chronic glomerulonephritis explored by network pharmacology, RNA-seq, and in vitro experiments. *BMC Complement Med Ther* 2023 Jul 24; 23(1): 263. doi: 10.1186/s12906-023-04079-5. PMID: 37488573; PMCID: PMC10367255.
 26. Qin X, Chen H, Zhu X, Xu X, Gao J. Identification of Rab7 as an autophagy marker: potential therapeutic approaches and the effect of Qi Teng Xiao Zhuo granule in chronic glomerulonephritis. *Pharm Biol* 2023 Dec; 61(1): 1120–34. doi: 10.1080/13880209.2023.2233998. PMID: 37477949; PMCID: PMC10364565.

Received: June 12, 2026

Accepted: July 31, 2026