ORIGINAL ARTICLE

Long-Term Outcomes of IgA Nephropathy with and without Immunosuppressive Therapy: A Retrospective Cohort Study from a Tertiary Care Hospital in Balochistan

Zarafshan Khan[1*], Ashfaq Altaf[1], Nadeem Fazal[2], Nouman Kashif[1]

ABSTRACT

Objective: To evaluate the long-term outcomes and prognostic factors associated with IgA nephropathy (IgAN) in patients treated with and without immunosuppressive drugs.

Study Design: A retrospective cohort study.

Place and Duration of Study: The study was conducted at the Department of Nephrology, Combined Military Hospital (CMH), Quetta, Pakistan, from May 2023 to May 2024.

Methods: A total of 70 patients diagnosed with biopsy-proven IgAN were included in the study. The patients were divided into two groups: 43 received immunosuppressive drug treatment (IS), and 27 did not (No IS). Patient records were analyzed for clinical, histological, and treatment data. Cox proportional hazards regression was used to assess factors associated with end-stage kidney disease (ESKD), kidney replacement therapy (KRT), and mortality.

Results: The median age of the patients was 32 years, predominantly male (61.4%). Patients in the immunosuppressive drug treatment group had significantly higher systolic blood pressure (*P*=0.005) and more severe proteinuria (*P*=0.002). The immunosuppressive drug treatment group showed a greater degree of renal damage, with increased glomerulosclerosis and tubulointerstitial injury, than the non-immunosuppressive drug treatment group. Severe proteinuria, reduced eGFR, and extensive tubulointerstitial damage, as determined by Cox regression analysis, independently predict ESKD and mortality. Patients with extensive tubulointerstitial damage (>50%) and an eGFR below 30 mL/min/1.73 m² were at heightened risk for adverse outcomes. At diagnosis, 35.2% of patients had an eGFR <60 mL/min/1.73 m². Over a median follow-up of 6.3 years, 13 patients (25.4%) progressed to ESRD, and 5 patients (9.8%) died. Complete remission was achieved in 13.7% of patients, partial remission in 39.2%, while 21.5% showed no response to treatment.

Conclusion: Immunosuppressive therapy was associated with more severe renal pathology in IgAN patients. Reduced eGFR, severe proteinuria, and significant histological damage were key predictors of poor renal outcomes.

Keywords: *Glomerulonephritis, IgA Nephropathy, Immunosuppressive Agents, Proteinuria.*

How to cite this: *Khan Z, Altaf A, Fazal N, Kashif N. Long-Term Outcomes of IgA Nephropathy with and without Immunosuppressive Therapy: A Retrospective Cohort Study from a Tertiary Care Hospital in Balochistan. Life and Science. 2026; 7(1): 3-9. doi: http://doi.org/10.37185/LnS.1.1.840*

[1]*Department of Nephrology*
Quetta Institute of Medical Sciences (QIMS),
Combined Military Hospital, Quetta, Pakistan
[2]*Department of Medicine*
Pak Emirates Military Hospital (PEMH), Rawalpindi, Pakistan
Correspondence:
Dr. Zarafshan Khan
Department of Nephrology
Quetta Institute of Medical Sciences (QIMS),
Combined Military Hospital, Quetta, Pakistan
E-mail: drzarafshankhan@gmail.com
Received: Feb 14, 2025; 1st Revision Received: July 04, 2025
2nd Revision Received: Nov 16, 2025; Accepted: Nov 27, 2025

Introduction

IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide, characterized by IgA deposition in the mesangial areas of the glomeruli. This leads to progressive deterioration of kidney function and is a major contributor to chronic kidney disease (CKD) and end-stage renal disease (ESRD).[1] Epidemiological data shows approximately 30–40% of IgAN patients may develop ESRD within a decade of diagnosis and require long-term follow-up.[2,3] There is wide clinical variability in the natural history

of IgAN, ranging from mild asymptomatic cases to a significant subset with rapid progression to renal failure, with key factors like proteinuria, hypertension, and histopathological findings influencing disease progression.[3,4]

In regions with limited healthcare resources, such as Balochistan, Pakistan early diagnosis and tailored treatment are essential for improving patient outcomes, especially in younger patients where most diagnoses are made before age 40.[2] Discussing IgAN in isolation as a distinct disease entity fails to account for its complex overlap with other renal pathologies, such as membranous nephropathy (MN), which may contribute to more severe kidney damage if not properly recognized.

Previous studies have identified several clinical and histological factors influencing IgAN outcomes. For example, a long-term study involving 106 patients with IgA vasculitis-related nephritis from Huashan Hospital, Fudan University, China identified hypertension and glomerular sclerosis as predictors of poor renal outcomes.[5] Another study involving 2724 biopsy-confirmed IgAN patients at Xijing Hospital, affiliated with the Fourth Military Medical University, China, examined long-term outcomes and treatment responses through a comprehensive analysis of clinical data. The results indicated that immunosuppressive treatments play a crucial role in improving renal survival in these patients.[4] The histological severity of IgAN is often assessed using the Oxford classification system. Moreover, the clinical grade (or risk grade) is evaluated according to the Japanese Society of Nephrology's criteria, which may or may not include histological data.[6,7] These frameworks provide a foundation for understanding disease progression. However, there remains a significant knowledge gap regarding long-term outcomes in patients with mild proteinuria, particularly in resource-limited regions like Balochistan, where sociodemographic and environmental factors drastically differ.

This study aims to address this gap by evaluating the long-term outcomes of patients with IgAN treated at a tertiary care hospital in Balochistan. Utilizing analysis of clinical and histological parameters and long-term follow-up data, the study seeks to identify prognostic factors affecting renal survival and disease progression, potentially revealing regional patterns of IgAN that differ from those in other populations.

Methods

To determine outcomes in patients with IgA nephropathy, a retrospective cohort study was conducted. The study period was from May 2023 to May 2024. The study protocol was approved by the Institutional Ethical Review Committee of the hospital vide letter no: CMH/QTA-IERB/15/2023, dated: 12th December 2023, ensuring compliance with ethical principles. Informed consent was waived because of the retrospective nature. Confidentiality was maintained for patient identification and data collection as well as during the analysis. This study included 70 cases of IgAN, 43 male and 27 female.

Included patients had to meet the following criteria: biopsy-proven IgAN**,** age ≥ 12 years, and availability of clinical, histological, and treatment data.

Patient medical records were analyzed for data regarding demographics, clinical presentation, diagnostic tests, specifically renal biopsy-proven IgA nephropathy, histological findings, and therapeutic details. Results were collected from the required national registries, including ESKD/KRT and all-cause mortality.

Among the clinical parameters measured in this study were age, blood pressure at the baseline visit, sex, and levels of serum creatinine and proteinuria. Histological parameters were assessed in kidney biopsy samples: tubular atrophy/interstitial fibrosis, mesangial hypercellularity, segmental glomerulosclerosis, and endocapillary hypercellularity. Tubular atrophy/interstitial fibrosis and crescent involvement were categorized as <25%, 25–50%, and >50% of the affected renal tissue.

Data on therapy lines were prospectively collected, including treatment with renin-angiotensin-aldosterone system (RAAS) inhibitors, corticosteroids, immunosuppressive agents, and other supportive therapies. Adherence and treatment changes were also recorded. Patients were categorized into two groups based on the immunosuppressive drugs they received. In the immunosuppressive treatment group, patients received oral prednisolone at 0.5 mg/kg/day for 2 months, followed by gradual tapering over 6 months. The Immunosuppressive Treatment Group (IS) comprises 43 patients treated with

Immunosuppressive drugs, 29 male and 14 female.
The No Immunosuppressive Treatment Group (No IS) comprises 27 patients not treated with immunosuppressive drugs, 15 male and 12 female.
There was a slight imbalance in sex distribution between the groups, with a slightly higher proportion of males in the IS group than in the No IS group. The study outcomes were end-stage kidney disease (ESKD), the need for kidney replacement therapy (KRT), and all-cause mortality. The mandatory national registries enabled complete long-term follow-up of all patients, and the results were linked to the registration data.
Means, standard deviations, and descriptive statistics of baseline characteristics were reported. Quantitative variables were expressed as mean ± SD or median (interquartile range), while categorical variables were reported as frequencies and percentages. Cox proportional hazards regression models were employed to examine factors significantly associated with adverse outcomes. Multivariate analyses were performed to adjust for potential confounders.

Results

The study population included 70 patients, with a median age of 32 years (interquartile range 21.5–40.9), and 61.4% were male. Table 1 and Table 2 displays the baseline characteristics of the study population, comparing patients treated with immunosuppressive drugs (IS) and those without immunosuppressive treatment (No IS). While most characteristics were comparable between the groups, significant differences were noted in systolic blood pressure (SBP) and proteinuria levels. SBP was significantly higher in the IS group (125 mmHg vs. 120 mmHg, $P = 0.005$), and 74.1% of the IS group had moderate to severe proteinuria, which was defined as nephrotic-range proteinuria (>3.5 g/24 h), compared to 34.9% in the No IS group ($P = 0.002$).
Microhematuria was observed in 68.6% of patients, with no significant difference between the groups. Kidney function, measured by estimated glomerular filtration rate (eGFR), was >90 mL/min/1.73 m² in 42.9% of the overall population, with no notable differences between the IS and No IS groups. Histological analysis revealed that 71.4% of patients had <25% glomerulosclerosis, with no significant variation between the groups.
Univariable Cox regression analysis was conducted to identify prognostic factors associated with adverse renal outcomes. Severe proteinuria, reduced eGFR, and extensive tubulointerstitial damage emerged as significant predictors of end-stage kidney disease (ESKD) and mortality. Patients with an eGFR <30 mL/min/1.73 m² had the highest risk of ESKD, with a hazard ratio (HR) of 20.123 (95% CI: 9.831–41.188). Severe proteinuria (HR: 3.375, 95% CI: 1.578–7.218) and tubulointerstitial damage >50% (HR: 26.115, 95% CI: 10.617–64.236) were also strongly associated with increased risk.
In the No IS group, severe proteinuria (HR: 3.463, 95% CI: 1.420–8.441) and tubulointerstitial damage >50% (HR: 16.027, 95% CI: 4.353–59.901) significantly elevated the risk of adverse outcomes, while age, sex, and blood pressure were not significant predictors. In the IS group, severe proteinuria (HR: 3.194, 95% CI: 1.430–6.354) and extensive tubulointerstitial damage (HR: 41.067, 95% CI: 10.816–155.929) were associated with a substantially higher risk of adverse outcomes. (Table 3).

Table 1: Patients' Age Distribution

Category	All (N=70)
Male Patients	43 (61.4%)
Female Patients	27 (38.6%)
Mean Age (Years) ± SD	35 ± 11.3
Median Age (Years)	32 (21.5-0.9)

SD ~ Standard Deviation

Table 2: Clinical Baseline Characteristics

Category	All (N=70)	Immunosuppressive Drug Treatment (IS)N=43	Non-Immunosuppressive Drug Treatment (No IS) N=27	*Chi-square/ Mann-Whitney* test	*P*-value
Male Patients	43 (61.4%)	25 (58.1%)	18 (66.7%)		0.451
Female Patients	27 (38.6%)	18 (41.9%)	9 (33.3%)		
Median Age (Years)	32 (21.5-0.9)	31 (20.0–40.0)	32 (23.0-41.0)	0.213	0.762[a]
SBP (mmHg)	123 (110-50)	120 (110–145)	125 (110–155)		0.005[a]
DBP (mmHg)	80 (70–95)	78 (70–92)	82 (70–97)		0.904[a]
BP ≥ 130/80 mmHg	25 (35.7%)	15 (34.9%)	10 (37.0%)		0.098[a]
Urine Protein Groups (UPG)					
<0.75 g/L or 1 g/d	25 (35.7%)	20 (46.5%)	5 (18.5%)		
0.75–3 g/L or 1–3 g/d	35 (50.0%)	15 (34.9%)	20 (74.1%)	12.4292	0.002[b]
>3 g/L or >3 g/d	10 (14.3%)	8 (18.6%)	2 (7.4%)		
Hematuria Groups					
No	22 (31.4%)	13 (30.2%)	9 (33.3%)		
Microhematuria	48 (68.6%)	30 (69.8%)	18 (66.7%)	0.3203	0.852[b]
Macrohematuria	0 (0.0%)	0 (0.0%)	0 (0.0%)		
eGFR Groups (mL/min/1.73 m²)					
>90	30 (42.9%)	20 (46.5%)	10 (37.0%)		
60–90	30 (42.9%)	17 (39.5%)	13 (48.1%)	6.7306	0.081[b]
30–60	8 (11.4%)	5 (11.6%)	3 (11.1%)		
<30	2 (2.9%)	1 (2.3%)	1 (3.7%)		
Histologic Data					
Endocapillary hypercellularity	12 (17.1%)	6 (14.0%)	6 (22.2%)	0.5707	0.450[b]
Glomerulosclerosis					
<25%	50 (71.4%)	35 (81.4%)	15 (55.6%)		
25-50%	15 (21.4%)	5 (11.6%)	10 (37.0%)	3.1598	0.206[b]
>50%	5 (7.1%)	3 (7.0%)	2 (7.4%)		
Tubulointerstitial Damage Groups					
<25%	38 (54.3%)	22 (51.2%)	16 (59.3%)		
25-50%	15 (21.4%)	9 (20.4%)	6 (22.2%)	21.1075	<0.001[b]
>50%	17 (24.3%)	12 (27.9%)	5 (18.5%)		
Crescents Groups					
<25%	55 (78.6%)	35 (81.4%)	20 (74.1%)		
25-50%	8 (11.4%)	4 (9.3%)	4 (14.8%)	21.1075	<0.001[b]
>50%	7 (11.4%)	4 (9.3%)	3 (11.1%)		
RASBs (yes)	55 (78.6%)	32 (74.4%)	23 (85.2%)	15.1367	<0.001[b]

[a]Mann–Whitney U test performed; U statistic not shown due to summarized data presentation, [b]Chi-square test
Systolic Blood Pressure ~SBP, Diastolic Blood Pressure ~ DBP

Table 3: Univariable Cox Regression Analysis

	All (70)			Non-Immunosuppressive Drug Treatment (IS group) 43			Immunosuppressive Drug Treatment (IS group) 27		
	HR	CI 95%	*P value*	HR	CI 95%	*P value*	HR	CI 95%	*P-value*
Age (years) (continuous)	1.018	0.999–1.037	0.068	1.017	0.989–1.046	0.229	1.018	0.991–1.045	0.19
Sex (Ref. male)	1.156	0.678–1.971	0.594	0.515	0.215–1.233	0.133	1.286	0.277–4.593	0.728
BP initial (Ref. < 130/80 mmHg)	1.365	0.770–2.418	0.287	1.671	0.771–3.873	0.231	1.032	0.471–2.262	0.937
eGFR initial (Ref. ≥ 60 mL/min/1.73 m²)									
>30–60 mL/min/1.73 m²	2.878	1.584–5.301	0	1.819	0.833–3.971	0.133	3.405	1.491–7.775	0.003
<30 mL/min/1.73 m²	20.123	9.831–41.188	0	15.069	5.254–43.223	0	24.508	7.898–68.267	*P*< 0.001
Proteinuria initial groups (Ref. mild)									
Moderate	2.244	1.144–4.399	0.02	2.266	0.895–5.742	0.085	1.705	0.653–3.739	0.55
Severe	3.375	1.578–7.218	0.002	3.463	1.420–8.441	0.006	3.194	1.430–6.354	0.004
Endocapillary hypercellularity (Ref.NO)	1.955	0.639–5.980	0.24	2.074	0.078–7.182	0.149	1.51	0.523–4.154	0.409
Glomerulosclerosis (Ref. NO)									
Tubule-interstitial damage groups (Ref. <25%)									
25–50%	4.592	2.463–7.873	0	3.826	1.529–9.341	0.004	5.264	2.312–11.887	<0.0010
>50%	26.115	10.617–64.236	0	16.027	4.353–59.901	0	41.067	10.816–155.929	<0.001
Crescents group (Ref. <25%)									
<25%	1.084	0.336–2.070	0.865	1.008	0.665–6.067	0.217	0.389	0.078–1.875	0.238
25-50%	1.957	0.961–3.984	0.064	1.388	0.377–5.107	0.663	2.114	0.767–5.823	0.148
>50%	6.664	3.329–13.324	0	5.668	1.623–19.876	0.007	6.697	2.414–18.582	*P*< 0.001

Discussion

The present study identified certain clinical and histological parameters as risk modifiers for ESKD, KRT, and mortality among patients with IgAN. Our analysis confirmed the predictive value of initial eGFR, proteinuria, and tubulointerstitial damage in determining patient outcomes. These findings are consistent with previous studies in larger cohorts across Asia and Europe, in which eGFR and proteinuria have consistently emerged as dominant predictors of renal survival.[8,9] These findings confirm international patterns but also provide novel, region-specific evidence from Balochistan, where limited healthcare access and delayed diagnosis may contribute to faster disease progression. In our cohort, 74.1% of patients in the immunosuppressive treatment group had nephrotic-range proteinuria (>3.5g/24 h), consistent with previous studies reporting that immunosuppression is predominantly used in patients with high-risk disease and heavy proteinuria.

The Cox regression analysis highlighted eGFR as a strong predictor of adverse outcomes, particularly when below 30 mL/min/1.73 m².[10] This is in line with the KDIGO 2021 guidelines and other cohort studies, which found reduced eGFR to be the most reliable predictor of progression to ESKD in IgAN.[11] Similarly, proteinuria has long been associated with glomerular injury and faster progression to kidney failure.[12] Our finding that severe proteinuria increased the risk of ESKD is consistent with findings from large observational cohorts and meta-analyses that confirm its dose-dependent predictive value.[13] However,

some studies suggest that in patients with chronic histological damage, immunosuppressive therapy may fail to significantly reverse outcomes associated with severe proteinuria. This may be due to underlying fibrotic changes that are not compliant with steroid-based treatment.[14]

Tubulointerstitial damage strongly predicts kidney function decline in IgA nephropathy, consistent with observations reported in previous literature.[15] Our results confirm that extensive (>50%) damage substantially increases the risk of ESRD and death. Interestingly, patients receiving immunosuppressive therapy with >50% interstitial damage showed the highest hazard ratio, possibly reflecting selection bias where sicker patients were more likely to receive aggressive treatment.

Our findings on crescents are also in line with prior studies. Patients with extensive crescents (>50%) were at significantly increased risk of ESKD, further highlighting the importance of early histologic assessment.[16]

The subgroup analysis further highlighted differential predictive patterns. In patients not receiving immunosuppressive therapy, moderate proteinuria and low eGFR were key predictors. In contrast, among those who received therapy, only severe proteinuria retained predictive power. This suggests a partial mitigation effect of therapy on moderate disease, although high-grade damage likely requires additional interventions beyond standard immunosuppression. Sex and baseline blood pressure values were not significantly associated with poor outcomes in our study. Although previous studies from Chinese and Korean cohorts have reported hypertension as an independent risk factor for disease progression in IgAN.[17,18] Our study did not include hypertension status as a variable, which limits direct comparison.

There are several limitations to this study. The retrospective nature inherently limits control over confounding variables and treatment adherence. The small sample size reduces statistical power and generalizability.

Future research should prioritize prospective multicenter studies in Pakistan to enhance generalizability and address regional variation in IgAN. Biomarker-guided risk stratification, grounded in the multi-hit pathogenesis model, may improve individualized care. Comparative trials between standard immunosuppression and novel agents such as SGLT2 inhibitors and targeted biologics are warranted, as SGLT2 inhibitors have shown proteinuria-reducing effects in IgAN.[19,20] Additionally, the long-term benefits of optimized supportive care, including RAAS blockade and blood pressure control, remain areas for further investigation.[21]

Although immunosuppression was frequently used in high-risk patients, causal benefit cannot be established due to the retrospective design. Blood pressure and sex did not significantly impact outcomes, but the presence of crescents and histological damage remained key risk factors. Our findings highlight the need for close monitoring and early intervention, especially in patients with reduced eGFR and high proteinuria, to improve renal outcomes. Future research should focus on individualized treatment options to enhance the effectiveness of immunosuppressive treatment and to explore new therapies targeting proteinuria and histological damage.

Conclusion

Reduced eGFR, severe proteinuria, and extensive tubulointerstitial damage were the strongest predictors of ESKD, KRT, and mortality in patients with IgAN. Overall, our findings emphasize the importance of early identification and continuous monitoring of IgAN patients, especially those with reduced eGFR, proteinuria, and histological damage. They contribute region-specific data that can inform local nephrology practice and guide personalized treatment planning.

Acknowledgement: None

Conflict of Interest: The authors declare no conflict of interest

Grant Support and Financial Disclosure: None

REFERENCE

1. Jia Q, Ma F, Zhao J, Yang X, Sun R, Li R, et al. Effect of corticosteroids combined with cyclophosphamide or mycophenolate mofetil therapy for IgA nephropathy with stage 3 or 4 chronic kidney disease: A retrospective cohort study. Frontiers in Pharmacology. 2022; 13: 946165. doi: 10.3389/fphar.2022.946165
2. Pitcher D, Braddon F, Hendry B, Mercer A, Osmaston K, Saleem MA, et al. Long-term outcomes in IgA nephropathy. Clinical Journal of the American Society of Nephrology. 2023; 18: 727-38. doi: 10.2215/CJN.0000000000000135
3. Qin Y, Yu Z, Wu H, Wang A, Wang F, Wang D, et al. Prognostic

factors affecting long-term outcomes in patients with concurrent IgA nephropathy and membranous nephropathy. Heliyon. 2024; 10: e23436. doi: 10.1016/j.heliyon.2023.e23436
4. Zhao J, Ma F, Bai M, Sun S. Low-dose corticosteroid combined with mycophenolate mofetil for IgA nephropathy with stage 3 or 4 CKD: A retrospective cohort study. Clinical Therapeutics. 2021; 43: 859-70. doi: 10.1016/j.clinthera.2021.03.009
5. Guan Y, Liu S, Hao CM, Lai L. Effect of nonimmune factors on renal prognosis in adult IgA vasculitis with nephritis: A long-term retrospective cohort study. Journal of Rheumatology. 2023; 50: 1032-8. doi: 10.3899/jrheum.2022-1100
6. Mohd R, Mohammad Kazmin NE, Abdul Cader R, Abd Shukor N, Wong YP, Shah SA, et al. Long-term outcome of immunoglobulin A (IgA) nephropathy: A single center experience. PLOS ONE. 2021; 16: e0249592. doi: 10.1371/journal.pone.0249592
7. Shirai S, Yasuda T, Kumagai H, Matsunobu H, Ichikawa D, Shibagaki Y, et al. Prognostic factors of IgA nephropathy presenting with mild proteinuria at the time of diagnosis: A multicenter cohort study. Clinical and Experimental Nephrology. 2023; 27: 340-8. doi: 10.1007/s10157-023-02316-2
8. Barbour SJ, Coppo R, Zhang H, Liu ZH, Suzuki Y, Matsuzaki K, et al. Application of the international IgA nephropathy prediction tool one or two years post-biopsy. Kidney International. 2022; 102: 160-72. doi: 10.1016/j.kint.2022.02.042
9. Barbour SJ, Coppo R, Zhang H, Liu ZH, Suzuki Y, Matsuzaki K, et al. Evaluating a new international risk-prediction tool in IgA nephropathy. JAMA Internal Medicine. 2019; 179: 942-52. doi: 10.1001/jamainternmed.2019.0600
10. Cox DR. Regression models and life tables. Journal of the Royal Statistical Society: Series B (Methodological). 1972; 34: 187-202. doi: 10.1111/j.2517-6161.1972.tb00899.x
11. Rovin BH, Adler SG, Barratt J, Bridoux F, Burdge KA, Chan TM, et al. KDIGO 2021 clinical practice guideline for the management of glomerular diseases. Kidney International. 2021; 100: S1-276. doi: 10.1016/j.kint.2021.05.021
12. Gorriz JL, Martinez-Castelao A. Proteinuria: Detection and role in native renal disease progression. Transplantation Reviews. 2012; 26: 3-13. doi: 10.1016/j.trre.2011.10.002
13. Coppo R, D'Arrigo G, Tripepi G, Russo ML, Roberts ISD, Bellur S, et al. Is there long-term value of pathology scoring in immunoglobulin A nephropathy? A validation study of the Oxford Classification for IgA Nephropathy (VALIGA) update. Nephrology Dialysis Transplantation. 2018; 35: 1002-9. doi: 10.1093/ndt/gfy302
14. Yang P, Zou H, Xiao B, Xu G. Comparative efficacy and safety of therapies in IgA nephropathy: A network meta-analysis of randomized controlled trials. Kidney International Reports. 2018; 3: 794-803. doi: 10.1016/j.ekir.2018.03.006
15. Hodgkins KS, Schnaper HW. Tubulointerstitial injury and the progression of chronic kidney disease. Pediatric Nephrology. 2011; 27: 901-9. doi: 10.1007/s00467-011-1992-9
16. Du Y, Chen S, Wang F, Zhang P, Liu M, Liu C, et al. The significance of crescents on the clinical features and outcomes of primary immunoglobulin A nephropathy. Frontiers in Medicine. 2022; 9: 864667. doi: 10.3389/fmed.2022.864667
17. Zheng Y, Wang Y, Liu S, Wu J, Duan S, Zhu H, et al. Potential blood pressure goals in IgA nephropathy: Prevalence, awareness, and treatment rates in chronic kidney disease among patients with hypertension in China (PATRIOTIC Study). Kidney and Blood Pressure Research. 2018; 43: 1786-95. doi: 10.1159/000495636
18. Oh TR, Choi HS, Oh SW, Oh J, Lee DW, Kim CS, et al. Association between the progression of immunoglobulin A nephropathy and a controlled status of hypertension in the first year after diagnosis. Korean Journal of Internal Medicine. 2022; 37: 146-53. doi: 10.3904/kjim.2020.205
19. Dong Y, Shi S, Liu L, Zhou X, Lv J, Zhang H. Effect of SGLT2 inhibitors on the proteinuria reduction in patients with IgA nephropathy. Frontiers in Medicine. 2023; 10: 1242241. doi: 10.3389/fmed.2023.1242241
20. Zou M, Xu G, Ge S, Guo K, Duo Q, Cheng Y. Network pharmacological analysis of hydroxychloroquine intervention in the treatment of IgA nephropathy. Current Pharmaceutical Design. 2025; 31: 730-40. doi: 10.2174/0113816128347345241028063515
21. Zhao Y, Fan H, Bao BY. Efficacy and safety of renin-angiotensin aldosterone system inhibitor in patients with IgA nephropathy: A meta-analysis of randomized controlled trials. Iranian Journal of Public Health. 2019; 48: 1577-88.

Author Contributions

ZK: Conception and design of the work
AA: Data acquisition, curation, and statistical analysis
NF: Manuscript writing for methodology design and investigation
NK: Revising, editing, and supervising for intellectual content