RESEARCH ARTICLE

Clinicopathological profile, risk factors and outcome of adult biopsy-proven infection-related glomerulonephritis: A single centre prospective study

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Abstract

Objective: To assess the clinicopathological profile, risk factors and outcomes of biopsy-proven infection-related glomerulonephritis.

Method: The prospective, cross-sectional, observational study was conducted at the Department of Nephrology, Ayub Teaching Hospital, Abbottabad, Pakistan, from April 5, 2023, to December 30, 2024, and comprised patients of either gender aged at least 18 years who underwent renal biopsy. Demographic, clinical, laboratory and histopathological data was collected prospectively. Outcomes were tracked, and comorbidities and histological features were assessed for prognostic relevance. Data was analysed using SPSS 23.

Results: Of the 180 renal biopsies, 22(12.2%) were diagnosed with biopsy-proven infection-related glomerulonephritis; 14(63.6%) males and 8(36.4%) females, with overall mean age being 36.23±19.79 years. Renal dysfunction was evident with elevated serum creatinine 3.30±3.11mg/dL and hypoalbuminaemia 3.19±0.6mg/dl accompanied by nephrotic-range proteinuria 3.82±3.05g/day. Haematuria 17(77.2%) and leucocyturia 12(54.5%) were the most common presentations. Elevated antistreptolysin O titers were found in 9(41%) cases, and hypocomplementaemia in 13(59%). Histopathology revealed neutrophilic infiltration 19(86.6%), endocapillary proliferation 18(81.8%), mesangial proliferation 17(77.2%), and crescents 5(22.2%), with interstitial fibrosis 6(27.2%) signalling chronicity. Immunofluorescence showed dominant complement component 3 deposits 22(100%) alongside immunoglobulins G, M and A co-deposits. Hypertension 8(36.4%) was a key comorbidity, alongside diabetes 2(9.1%) and anaemia 5(22.7%). While 18(81.8%) patients achieved full recovery, 2(9.1%) progressed to chronic kidney disease and 2(9.1%) to end-stage renal disease.

Conclusion: Infection-related glomerulonephritis in adult patients was found to present with diverse clinicopathological features, often post-infection. Most patients recovered, but progression to chronic kidney disease and end-stage renal disease correlated with hypertension and chronic histological changes.

Keywords: Glomerulonephritis, Infection, Kidney diseases, Biopsy, Risk factors. (JPMA 76: 909; 2026)

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Introduction

Infection-related glomerulonephritis (IRGN) has re-emerged as an important cause of glomerular disease in the adult population, particularly among the elderly and those with comorbid conditions, such as diabetes mellitus (DM), malignancies and chronic infections. Although it was historically considered a paediatric condition predominantly linked to streptococcal infections, recent epidemiological trends highlight a growing incidence among adults. In this group, *staphylococcus (S.) aureus*, gram-negative bacilli, and various non-streptococcal pathogens have become the predominant culprits, frequently in association with healthcare-related infections and immunocompromised states.[1,2]

Compared to paediatric cases, IRGN in adults often follows a more aggressive clinical trajectory. Common presentations include acute kidney injury (AKI), nephritic or nephrotic-range proteinuria, and an elevated risk of progression to chronic kidney disease (CKD) or end-stage renal disease (ESRD).[3,4] Histopathological evaluation remains essential for diagnosis, typically showing endocapillary proliferative and exudative glomerulonephritis (GN), along with immune complex deposits observable through immunofluorescence (IF) and electron microscopy.[5]

Despite progress in diagnostic methodologies, IRGN remains a challenging condition to diagnose and manage due to its broad clinical spectrum and histological similarities with other glomerular diseases. Factors associated with poor renal prognosis include advanced age, DM, hypoalbuminaemia, and delays in diagnosis.[6] Notably, there is a scarcity of prospective data from low- and middle-income countries (LMICs), where infections are more prevalent and patients often present late. Thus, region-specific research is essential to enhance understanding of the clinical and pathological features as well as the prognostic factors of adult IRGN.

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The current study was planned to characterise the clinicopathological profile, identify contributing risk factors, and evaluate renal outcomes in adults with biopsy-confirmed IRGN.

Patients and Methods

The prospective, cross-sectional, observational study was conducted at the Department of Nephrology, Ayub Teaching Hospital, Abbottabad, Pakistan, from April 5, 2023, to December 30, 2024. After approval from the institutional review board of the Medical Teaching Institution, Abbottabad, patients of either gender aged at least 18 years who underwent renal biopsy were included, and their biopsy reports and clinical records were reviewed to collect relevant information. Written consent was obtained from all patients before the biopsies. The diagnosis of IRGN was established on the basis of characteristic clinical, laboratory and histopathological features. These included a recent or concurrent history of infection (such as skin, respiratory tract, urinary tract, or other systemic infections), compatible light microscopy findings, and IF patterns showing dominant or co-dominant complement component 3 (C3) deposition. Complete clinical, laboratory, and follow-up data was acquired for the cases included.

Patients with inadequate biopsy samples (e.g., insufficient glomeruli <5 for diagnosis), those whose biopsy revealed alternative or confounding diagnoses, such as lupus nephritis, anti-neutrophil cytoplasmic antibodies (ANCA)-associated vasculitis, or pure diabetic nephropathy without evidence of IRGN, and those with coexisting autoimmune diseases or CKD not attributed to infection were excluded. Patients with post-transplant kidney biopsies were also excluded.

The biopsies, done using ultrasound guidance with a C.R. Bard, Inc (Murray Hill, New Jersey, USA) Monopty Gun (18G×20cm for native biopsies), were analysed using light microscopy (LM) and IF. For LM, special stains, like haematoxylin and eosin (H&E), periodic acid schiff (PAS), Masson's trichrome and silver stains, were used. IF was done with polyclonal antibodies to detect immunoglobulin g (IgG), IgM, IgA, C3 and C1q, following the manufacturer's guidelines. Specific manufacturer details of the laboratory equipment, reagents, and diagnostic kits were not included in the reports provided to the authors and therefore could not be specified. The diagnosis of IRGN was done clinically and biopsy LM features and IF findings. Endocapillary proliferative pattern with neutrophilic infiltration on LM may provide a first diagnostic clue for IRGN. IF findings of infectious GN typically showed C3 deposition with or without staining for IGs, mainly IgG co-distributed with C3. Notably, infection-related GN may be linked to underlying complement dysregulation, positioning it within the spectrum of C3 glomerulopathies.[7] These findings were combined with clinical features to establish the diagnosis.

Data was analysed using SPSS 23. Descriptive statistics were used to summarise patient characteristics. Categorical variables were analysed using the chi-square test. Continuous variables were analysed using the independent sample *t*-test. P<0.05 was considered statistically significant.

Results

Of the 180 renal biopsies, 22(12.2%) were diagnosed with biopsy-proven infection-related glomerulonephritis; 14(63.6%) males and 8(36.4%) females, with overall mean age being 36.23±19.79 years. Renal dysfunction was evident with elevated serum creatinine 3.30±3.11mg/dL and hypoalbuminaemia 3.19±0.6mg/dl accompanied by nephrotic-range proteinuria 3.82±3.05g/day. Mean eGFR was 47.18±38.18mL/min/1.73m^2 Haematuria 17(77.2%) and leucocyturia 12(54.5%) were the most common presentations. Elevated antistreptolysin O (ASO) titers were found in 9(41%) cases, and hypocomplementaemia in 13(59%) (Table 1).

Table-1: Demographics and clinical features of the patients (n=22).

Parameters		Male	Female	*p*-alue
Gender		14 (63.6%)	8 (36.3%)	
Mean Age	36.23±19.79	33.64±18±85	40.75±21.88	0.72
eGFR	47.18±38.18	49.00±39.37	44.00±38.43	0.46
S. Albumin	3.19±0.6	3.23±0.6	3.11±0.6	0.27
24 hr. Proteinuria	.82±3.05	3.58±3.36	4.25±2.59	0.45
Mean Creatinine	3.30±3.11	3.66±3.51	2.65±2.32	0.09
Haematuria				
Gross		4 (18.1%)	4 (18.1%)	0.52
Microscopic		6 (27.2%)	3 (13.6%)	
No		4 (18.1%)	0	
ASO titre				
Raised		5 (22.7%)	4 (18.1%)	0.51
Normal		9 (40.9%)	4 (18.1%)	
Leucocyturia				
Yes		7 (31.8%)	5 (22.7%)	0.57
No		7 (31.8%)	3 (13.6%)	
Presentation				
Nephrotic		6 (27.2%)	4 (18.1%)	0.68
Nephritic		4 (18.1%)	3 (13.6%)	
RPGN		4(18.1%)	1 (4.5%)	
Oedema				
Localized		9 (40.9%)	2 (9%)	0.07
Generalised		5 (22.7%)	6 (27.2%)	
Complement				
Low C3, C4		5 (22.7%)	2 (9%)	
Low C3		4 (18.1%)	2 (9%)	0.79
Normal		5 (22.7%)	4 (18.1%)	

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Table-1: *Continued from previous page*

Parameters	Male	Female	*p*-alue
Need of Dialysis			
Yes	4 (18.1%)	1 (4.5%)	0.38
No	10 (45.45%)	7 (31.8%)	
IFTA			
Positive	2 (9%)	4 (18.1%)	0.07
Negative	12 (54.54%)	4 (18.1%)	
Outcome			
CKD	2 (9%)	1 (4.5%)	0.51
ESRD	2 (9%)	0	
Recovery	10 (45.45%)	7 (31.8%)	
Hypertension			
Yes	4 (18.1%)	4 (18.1%)	0.31
No	10 (45.45%)	4 (18.1%)	
Diabetes			
Yes	1 (4.5%)	1 (4.5%)	0.75
No	13 (59.09%)	7 (31.8%)	
Anaemia			
Yes	4 (18.1%)	1 (4.5%)	0.38
No	10 (45.45%)	7 (31.8%)	

eGFR: Estimated glomerular filtration rate, CKD: Chronic kidney disease, ESRD: End-stage renal disease, RPGN: Rapidly progressive glomerulonephritis, IFTA: Interstitial fibrosis/tubular atrophy, ASO: Antistreptolysin O, C3: Complement component 3.

Histopathology revealed neutrophilic infiltration 19(86.6%), endocapillary proliferation 18(81.8%), mesangial proliferation 17(77.2%), and crescents 5(22.2%), with interstitial fibrosis/tubular atrophy (IFTA) 6(27.2%) signalling chronicity (Table 2).

IF showed dominant complement component 3 deposits 22(100%) alongside IgG, IgM and IgA co-deposits (Table 3). Hypertension (HTN) 8(36.4%) was a key comorbidity, alongside DM 2(9.1%) and anaemia 5(22.7%). While 18(81.8%) patients achieved full recovery, 2(9.1%) progressed to CKD and 2(9.1%) to ESRD.

Discussion

The current prospective study, conducted at a tertiary care hospital in Abbottabad, Pakistan, evaluated 22 biopsy-proven IRGN cases that represented 12.2% of the 180 total renal biopsies, providing critical insights into the clinicopathological profile, risk factors and outcomes in a resource-limited setting.

Table-2: Light microscopy features.

Histopathology	n (%)
Endocapillary proliferative pattern	18 (81.8)
Mesangial proliferative pattern	17 (77.2)
Membranoproliferative GN	03 (13.6)
Crescentic Infection Related GN	05 (22.7)
Neutrophilic infiltration (exudative)	19 (86.6)
Interstitial inflammation	15 (68.1)
Plasma cell infiltration	07 (31.8)
Interstitial Oedema	13 (59)
Acute tubular injury	15 (68.1)
Interstitial Fibrosis and Tubular Atrophy	06 (27.3)
Chronic interstitial nephritis	09 (40.9)

GN: Glomerulonephritis.

The 12.2% prevalence of IRGN in the current study is comparable to rates reported in other South Asian studies. A retrospective study in India reported an IRGN incidence of 4.6% among renal biopsies.[8] Another study in India noted that IRGN was the most common glomerulopathy in elderly patients.[2]

A male predominance (63.6%) was noted in the current cohort, consistent with other reports.[9,10] This pattern may be due to higher infection risk or delayed health-seeking behaviour in men, especially in resource-limited settings.

Comorbidities, such as HTN (36.4%), DM (9.1%) and anaemia (22.7%), were prominent in the current sample. DM is a known risk factor for IRGN due to immune dysregulation. Studies have shown that IRGN

Table-3: Characteristics of infectious glomerulonephritis cases.

S. No	Biopsy Diagnosis	Immunofluorescence pattern	Deposits site	Outcome
1	IgA-dominant Post-infectious GN	C3, IgA, IgM	Mesangium	Recovery
2	Membranoproliferative GN	C3, IgA, IgM, IgG	Mesangium & tubules	Recovery
3	Membranous Glomerulonephritis	C3, IgA, IgM, IgG	Mesangium & GBM	Recovery
4	Membranous GN with Granulomatous necrotizing nephritis	C3, IgM, IgG	Mesangium	Recovery
5	IgA-dominant Post-infectious GN	C3, IgM, IgA	Mesangium	Recovery
6	FSGS Secondary to Infection	C3, IgG	Mesangium	Recovery
7	Diffuse Proliferative GN	C3, IgG	Mesangium	Recovery
8	Post Infectious Glomerulonephritis	C3, IgM	Mesangium	Recovery
9	Post Infectious Glomerulonephritis	C3, IgM, IgG	Mesangium & GBM	CKD
10	Post Infectious Glomerulonephritis	C3, IgM, IgG	Mesangium & GBM	Recovery
11	Membranous GN secondary to Tuberculosis	C3, IgG	Mesangium & GBM	CKD
12	Pauci-immune Glomerulonephritis	Weak C3, IgM	Mesangium	Recovery
13	Membranous GN secondary to Hepatitis B	C3, IgG	Mesangium & GBM	Recovery
14	Membranoproliferative GN	C3, IgM, IgG, IgA	Mesangium & GBM	Recovery
15	Immune complex Glomerulonephritis	C3, IgG, IgM	Mesangium	Recovery
16	MPGN secondary to Hepatitis C	C3, IgG	Mesangium & GBM	ESRD
17	FSGS secondary to chronic infection	C3, IgM	Mesangium	ESRD
18	Infectious GN secondary to Haemolytic uraemic syndrome	C3, IgM	Mesangium	Recovery
19	IgA-Dominant Post-Infectious GN	C3, IgA, IgM	Mesangium	Recovery
20	Post-Infectious Glomerulonephritis	C3, IgM	Mesangium	Recovery
21	Chronic Granulomatous GN Secondary to Tuberculosis	C3, IgM	Mesangium	Recovery
22	Diffuse Proliferative GN	C3, IgM	Mesangium	Recovery

GN: Glomerulonephritis, FSGS: Focal segmental glomerulosclerosis, GBM: Glomerular basement membrane, ESRD: End-stage renal disease, CKD: Chronic kidney disease, Ig: Immunoglobulin, C3: Complement component 3.

superimposed on diabetic kidney disease leads to poorer renal outcomes.[10] Anaemia, common in renal inflammation and dysfunction, contributes to glomerular hypoxia, thereby accelerating injury.[11]

Clinically, most of the current patients presented with acute nephritic syndrome, including haematuria, proteinuria and renal impairment, which is consistent with the classical presentation of IRGN.[12] Histopathologically, 86% patients showed diffuse proliferative GN with neutrophilic infiltration — hallmarks of IRGN.[13] IF findings revealed dominant C3 deposition in 95% of the cases, often with IgG and IgM co-deposits (45%), aligning with the literature.[10,13]

Renal recovery occurred in 81.8% of the current patients, while 9.1% progressed to CKD and 9.1% to ESRD. These findings are similar to those of Sanathkumar et al., who reported that delayed diagnosis and IFTA were predictors of poor outcome.[8] These outcomes also mirror data from Trivedi et al.,[14] where delayed diagnosis and comorbidities worsened prognosis. Similarly, Ramineni et al. in 2021 reported complete recovery in 84.6% patients, while 11.1% developed CKD, and 3.7% progressed to ESRD.[3] In addition, immunocompromised status and persistent infections were linked to delayed recovery. Forster et al. highlighted that mycobacterium tuberculosis-associated GN often presents atypically in immunocompromised individuals, complicating diagnosis and management.[15]

The current study has limitations, including its single-centre design, which may restrict the generalisability of the findings, and a relatively small sample size. Besides, the sample size was not calculated. There is also a lack of culture evidence to confirm infectious aetiology, and the diagnosis relied on clinical presentation, LM and IF findings.

Conclusion

Most patients presented with acute nephritic syndrome and low complement levels. The predominant histopathological pattern was endocapillary proliferative GN with C3-dominant immune deposits. Factors, such as advanced age, presence of comorbidities, elevated serum creatinine at presentation, and chronic histological changes were associated with poorer renal outcomes.

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Author Contribution:

AA: Performed the renal biopsies, design, data interpretation and analysis.

AW: Concept, collected and assembled the data, wrote the initial draft and revision.