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ORIGINAL ARTICLE

Infection Related Glomerulonephritis in Adults: A Prospective Observational Study from South India

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Abstract

Introduction: Infection-related glomerulonephritis, IRGN in adults is increasing in prevalence in India. However, there are only a few prospective studies from south India on the long term prognosis of this disease. **Materials and Methods:** This is a prospective observational study on patients above 18 years of age,with biopsy proven IRGN, at a tertiary care centre in south India with a minimum follow up of 6 months. **Results:** A total of 100 patients were included in the study. Peak incidence was in the age group of 40-60 years (n=45, 45%) with a male preponderance (n=56,56%). The most common co-morbidities were diabetes mellitus (n=27, 27%) and alcohol intake (n=13,13%). The most common source of infection was skin and soft tissues (n=41, 41%). Streptococcus and Staphylococcus were the most common isolated organisms. One patient had IRGN following SARS C0V-2 virus infection. Twenty three (n=23, 23%) required dialysis initiation. The mean follow up was 31.4 weeks. 3 patients died within 1 month of illness. At the end of 6 months, out of the 97 surviving patients, 34 (n=34,35%) patients had chronic kidney disease,CKD (eGFR <60 ml/per/1.73 m^2) and 9 (n=9,9.27%) were dialysis dependant. Age >40 years, associated diabetes mellitus,underlying CKD, dialysis requiring renal failure at presentation,presence of crescentric GN and moderate to severe Interstitial Fibrosis and Tubular Atrophy in renal biopsy were identified as risk factors for development of CKD by univariate analysis. **Conclusion:** Adult IRGN is as an important cause of CKD and end stage renal disease in Indian adults especially in diabetics and those above 40 years of age.

Keywords: Adult IRGN, South India, Clinical profile, Outcome

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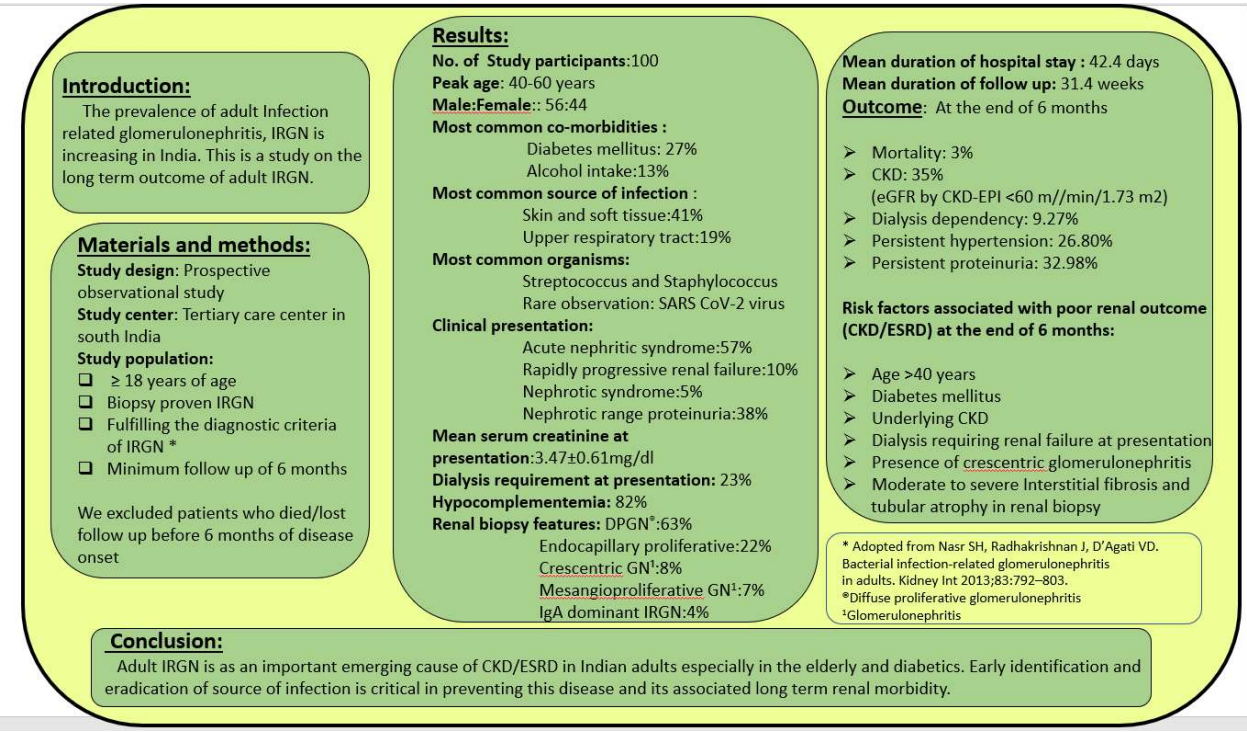
Graphical Abstract



Introduction

Infection-related glomerulonephritis (IRGN) is an immune complex-mediated acute glomerulonephritis occurring in association with a variety of non-renal infections [1]. The incidence and prevalence of postinfectious glomerulonephritis in India, based on data from biopsy studies, is 39.24 cases per year and 10.14 cases per 100,000 population respectively [2]. Also its prevalence in adults has been increasing in recent decades [2] thereby causing significant renal morbidity. However, there are limited studies on the long-term prognosis of this disease in adults from South India.

Materials and Methods

We did a prospective observational study on patients with biopsy-proven IRGN from June 2017 to July 2021, at Government Kilpauk Medical College and Government Royapettah Hospital, Chennai, India. We included patients above 18 years of age with the diagnosis of IRGN and were followed up for a minimum period of 6 months. The diagnosis of IRGN was done based on the criteria put forth by Nasr et al. [3], which require the presence of at least three of the following five criteria: (i) clinical or laboratory evidence of infection preceding or at the onset of glomerulonephritis; (ii) depressed serum complement; (iii) endocapillary proliferative and exudative glomerulonephritis; (iv) C3-dominant or co-dominant glomerular immunofluorescence staining; and (v) hump shaped sub-epithelial deposits on electron microscopy (EM).

Demographic data, co-morbidities, and clinical presentation were documented. Investigations namely complete blood count, random blood sugar; renal function tests, urine analysis; urine protein–creatinine ratio (uPCR); serum electrolytes; liver function tests; serological tests for HIV, hepatitis B

and hepatitis C; and complement levels (C3 and C4) were done in all patients.

Renal biopsy was done by trained nephrologists under ultrasonic guidance and sent for histopathological examination to the renal pathologist. Immuno-fluorescence (IF) and light microscopic (LM) examination were done in all samples. Haematoxylin and eosin, periodic acid–Schiff, Jone's methenamine silver and Masson's trichrome stains were used for light microscopic study. Electron Microscopy (EM) was done only in selected patients in whom EM was needed to fulfill the adopted diagnostic criteria.

Complete physical examination, and investigations to find the infective foci namely echocardiography, chest X-ray, ultrasound abdomen and cultures of blood, urine, pus and other appropriate samples in individual patients were done. ENT (ear, nose, throat) opinion, dental opinion, dermatologist opinion for infective foci were obtained in all patients even in those with an identifiable source of infection to rule out multiple foci. The clinical presentation of patients was classified based on the following definition

1. Acute nephritic syndrome: defined by the occurrence of hematuria, proteinuria, and edema, often with hypertension and azotemia in temporal relation.
2. Rapidly progressive renal failure: defined by a rapid decline in GFR over days to weeks.
3. Nephrotic proteinuria: defined by the presence of more than 3.5 gm of proteinuria per day.
4. Nephrotic syndrome: defined by the triad of nephrotic range proteinuria (more than 3.5 gm of proteinuria per day), edema and hypoalbuminemia.

All patients were treated with standard of care for the management of IRGN namely salt and fluid restriction; blood pressure control with antihypertensives and diuretics. Calcium channel blockers were used as the first choice anti-hypertensives. Dialysis initiation was done in patients who presented with severe renal failure (serum creatinine >7 mg/dl), or if encephalopathy/ refractory pulmonary edema/ metabolic acidosis/ hyperkalemia occurred. Steroids were used in patients who presented with dialysis-dependent renal failure and/or in those with crescents in renal biopsy study after ruling out all possible sources of active infection. Our steroid regimen was injection Methylprednisolone 250 mg/day for 3 consecutive days followed by oral prednisolone of 0.5 mg per kg for 4 weeks and rapid tapering over the next two weeks.

All patients were followed up with weekly visits in the first month and with monthly visits after that, for 5 months. Blood pressure, renal function and proteinuria were monitored in each visit. Glomerular filtration rate, eGFR was calculated using the CKD-EPI (chronic kidney disease epidemiology collaboration) formula. Complement levels were not repeated in all of our patients in view of unaffordability.

The primary objective was to analyse the risk of CKD (eGFR < 60 m/min/1.73 m^2)/dialysis dependency at the end of 6 months and the secondary objective was to analyse the risk factors associated with CKD/dialysis dependency at the end of 6 months. Patients who died and those who lost follow-up were excluded from the final analysis.

Statistical analysis was performed using SPSS for Windows version 15.0. Univariate analysis was done using Fischer's exact test. The Institutional Ethics

Committee approval was obtained with IEC Protocol No. 816/2022.

Results

A total of 100 patients were included in the study. Peak incidence was in the age group of 40–60 years (n=45,45%) with a male preponderance (n=56,56%) (Table 1). Eleven patients (n=11,11%) were above 60 years of age (Table 1). The most common co-morbities observed were diabetes mellitus (n=27, 27%) and alcohol intake (n=13,13%) (Table 1). The most common source of infection was skin and soft tissues (n=41,41%) in both diabetics and non-diabetics followed by the upper respiratory tract (n=19,19%) (Figure 1). About twenty-three patients (n=23,23%,) had no identifiable infection source despite a meticulous search (Figure 1). Streptococcus and Staphylococcus were the most common organisms identified in our study. One of our patients had IRGN in association with SARS CoV-2 infection.

The clinical presentations are shown in Table 2. Edema and proteinuria were universally present. New onset hypertension was seen in 82 (n=82,82%) patients and macrohaematuria in 32 (n=32,32%) patients. Acute nephritic syndrome was the most common renal syndrome at presentation (n=57,57%); ten (n=10,10%) presented with Rapidly progressive renal failure (RPRF).

Nephrotic range proteinuria was present in 38 (n=38, 38%) patients. Sixty nine (n=69, 69%) patients had renal failure at presentation. The mean serum creatinine at admission was 3.47±0.611 mg/dl. Thirty (n=30,30%) patients presented with serum creatinine >4.0 mg/dl and twenty three (n=23,23%) required initiation of haemodialysis. Hypocomplementaemia was seen in 82 (n=82,82%) patients, out of which 74 (n=74,74%) had low C3 with normal C4, and eight (n=8,8%) had low C3 with low C4.

In the renal biopsy study (Table 3), C3 with IgG (n=61,61%) and isolated C3 (n=23,23%) were the most common immunofluorescence pattern seen. About four patients (n=4,4%) had IgA-dominant IRGN. Diffuse proliferative glomerulonephritis (DPGN) (n=63,63%) and endocapillary proliferative glomerulonephritis (n=22,22%) were the most common light microscopic finding Crescents were seen in twenty-eight (n=28,28%) patients, while crescentic glomerulonephritis (GN) was seen in eight (n=8,8%) patients. Thirteen out of the 27 diabetics had coexisting diabetic nephropathy. Steroids were used in fifteen of our patients.

The mean duration of hospital stay was 42.4 days.The mean follow-up was 31.4 weeks. Three patients died within 1 month of the onset of illness. All 3 patients were elderly males who presented with dialysis requiring renal failure. Two of them were diabetics with diabetic foot syndrome and coronary artery disease and died of sepsis/septic shock. Steroids were not given to them. Steroids were given to the other non-diabetic who presented with crescentric GN; nil identifiable source of infection. He did not respond to steroids and subsequently developed CRBSI/sepsis/septic shock and expired.

Table 1. Demographic Factors

Demographic Factors	
Male : Female	56:44
Age in years	**No. of patients (%)**
<20	16 (16%)
20-40	28 (28%)
40-60	45 (45%)
>60	11 (11%)
Co-morbidities	**No. of patients (%)**
Diabetes mellitus	27 (27%)
Alcoholism	13 (13%)
Pre-existing Hypertension	6 (6%)
Smoking	11 (11%)
Pre-existing chronic kidney disease	7 (7%)

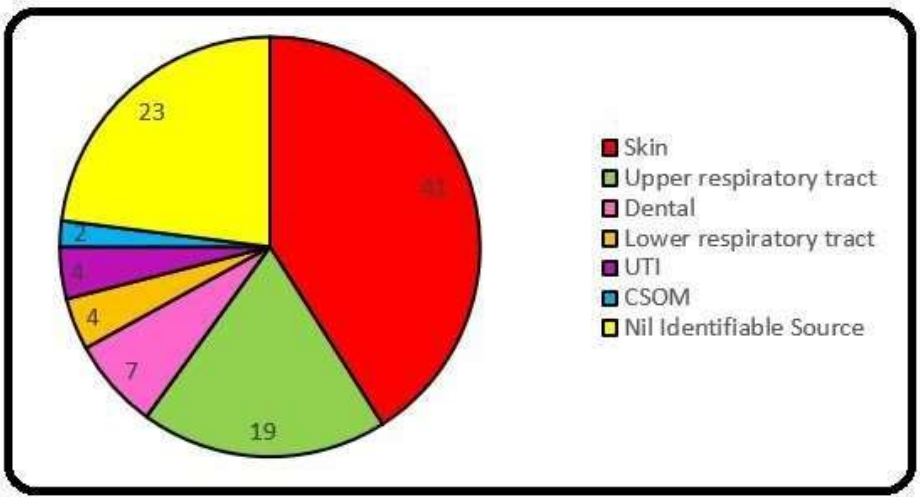


Figure 1. Source of infection

UTI-Urinary Tract Infection, CSOM-Chronic Suppurative Otitis Media

Table 2. Clinical Presentation

Clinical presentation	**No. of patients (%)**
New onset hypertension (>140/90 mm hg)	82 (82%)
Renal failure (Sr. creatinine >1. 2 mg/dl)	69 (69%)
Serum creatinine 1. 2-4 mg/dl	39 (39%)
Serum creatinine >4	30 (30%)
Dialysis requirement	23 (23%)
Acute Nephritic syndrome	57 (57%)
Renal failure with Nephrotic proteinuria	22 (22%)
Renal failure with sub-nephrotic proteinuria	6 (6%)

Rapidly progressive renal failure	10 (10%)
Nephrotic syndrome	5 (5%)
Macrohaematuria	32 (32%)
Nephrotic proteinuria (urine protein >3.5 mg/dl)	38 (38%)
Low C3 and low C4	8 (8%)
Low C3 and normal C4	74 (74%)

Table 3. Renal Biopsy Features

Renal biopsy features	**No. of patients (%)**
Light microscopic features	
Diffuse proliferative glomerulonephritis	63 (63%)
Endocapillary proliferative Glomerulonephritis	22(22%)
Crescentric Glomerulonephritis (>50% glomeruli with crescents)	8(8%)
Mesangioproliferative Glomerulonephritis	7(7%)
Crescents	28 (28%)
Co-existing Diabetic nephropathy	13 (48.14%)
Moderate IFTA*	7 (7%)
Severe IFTA*	3 (3%)
Immunofluorescence study	**No. of patients (%)**
c3+IgG c3 alone c3+IgG+IgM c3+IgA c3+IgG+c1q	61 (61%) 23 (23%) 11 (11%) 4 (4%) 1 (1%)

*IFTA-interstitial fibrosis and tubular atrophy

Table 4. Outcome of Adults with IRGN

Outcome	**No. of patients (%)**
Death	3 (3%)
Chronic kidney disease (e-GFR<60 ml/min/1.73 m^2)	34 (35%)
Dialysis dependant	9 (9. 27%)
Persistent hypertension	26 (26.80%)
Persistent proteinuria	32 (32.98%)

Table 5. Factors Associated with Progression to CKD

Factors	**Normal renal function at end of 6 months (n=63)**	**CKD (e-GFR <60 ml/min/1. 73 m^2) at 6 months (n=34)**	**pvalue (<0.05)**	**Odds ratio**
Age >40	26 (41.26%)	27 (79.41%)	0.0005	5.489 (2.0789 to 14.4929)
Males	32 (50.79%)	21 (61.76%)	0.39	1.5649 (0.6689 to 3.6611)
Diabetes Mellitus	10 (15.87%)	15 (44.11%)	0.0035	4.1842 (1.6077 to 10.8900)
Alcoholism	7 (11.11%)	5 (14.70%)	0.75	1.3793 (0.4023 to 4.7291)
Underlying CKD	1 (1.58%)	5 (14.70%)	0.0191	10.6897 (1.1941 to 95.6944)
Presenting creatinine >4	15 (23.80%)	12 (35.29%)	0.24	1.7455 (0.7015 to 4.3429)
Crescentric GN	1 (1.58%)	5 (14.70%)	0.0191	10.6897 (1.1941 to 95.6944)
Crescents	15 (23.80%)	13 (38.24%)	0.1619	1.9810 (0.8034 to 4.8848)
IgA dominant IRGN	3 (4.76%)	1 (2.94%)	1.0	0.6061 (0.0606 to 6.0615)
Moderate To Severe IFTA	2 (3.17%)	6 (17.64%)	0.0206	6.5357 (1.2406 to 34.4318)
HD requirement at presentation	4 (6.35%)	16 (47.05%)	0.00001	13.1111 (3.8857 to 44.2394)
Diabetic Nephropathy	7 (11.11%)	5 (14.70%)	0.75	1.3793 (0.4023 to 4.7291)

At the end of 6 months, out of the 97 surviving patients, 34 (n=34,35%) patients had persistent renal dysfunction,CKD (eGFR by CKD-EPI <60ml/per/1.73 m^2)) and 9 (n=9,9.27%) were dialysis dependant. (Table 4). Age >40 years, associated diabetes mellitus,underlying CKD, dialysis requiring renal failure at presentation, the presence of crescentric GN and moderate-to-severe IFTA in renal biopsy were identified as risk factors for the development of CKD by univariate analysis (Table 5).

Discussion

The classical post-streptococcal glomerulonephritis comprises of an initial infectious episode, usually a pharyngitis or a skin infection followed by a symptom-free period of 2 to 3 weeks and an acute nephritic syndrome; usually affecting children and young adults [4,5]. Over the past few decades, there has been a shift in the epidemiology of this disease. Its prevalence among adults has been increasing over the past few decades in developing countries like India [2]. Also, we could evidence that the mean or the peak age of presentation of adult

IRGN in India has increased over the years. Most recent studies have observed peak incidence in those above >40 years of age [6-8] similar to our study.

A bimodal distribution of cases with respect to age, with peaks in the second and fourth to fifth decades was observed in a study from south India [7]. In our study, the peak incidence in females was at 20-40 years of age and among males was 40-60 years.

The prognosis of post-infectious glomerulonephritis among elderly patients more than 60 years of age is poor, with 44% progressing to CKD and 33% progressing to end stage renal disease [9]. Also, biopsy studies from South India have reported IRGN as the common renal biopsy finding in the elderly and the very elderly thereby contributing to renal morbidity in the elderly and very elderly [10]. A single-centre observational study from South India reported 10% of patients to be above 60 years of age [7]. In our study, 11% were above 60 years of age.

There was a male predominance in our study similar to the observation in most other studies [9,11-14].

About one-third of adults with post-infectious glomerulonephritis have been found to have one or more comorbidities in recent studies [12]. This is in contrast to the earlier reports published, in which most of the affected patients had no notable medical history [15,16].

The various co-morbidities that have been identified are diabetes mellitus, alcohol intake, malignancy, prosthetic heart valve, intravenous drug use and retroviral disease [9,12]. Among these, most studies have observed diabetes mellitus and alcoholism [6,7,17] as the most common co-morbidities similar to our study.

The various sites of infection identified to be associated with IRGN in adults include the upper respiratory tract, skin, lung, heart, urinary tract, oral cavity, bone and deep-seated visceral or somatic abscess [3] out of which, the upper respiratory tract and skin are the most common sites [3,6,7]. As per literature evidence, skin has been found to be the most common site in the elderly and diabetics [9], in contrast to dental infection in those consuming alcohol [17]. An infective source was identified in 77% of our patients and the most common source was skin and soft tissues

Though acute nephritic syndrome is the most common presentation in adult IRGN [3,7,18], they can also present with rapidly progressive renal failure and nephrotic syndrome, unlike children. Also a significant number of adult patients present with dialysis requiring renal failure at presentation [6,7,9,13,18].

The majority of our patients presented with acute nephritic syndrome, classically defined by the occurrence of haematuria, proteinuria, edema often with hypertension and a mild degree renal impairment occurring in temporal relation. Edema and proteinuria were universally present in our patients. New onset hypertension was present in 82% of patients and macrohaematuria in 32% of patients.

Nephrotic-range proteinuria was rarely observed in earlier literature [19]. However nephrotic range proteinuria has been a frequent presentation in recent studies ranging from 13.8% to as high as 60% in a few studies [6,13]. In our study, nephrotic proteinuria was seen in 38% of our patients.

Nephrotic syndrome is a rare presentation in adults [6,13] and was observed in 5% of our patients.

The reported incidence of RRT requirement in Indian studies has been high in recent years, ranging from 17.5 to 35.6% [6,7,13] in contrast to earlier studies [14]. In

our study, 23% required RRT initiation at presentation.

Hypocomplementaemia occurs in 35–80% of adults with IRGN [9,12,20]. In our study, hypocomplementaemia (low C3 with or without low C4) was observed in 82% of patients.

The diverse organisms reported to be associated with postinfectious glomerulonephritis have been increasing over recent years namely group A streptococci, groups C and G streptococci, staphylococci, gram-negative bacilli, mycobacteria, parasites, fungi, and various viruses [20]. However, the predominant association is with staphylococcus and gram-negative bacteria [9].

In our study, Streptococcus and Staphylococcus were the most common isolated organisms.

Though IRGN has been classically expressed following bacterial infections, there are reports of IRGN following viral infections [21]. One of our patients had IRGN in association with covid-19 infection [22] which is rare in literature [23].

The glomerular picture in IRGN can vary depending on the characteristics of the host and invading organism. The different pathological features, include diffuse proliferative glomerulonephritis (DPGN), endocapillary proliferative GN, crescentric GN, mesangioproliferative GN and rarely membranoproliferative GN [12]; the most common being diffuse proliferative glomerulonephiritis [6,12,14]. We had similar observations in our study. DPGN and endocapillary proliferative glomerulonephritis were the most common histopathologies observed in our study. Mesangioproligerative and crescentric GN were observed in a few patients.

In immunofluorescence study, granular deposition of complement C3 is observed commonly, often with IgG and occasionally with IgM and rarely IgA. Isolated C3 and C3 with IgG were the common IF findings in our study.

IgA-dominant IRGN is rare and it usually occurs in association with diabetes and in those with staphylococcal infection [12] and carries a poor prognosis [24]. We observed IgA-dominant IRGN in 4 of our patients.

Treatment of acute postinfectious glomerulonephritis has been predominantly supportive considering the benign course of the disease and that the disease resolves with tthe eradication of the infection source. However, current insight into the pathophysiology of IRGN is such that the cascade of glomerular inflammatory events continues despite removal of the triggering insult especially in those with glomerulonephritis secondary to Staphylococcus aureus [12]. In these situations, treatment with corticosteroids or cytotoxic agents might help [25,26]. A single-center randomized control trial from south India reported that the use of corticosteroids in patients with IRGN and serum creatinine >1.5 mg/dl did not increase the rate of complete renal recovery at 6 months and that the infectious complications occurred more often in the steroid arm [27]. However, multicentric randomized clinical trials are needed to test the usefulness of immunosuppressants in IRGN.

In our study, we restricted the usage of corticosteroid to patients who presented with dialysis-dependant renal failure and/or in those with crescents in renal biopsy after ruling out ongoing infection with a meticulous search for all possible active sources of infection in each patient.

The prognosis of IRGN in children especially those occurring as epidemics has

an excellent prognosis [28,29] in contrast to adult and sporadic IRGN [15,30]. The prognosis has been found to be worse in the elderly and those with co-morbidities [9].

In our study 35% had persistent renal dysfunction (CKD) at the end of 6 months and 9.27% of patients were dialysis dependent at the end of 6 months.

Various poor prognostic factors have been enlisted in IRGN that can predict the progression to CKD [11,14]. In our study, age >40 years, diabetes mellitus, underlying CKD, dialysis requiring renal failure at presentation, crescentric GN and moderate to severe IFTA were identified as risk factors by univariate analysis.

The strengths of our study are the inclusion of only biopsy-proven cases and prospective follow-up for 6 months. However limitations include short follow-up period and not excluding cases of C3 GN with repeat complement levels during follow -up.

Conclusion

This study highlights the changing trend in the epidemiology of this disease;the increasing significance of this disease as an important cause of CKD/ESRD in adults especially in low-income countries and the risk factors associated with the progression to CKD/ESRD. Because of the increasing incidence and poor prognosis, it is important for the physician to be aware of this emerging entity and the importance of early identification and eradication of the infection source. This study also emphasizes the need for future research on the usefulness of immunusupressive therapy in adult IRGN.

Conflicts of interest

The authors declare that they do not have any conflict of interest.

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References

1. Nadasdy T, Hebert LA. Infection-related glomerulonephritis: Understanding mechanisms. Semin Nephrol 2011;31:369–75.
2. Narasimhan B, Chacko B, John GT, Korula A, Kirubakaran MG, Ja ob CK. Characterization of kidney lesions in Indian adults: towards a renal biopsy registry. J Nephrol. 2006 Mar-Apr;19 (2):205-10. PMID: 16736422.
3. Nasr SH, Radhakrishnan J, D'Agati VD. Bacterial infection-related glomerulonephritis in adults. Kidney Int 2013;83:792–803.
4. Rammelkamp CH, Weaver RS. Acute glomerulonephritis: The significance of the variation in the incidence of the disease. J Clin Invest 1953;32:346-58.
5. Poon-King T, Mohammed I, Cox R, et al. Recurrent epidemic nephritis in SouthTrinidad. N Engl J Med 1967;277:728-33.
6. S. Arivazhagan, D. Rajasekar, N. Gopalakrishnan, R. Sakthirajan, J. Dhanapriya, T. Dinesh Kumar, T. Balasubramanian, N. Malathy. Clinicopathological profile and outcome of adult infectionrelated glomerulonephritis: A prospective follow-up study. Natl Med J India 2000;33:260–4.
7. Sanathkumar HT, Fernando EM, Kurien AA, Srinivasaprasad ND, Suren S, Thirumalvalavan K. Clinical profile, histopathology, and outcomes in infection-related glomerulonephritis – single-center experience. Indian J Nephrol 2022;32:546-54.
8. Clinicopathological Profile Of Patients With Infection Related Glomerulonephritis In A Tertiary Care Centre. Elsa Thomas, Aneeb Raj V. P, Renny Issac, Usha Nagadevi C S. Int J Acad Med Pharm 2023; 5 (1); 103-107.

9. Postinfectious Glomerulonephritis in the Elderly Samih H. Nasr, Mary E. Fidler,Anthony M. Valeri,Lynn D. Cornell,Sanjeev Sethi,Amy Zoller,Michael B. Stokes,Glen S. Markowitz, and Vivette D. D'Agati. J Am Soc Nephrol 22: 187–195, 2011.
10. Infection-related glomerulonephritis is the most common finding in renal biopsies in the very elderly in India Anila A. Kurien1, K. S. Jansi Prema1 and Natarajan Gopalakrishnan2 Clinical Kidney Journal, 2021, vol. 14, no. 1, 454–456.
11. Moroni G, Pozzi C, Quaglini S, Segagni S, Banfi G, Baroli A, et al. Long-term prognosis of diffuse proliferative glomerulonephritis associated with infection in adults. Nephrol Dial Transplant 2002;17:1204–11.
12. Nasr SH, Markowitz GS, Stokes MB, Said SM, Valeri AM, D'Agati VD, et al. Acute postinfectious glomerulonephritis in the modern era: Experience with 86 adults and review of the literature. Medicine (Baltimore) 2008;87:21–32.
13. The epidemiology, clinical features, and outcome of infection-related glomerulonephritis from East India: A single center experience. Trivedi M, Pasari A, Chowdhury AR, Kurien AA, Pandey R. Indian J Nephrol 2017;27:307-12.
14. Follow-Up Study of Post-Infectious Glomerulonephritis in Adults: Analysis of Predictors of Poor Renal Outcome Gopalakrishnan Natarajan, Sakthirajan Ramanathan, Dhanapriya Jeyachandran, Balasubramaniyan T., Srinivasa Prasad N. D., Dineshkumar Thanigachalam. Saudi J Kidney Dis Transpl 2014;25 (6):1210-1216.
15. Baldwin, D. s., Gluck, M. C., schacht, r. G. &Gallo, G. The long-term course ofpoststreptococcal glomerulonephritis. Ann. Intern. Med. 80, 342–358 (1974).
16. Lien, J. w., Mathew, T. H. & Meadows, r. Acute post-streptococcal glomerulonephritis in adults: a long-term study. Q. J. Med. 48, 99–111 (1979).
17. Keller CK, Andrassy K, Waldherr R, Ritz E. Postinfectious glomerulonephritis –Is there a link to alcoholism? Q J Med 1994;87:97–102.
18. Aditya, S, Bhushan, S, Mythri, S, Kishan, A, Gireesh, R, Ranjitha, S, Viswas, T, Gouri, S, Sravani, V, Sreedhara, CG. Institute of Nephrourology, Nephrology, Bangalore, India Kidney International Reports (2022) 7, S463–S530.
19. Sarkissian, A. et al. An epidemic of acute postinfectious glomerulonephritis in Armenia. Arch. Dis. Child. 77, 342–344 (1997).
20. Montseny, J. J., Meyrier, A., Kleinknecht, D. & Callard, P. The current spectrum of infectious Glomerulonephritis, experience with 76 patients and review of the literature. Medicine (Baltimore) 74, 63–73 (1995).
21. Komatsuda, A. et al. endocapillary proliferative glomerulonephritis in a patient with parvovirus B19 infection. Am. J. Kidney Dis. 36, 851–854 (2000).
22. Gandhi TS, Angusamy S, Jeyachandran D, Ramudu V, Thopalan B. Infection-related glomerulonephritis with reactive arthritis in a patient with COVID-19 infection. Indian J Kidney Dis 2022;1:22-5.
23. Pérez A, Torregrosa I, D'Marco L, Juan I, Terradez L, Solís MÁ, Moncho F, Carda-Batalla C, Forner MJ, Gorriz JL. IgA-Dominant Infection-Associated Glomerulonephritis Following SARS-CoV-2 Infection. Viruses. 2021 Mar 31;13 (4):587.
24. Dhanapriya J, Balasubramaniyan T, Maharajan SP, Dineshkumar T, Sakthirajan R,Gopalakrishnan N, et al. IgA-dominant infection-related glomerulonephritis inIndia: A single-center experience. Indian J Nephrol 2017;27:435–9.
25. el-Husseini, A. A., sheashaa, H. A., sabry, A. A.,Moustafa, F. e. & sobh, M. A. Acute postinfectious crescentic glomerulonephritis: clinicopathologic presentation and risk factors. Int. Urol. Nephrol. 37, 603–609 (2005).
26. Raff, A., Hebert, T., Pullman, J. & Coco, M. Crescentic post-streptococcal glomerulonephritis with nephrotic syndrome in the adult: is aggressive

therapy warranted? Clin. Nephrol. 63, 375–380 (2005).
27. Srinivasan Arivazhagan et al. Efficacy of Corticosteroids in Infection-Related Glomerulonephritis–A Randomized Controlled Trial. Kidney Int Rep (2022) 7, 2160–2165.
28. Lien, J. w., Mathew, T. H. & Meadows, r. Acute post-streptococcal glomerulonephritis in adults: a long-term study. Q. J. Med. 48, 99–111 (1979).
29. Berrios, X. et al. Post-streptococcal acute glomerulonephritis in Chile—20 years of experience. Pediatr. Nephrol. 19, 306–312 (2004).
30. Hinglais, N., Garcia-Torres, R. & Kleinknecht, D. Long-term prognosis in acute glomerulonephritis. The predictive value of early clinical and pathological features observed in 65 patients. Am. J. Med. 56, 52–60 (1974).