Original Article

Histomorphologic Pattern of Renal Disease in Patients with Acute Nephritic syndrome: A Single Centre South Indian Study

Clement Wilfred Devadass[1]*, VijayaMysorekar V[1], GireeshSiddaiah M[2], Mahesh E[2] and GurudevChannabasappa K[2]

[1]Department of Pathology, MS Ramaiah Medical College And Hospitals. MSRNagar, Bangalore, India.
[2]Department of Nephrology, MS Ramaiah Medical College and Hospitals. MSRNagar, Bangalore. India

Keywords: *Acute Nephritic Syndrome, Glomerulonephritis, Post Infectious Glomerulonephritis, Renal Biopsy.*

ABSTRACT

Background: The frequency and distribution of primary and secondary glomerular disorders presenting as acute nephritic syndrome (ANS) varies according to the geographic and racial characteristics, renal biopsy practices and patient selection criteria. The present study was carried out to determine the histomorphologic patterns of lesions in renal biopsies from patients presenting with ANS.

Methods: The study was conducted on patients presenting with ANS, in a tertiary care hospital in South India, during the period between 2008 and 2015. The renal biopsies performed were studied by light and immunofluorescence microscopy.

Results: A total of 112 patients of ANS, with a male: female ratio of 1: 1.5 and mean age of 32.9 ±16.85 years, were included. Primary glomerular disease (PGD) was present in 77.7% and secondary glomerular disease (SGD) in 22.3% of the cases. The commonest PGD presenting as ANS was postinfectious glomerulonephritis followed by crescentic glomerulonephritis. The commonest SGD causing ANS was lupus nephritis (LN).

Conclusion: A range of PGD and SGD can present as ANS. Various studies, done in India and abroad, show variations in the frequency and distribution of PGD presenting as ANS. The distribution pattern of SGD presenting as ANS largely corresponds to the pattern described in other Indian studies with LN being the commonest.

***Corresponding author:**
Dr Clement Wilfred D, Associate Professor, Department of Pathology, M.S Ramaiah Medical College and Hospitals, MSRIT Post, MSRNagar, Bangalore- 560060, INDIA
Phone: +91 9945226314
Email: clement.wilfred@yahoo.com

This work is licensed under the Creative Commons Attribution 4.0 License. Published by Pacific Group of e-Journals (PaGe)

Introduction

Acute nephritic syndrome (ANS) is one of the presentations of renal disease that is characterised by hematuria, subnephrotic proteinuria and fall in glomerular filtration rate with azotemia, hypertension, oliguria and edema.[1,2] Varied histopathologic lesions, especially affecting the glomeruli, can cause the syndrome.[1,3] A meticulous histopathologic examination of renal biopsy and clinical correlation is required to distinguish between the various causes of ANS, as they exhibit different clinical behaviour and require different treatment protocols. Early recognition of the underlying cause of ANS and prompt institution of relevant therapy is vital for improvement in or preservation of renal function.[4]Further, the lesions causing ANS vary according to geographic area, socioeconomic conditions, demography and race, and there is paucity of this information in the native south Indian population.[5]

The aim of the present study was to determine the histomorphologic patterns of lesions in renal biopsies from patients presenting with ANS in a tertiary care hospital in south India.

Material and Methods

The study was conducted in the department of Pathology in conjunction with the department of Nephrology, M.S Ramaiah Medical College and Hospitals, Bangalore; over a duration of seven years (between 2008 and 2015) and was a cross-sectional, hospital based study. Patients presenting with ANS, who underwent percutaneous renal biopsy were included in the study. ANS was defined as rapid onset of edema, oliguria and hypertension with hematuria (microscopic or macroscopic) and mild to moderate proteinuria (< 3.5 g per day per 1.73m^2 surface area).[1,3]Patients with nonglomerular/ urothelial/ urological hematuria caused by interstitial diseases, urolithiasis and trauma, benign or malignant mass lesions and infections of the urinary tract were excluded from the study.[6]The renal biopsy specimens were processed for light microscopy (LM) and immunofluorescence microscopy (IFM) as perstandard protocol. For LM, 3 to 4 µm thick paraffin embedded tissue sections were stained with haematoxylin and eosin (H&E), periodic acid Schiff (PAS), Jones silver methenamine (JMS) and Masson's trichrome stains. For IFM, cryo-sections were stained with fluorescein isothiocyanate (FITC) conjugated antisera (Bio Genex) specific for immunoglobulin IgG, IgM, IgA and complement component 3 (C3). The staining distribution was described as mesangial or membranous in a granular or linear pattern and the staining intensity was graded semi-quantitatively from 0 to +++. The clinical data and relevant investigations, including age, gender, urinalysis, 24 hour proteinuria, serum creatinine, blood urea nitrogen levels and serological data [complement levels (C3 and C4), anti-nuclear antibodies, dsDNA and ASLO titers] were retrieved from the patients' case files. The renal diseases were classified into primary glomerular disease (PGD) and secondary glomerular disease (SGD).

The various PGD and SGD, presenting as ANS, were diagnosed on the basis of both clinical and histopathological (LM and IFM) investigations. The following criteria were used to diagnose the various disease entities: Postinfectious glomerulonephritis (PIGN) was diagnosed when acute glomerulonephritis, characterised by diffuse global glomerular endocapillary proliferation with neutrophilic exudation on LM and coarse granular predominantly membranous and a less prominent mesangial deposit of C3 and IgG on IFM, was present related to recent streptococcal / nonstreptococcal infection.[7]

Crescentic glomerulonephritis (CresGN) was diagnosed when 50% or more of the glomeruli showed crescent formation on LM.[8] In addition "anti-glomerular basement CresGN" was diagnosed when linear IgG and C3 staining of glomerular basement membrane was discerned on IFM (usually with associated elevated anti-glomerular basement membrane antibody titer); "immune complex CresGN" was diagnosed when glomerular deposits of immunoglobulins and or complement were clearly present on IFM; "pauci-immune CresGN" was diagnosed when little or no immunoglobulin and complement deposits were discerned on IFM (usually with presence of antineutrophil cytoplasmic antibodies).[8,9] Goodpasture syndrome (GPS) was diagnosed when features of anti-glomerular basement CresGN was present with pulmonary involvement (pulmonary haemorrhage).[8] Membranoproliferative glomerulonephritis (MPGN) was diagnosed when lobular hypercellular glomeruli with thickened capillary walls (with " tram tracks" on PAS/ JMS stains) and increased mesangial substance was present on LM, and membranous and mesangial deposits of C3 with or without accompanying immunoglobulin deposits was present of IFM with clinical finding of hypocomplementemia.[10] IgA nephropathy (IgAN) was diagnosed when glomerulonephritis with predominantly IgA deposits in the mesangium was present in the absence of systemic disease.[11] Membranous nephropathy (MN) was diagnosed when diffuse global uniform thickening of glomerular capillary wall was present on LM (with subepithelial "spikes" in PAS/ JMS) and diffuse fine granular membranous deposits of IgG, and to a lesser extent C3 deposits were seen on IFM.[12] Focal and segmental glomerulosclerosis (FSGS) was diagnosed when sclerosis with associated synechiae formation and hyalinosis was present, involving the glomeruli in a focal and segmental fashion.[13]

Lupus nephritis (LN) was diagnosed when glomerular mesangial matrix expansion/ mesangial proliferation/ capillary wall thickening/ endocapillary proliferation/ necrosis/ crescents were present in the appropriate clinical setting (i.e patients fulfilling the American college of Rheumatology revised criteria for classification of Systemic lupus erythematosus), usually with "full house" mesangial or membranous deposits of immunoglobulins and complement.[14] Henoch-Schönlein purpura (HSP) was diagnosed when glomerular IgA immune complex deposits were present with extrarenal manifestations such as purpura.[15] Hemolytic uremic syndrome (HUS) was diagnosed when histological evidence of glomerular/ arteriolar thrombotic microangiopathy (fibrinoid necrosis/ thrombosis) was present in the clinical setting of microangiopathic hemolytic anemia.[16] Malignant nephrosclerosis was diagnosed when histological features of arterio/ arteriolonephrosclerosis was present in the clinical setting of accelerated hypertension, papilledema and retinal hemorrhage.[17]

Statistical Analysis: Data was analysed using Statistical Package for Social Sciences version 20.0, (SPSS, IBM, USA). All the continuous parameters were expressed as mean and standard deviation and all qualitative data as proportion. The frequency and percentage of each type of renal disease was determined.

Results

A total of 112 cases were included in the study of which 44 were males and 68 were females (male: female ratio = 1: 1.5). The distribution of sex in each decade is shown in Figure 1. The mean age of the patients was 32.9 ±16.85 (range 5-75) years. Majority of the patients were in the second (23.2%, 26/112) and third (21.4%, 24/112) decades. The mean 24 hour proteinuria and serum creatinine were 2.27±0.68 (range: 1.0- 3.3) and 2.91±2.35 (range: 0.7-13.7) respectively.

The frequencies and histologic patterns of renal lesions and some basic data in patients with ANS, are shown in Table 1. PGD was present in 77.7% (87/112) and SGD in 22.3% (25/112) of the cases. The commonest PGD presenting as ANS was postinfectious glomerulonephritis (PIGN) (37.9%, 33/87), followed by crescentic glomerulonephritis (CresGN) (23%, 20/87) (Fig 2). The commonest SGD causing ANS was lupus nephritis (LN) (68%, 17/25) (Fig 3). The distribution of renal disease in each decade is shown in Table 2 and the immunofluoresence findings in the different histomorphological lesions of ANS are depicted in Table 3.

Table 1: Histologic patterns of renal disease and laboratory parameters.

Histological diagnosis	Number of cases (%)	Mean age ± SD	Males	Females	Male: Female ratio	Serum creatinine mg/dl ± SD	24 hour proteinuria g/ day ± SD
Primary glomerular disorders							
PIGN	33 (29.5)	33.7±19.74	16	17	1:1.1	2.26±1.08	1.92±0.62
CresGN	20(17.9)	36.2±17.0	9	11	1:1.2	5.18±3.61	2.21±0.63
MPGN	17 (15.2)	33.8±13.84	8	9	1:1.1	2.16±1.6	2.37±0.74
IgAN	14 (12.5)	31.4±13.41	8	6	1.3:1	3.13±2.71	2.67±0.64
MN	2 (1.8)	32.5±3.53	1	1	1:1	1.3±0.84	3.1±0.42
FSGS	1 (0.9)	65	-	1	-	2.2	2.9
Secondary glomerular disease							
LN	17 (15.2)	27.1±12.89	-	17	-	2.39±1.56	2.52±0.68
MNS	2 (1.8)	27±5.65	1	1	1:1	5.1±.14	2.45±0.78
HUS	2 (1.8)	8.5±4.94	1	1	1:1	2.75±0.91	1.7±0.28
HSP	2 (1.8)	24.5±19.09	-	2	-	2.05±0.07	2.25±0.35
GPS	1 (0.9)	65	-	1	-	1.9	1.8
Vasculitis	1(0.9)	60	-	1	-	1.4	1.9
Total	**112**	**32.9±16.85**	**44**	**68**	**1:1.5**	**-**	**-**

CresGN (Crescentic glomerulonephritis), FSGS (Focal and segmental glomerulosclerosis), GPS (Goodpasture syndrome), HSP (Henoch-Schönlein purpura), HUS (Hemolytic uremic syndrome), IgAN (IgA nephropathy), LN (Lupus nephritis), MN (Membranous nephropathy), MNS (Malignant nephrosclerosis), MPGN (Membranoproliferative glomerulonephritis), PIGN (Postinfectious glomerulonephritis).

Table 2: Distribution of renal disease in each decade.

Disease	≤10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
PIGN	4	10	1	7	6	1	3	1	33
CresGN	1	4	3	3	6	2	-	1	20
MPGN	-	3	5	4	2	3	-	-	17
IgAN	-	2	5	4	1	2	-	-	14
MN	-	-	1	1	-	-	-	-	2
FSGS	-	-	-	-	-	-	1	-	1
LN	-	5	8	1	3	-	-	-	17
MNS	-	-	1	1	-	-	-	-	2
HUS	1	1	-	-	-	-	-	-	2
HSP	-	1	-	1	-	-	-	-	2
GPS	-	-	-	-	-	-	1	-	1
Vasculitis	-	-	-	-	-	1	-	-	1
Total	**6**	**26**	**24**	**22**	**18**	**9**	**5**	**2**	**112**

Table 3: Immunofluorescence findings in the various histomorphological lesions of ANS.

Histological diagnosis	IgG	IgM	IgA	C3
PIGN (N=33)	33	10	5	33
CresGN (N=20)	12	4	3	8
MPGN(N=17)	12	8	0	17
IgAN(N=14)	7	4	14	8
MN(N=2)	2	0	0	1
FSGS(N=1)	0	1	0	1
LN(N=17)	17	12	10	16
MNS(N=2)	0	0	0	0
HUS(N=2)	0	0	0	0
HSP(N=2)	1	0	2	1
GPS(N=1)	1	0	0	0
Vascuitis(N=1)	0	1	0	1
Total(N=112)	**85 (76%)**	**40 (36%)**	**34 (30%)**	**86 (77%)**

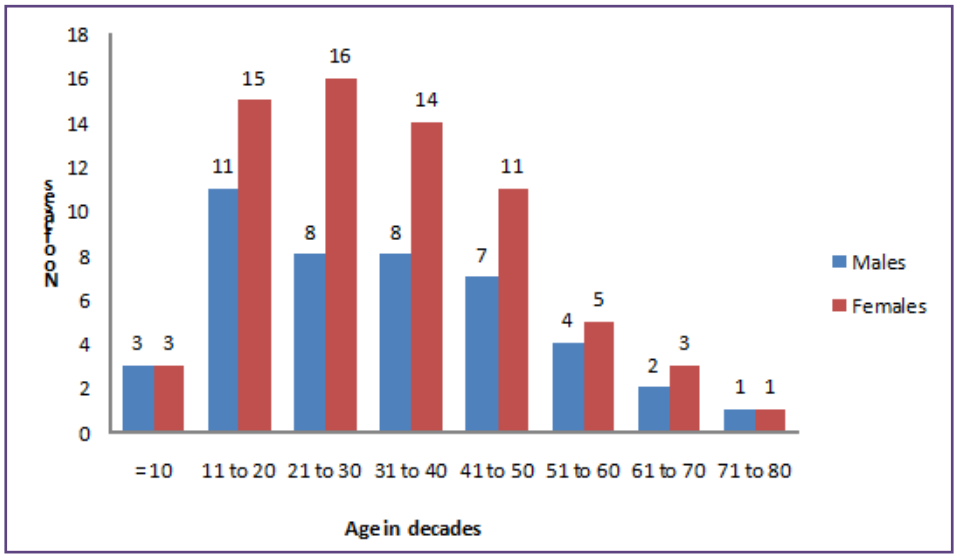

Fig. 1: Distribution of sex in each decade.

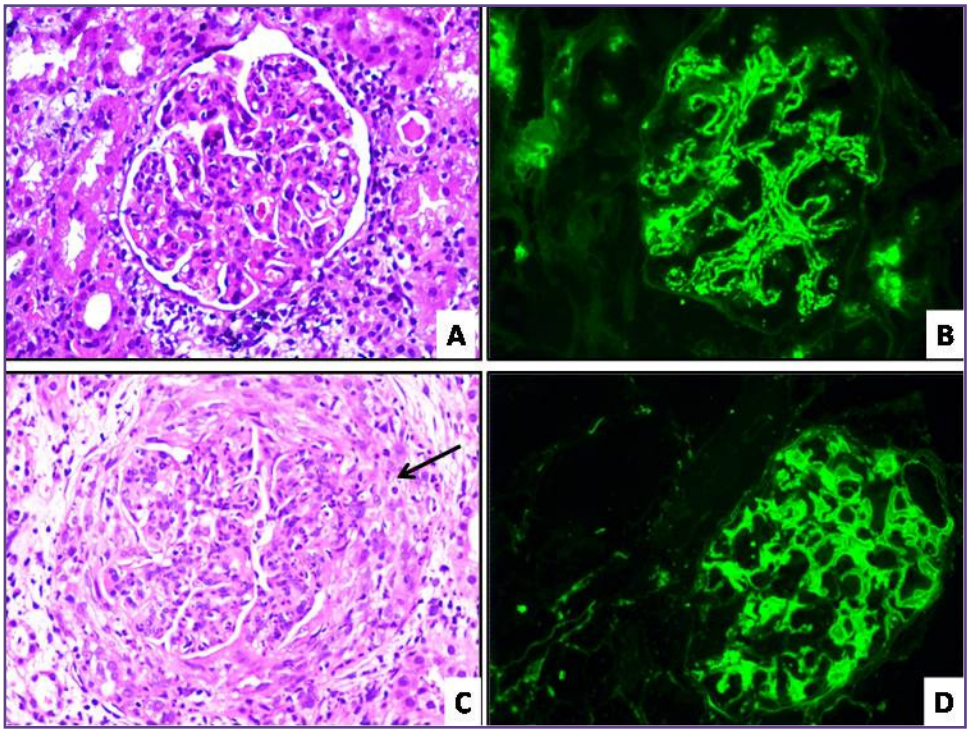

Fig 2: (A) Postinfectious glomerulonephritis with global glomerular endocapillary proliferation (H&E, x 200); (B) Postinfectious glomerulonephritis with predominantly membranous deposits of C3 (IFM, x200); (C) Immune complex CresGN with crescent (arrow) formation (H&E, x 200); (D) Immune complex CresGN with membranous deposits of IgG (IFM, x200).

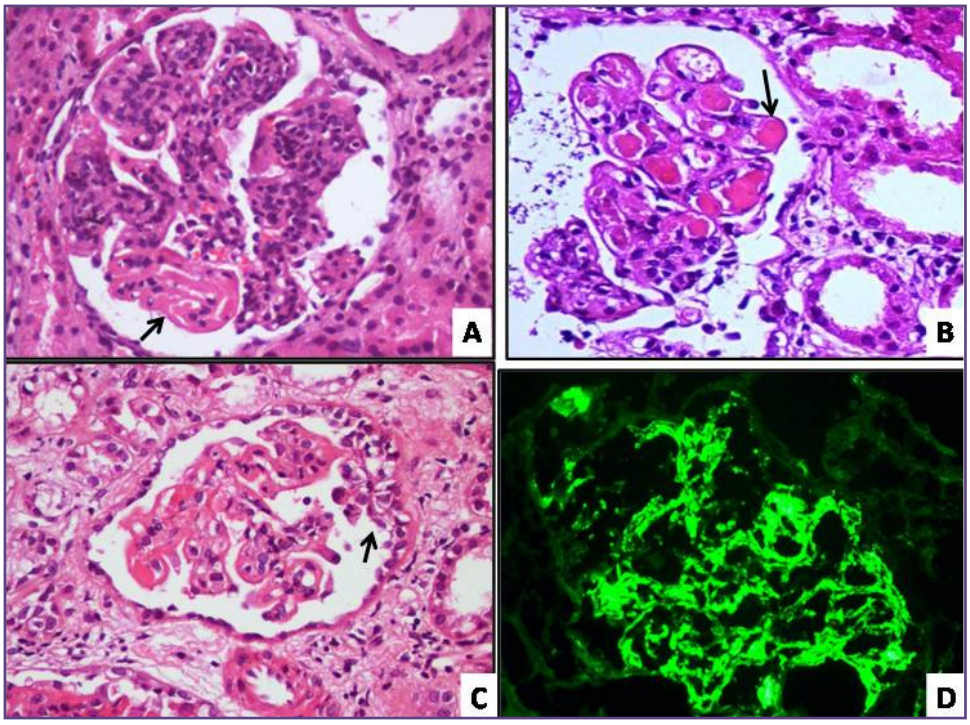

Fig. 3: (A) Lupus nephritis with glomerular endocapillary and mesangial proliferation and wire loop lesions (arrow) (H&E, x 200); (B) Lupus nephritis with glomerular capillary hyaline thrombi (arrow) (H&E, x200); (C) Lupus Nephritis with early crescent (arrow) formation (H&E, x 200); (D) Lupus Nephritis with membranous and mesangial deposits of IgG (IFM, x200).

Discussion

ANS is a glomerular syndrome that is characterised by inflammatory changes within the glomerulus.[6] The inflammatory changes are associated with complement activation, production of pro-inflammatory cytokines and mediators, infiltration by neutrophils and monocytes, and mesangial and endothelial cell proliferation with swelling which cause i) local hemodynamic alterations and reduced filtration area resulting in decreased GFR with consequent salt and water retention, oliguria, edema and varying degrees of hypertension ii) glomerular hematuria manifested by dysmorphic red blood cells (RBCs) and RBC casts and iii) damage to glomerular filtration barrier with resultant mild to moderate proteinuria.[6,18]A variety of PGD and SGD can present as ANS. The common PGD that present as ANS are PIGN, membranoproliferative glomerulonephritis (MPGN), IgA nephropathy (IgAN) and CresGN (rapidly progressive glomerulonephritis). The SGD that manifest as ANS include LN, Henoch-Schönlein purpura (HSP), hemolytic uremic syndrome (HUS), Goodpasture syndrome (GPS), bacterial endocarditis, shunt nephritis, hypersensitivity vasculitis, Wegener granulomatosis and polyarteritis nodosa.[6,19]

Our gender distribution of mild female preponderance was comparable to a study from Bangladesh (M:F= 1: 1.15), however mild male preponderance was found in a west Indian (Mumbai) study (M:F= 1.8:1).[4,20]LN frequently occurs in females and the higher relative frequency of this disease in our series compared to the latter study, may partly explain the female predominance. The age distribution was similar to other Indian studies.[4,20] Even though ANS can occur in any age group, it is most prevalent in children.[4] However in the present study only 18% (20/112) of the cases occurred in children (age ≤15 years). Similarly Rahman et al, in their study, observed that paediatric patients accounted for only 4% of the ANS cases.[4]This discrepancy could due to the fact that paediatric ANS cases generally respond well to therapy and therefore are seldom biopsied.[4]

The frequency and distribution of the various PGD and SGD presenting as ANS vary according to geographic area, race, socio-economic conditions, prevalence of infectious diseases and policies in renal biopsy practice. Table 4 shows the comparison of our data with a few other national and international published studies.

The most common cause of ANS in the present study was PIGN (29.5%) which is in synchrony with another south Indian study (Hyderabad) and Brazilian study, where PIGN constituted the commonest cause of ANS, accounting for 56.6% and 17.2 % of the cases respectively.[5,21] PIGN constituted the second and third commonest causes of ANS respectively in studies from west India (Mumbai) (25.7%) and Serbia (18.1%).[20,22] However, in a study from Bangladesh PIGN was the least common cause (2.7%).[4] The authors of the latter study attribute the low prevalence of the disease to improvement of personal hygiene and health care. Further, the authors commented that as PIGN cases resolve with treatment they are seldom biopsied. In our institution, we biopsy children with clinically suspected PIGN, only if persistent hypocomplementemia (> 6weeks) or gross hematuria (> 1month) or hypertension (> 2 months) or progressive decline in GFR or occurrence of ANS within 48 hours of pharyngitis is present. However we biopsy adults more liberally as PIGN is not as common in adults as in children and a number of glomerulonephritis may clinically masquerade as PIGN.

CresGN constituted the second most common cause of ANS (17.9%) which is similar to the study conducted in Serbia (21.7%).[22] This is in contrast to studies from south India (Hyderabad), west India(Mumbai), Bangladesh and Brazil where the disease was either muchless frequent or was not reported.[4, 5, 20, 21]The reasons for these variations in disease frequency, whether due to differences in patient selection criteria or differing environmental influences, is not clear. Further evaluation of our cases of CresGN, by IFM, revealed 5 cases (20%) of anti-glomerular basement CresGN, 7 cases (35%) of immune complex CresGN and 8 cases (40%) of pauci-immune CresGN. Similarly literature review showed that pauci-immune CresGN is the most frequent category of CresGN (60%) followed by immune complex CresGN(24%), with anti-glomerular basement CresGN (15%) being the least frequent.[8]

In the present study MPGN (15.2%) and LN (15.2%) constituted the third most frequent cause of ANS. MPGN (Fig 4) was the commonest cause of ANS in studies conducted in India (Mumbai) and Bangladesh unlike Serbian and Brazilian studies where low frequencies were reported.[4,20,21,22] The latter study attributed the low frequency of MPGN to improved socioeconomic conditions and decline in regional endemic diseases.[21]Similar to other Indian studies and a study from Bangladesh, LN was the single most common secondary cause of ANS. In the present study diffuse LN (class IV) was the most common histologic pattern (53%, 9/17) followed by focal LN (class III) (24%, 4/17), mesangial proliferative LN (class II) (12%, 2/17), membranous LN (class V) (6%, 1/17) and minimal mesangial LN (class I) (6%, 1/17). Similar to our observations, literature review reveals that ANS is a commoner manifestation of proliferative LN (classes III and IV) rather than classes I, II and V.[23]

IgAN (Fig 4) was the commonest cause of ANS in a Serbian study (35.7%). It is considered as the most common primary glomerular disease especially in young adult Caucasians and the most common cause of end-stage renal disease.[24]In the current study the frequency of IgAN presenting as ANS was 12.5%, which is slightly higher than the other Indian studies.[4,20]

Immunofluorescence studies in the cases presenting with ANS, showed deposits of single or multiple immunoglobulin and/ or complement component (C3). C3 deposits were the most frequent and were present in 77% of the cases followed by IgG deposits (76%) and IgM deposits (36%). Rahman et al in their study, observed that IgM was the predominant deposit (52%) followed by C3 (48%) and IgG (47%) deposits.[4] The differing immunofluorescence findings could be due to the differing patterns of glomerulonephritis causing ANS and technical factors inherent in the methodology.

Table 4: Comparison of our data with other published studies.

Variables	Present study	Bangladesh[4]	Mumbai[20]	Hyderabad[5]*	Serbia[22]	Brazil[21]**
No of ANS cases	112	73	74	166	83	789
PIGN	29.5	2.7	25.6	56.6	18.1	17.2
CresGN	17.9	0	0	3.6	21.7	3.1
MPGN	15.2	43.8	33.8	1.8	2.4	4.7
IgAN	12.5	5.5	2.7	4.8	35.7	15.8
MN	1.8	9.6	9.5	0	0	3.9
FSGS	0.9	0	0	2.4	4.8	10.2
LN	15.2	11	0	13.3	-	14.2
HUS	1.8	0	0	0	-	-
HSP	1.8	0	4.1	1.2	-	-

These figures represent percent of total cases of ANS.
** figures have been calculated from Table 2 of the article.*
*** data obtained from Table 7 of the article.*
- indicates that data are not available

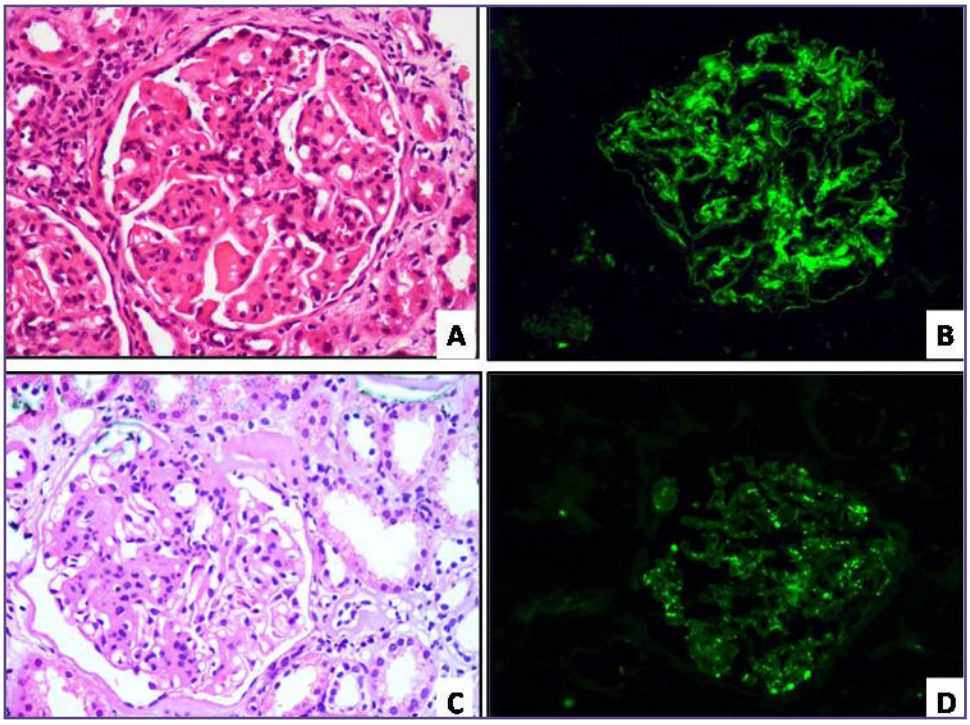

Fig. 4: (A) Membranoproliferative glomerulonephritis with lobular hypercellular glomeruli exhibiting thickened capillary walls (H&E, x 200); (B) Membranoproliferative glomerulonephritis with membranous and mesangial deposits of C3 (IFM, x200); (C) IgA nephropathy with mesangial hypercellularity (H&E, x 200); (D) IgA nephropathy with mesangial IgA deposis (IFM, x200).

Conclusion

Various studies, done in India and abroad, show variations in the frequency and distribution of PGD presenting as ANS. The reasons for this include varying renal biopsy practices, differing patient selection criteria, differences in the prevalence of infectious diseases and variations in the prevalence of various glomerulonephritis in different regions of the world. The commonest PGD presenting as ANS in the present study, was PIGN followed by CresGN. The distribution pattern of SGD presenting as ANS largely corresponds to the pattern described in other Indian studies with LN being the commonest.

The present study represents a contribution to understanding the epidemiology of renal disease manifesting as ANS in South India.

Acknowledgment

Nil

Funding

Nil

Competing Interests

Nil

References

1. Lewis JB, Neilson EG. Glomerular disease. In: Kasper DL, Hauser SL, Jameion LJ, Fauci AS, Longo DL, Loscalzo J, editors. Harrison's principles of Internal Medicine. 19thed. New York: MC Graw-Hill Education; 2015. p.1831-50.
2. Colvin RB editor. Introduction to Renal Pathology. In: Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.1-2.
3. Iseki K, Miyasato F, Uehara H, Tokuyama K, Toma S, Nishime K, et al. Outcome study of renal biopsy patients in Okinawa, Japan. Kidney International. 2004;66:914-9.
4. Rahaman MA, Kamal M, Rahman MS, Biswas MA, Sarker RA: Histomorphological Patterns of Glomerulonephritis in Patients Presenting with Acute Nephritic Syndrome Dinajpur Med Col J. 2012;5:26-33.
5. Das U, Dakshinamurthy KV, Prayaga A. Pattern of biopsy-proven renal disease in a single center of south India: 19 years experience . Indian J of Nephrol. 2011;21:250-7.
6. Emmett M, Fenves AZ, Schwartz JC. Approach to the patient with Kidney disease. In: Tall MW, Chertow GM, Marsden PA, Skorecki K, Yu ASL, Brenner BM, editors. Brenner and Rector's The Kidney, 9th ed. Philadelphia: Elsevier; 2012.p.844-67.
7. Satoskar AA, Nadasky T, Silva FG. Acute Postinfectious Glomerulonephritis and Glomerulonephritis caused by persistent Bacterial Infection. Chapter 10. In: Jennette JC, Olson JL, Silva FG, D'Agati VD, editors. Hepinstall's Pathology of the Kidney, 7th ed.. Lippincott Williams & Wilkins, Philadelphia. 2015;1:367-436.
8. Jennette JC, Nickeleit V. Anti-Glomerular Basement Membrane Glomerulonephritis and Goodpasture Syndrome. Chapter 15. In: Jennette JC, Olson JL, Silva FG, D'Agati VD, editors. Hepinstall's Pathology of the Kidney, 7th ed. Lippincott Williams & Wilkins, Philadelphia. 2015;1:657-84.
9. Jennette JC, Thomas DB. Pauci-immune and Antineutrophil Cytoplasmic Autoantibody Mediated Crescentic Glomerulonephritis and Vasculitis. Chapter 16. In: Jennette JC, Olson JL, Silva FG, D'Agati VD, editors. Hepinstall's Pathology of the Kidney, 7th ed. Lippincott Williams & Wilkins, Philadelphia. 2015;1:685-713.
10. Colvin RB. Classification of Membranoproliferative Glomerulonephritis and Complement- related Diseases. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.64-73.
11. Cornell LD. IgA Nephropathy. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.142-155.
12. Chang A. Membranous Glomerulonephritis. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.46-55.
13. Farris AB. Focal Segmental Glomerulosclerosis Classification. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.14-17.
14. Meehan SM. Systemic Lupus Erythematosus. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.170-187.
15. Chang A. Henoch- Schonlein Purpura. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.156-63.
16. Kambham N. Haemolytic Uremic Syndrome, Infection related. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.70-73.

17. Farris AB. Hypertensive Renovascular Disease. In: Colvin RB editor. Diagnostic Pathology. Kidney Diseases. 1st ed. Canada: Amirys Publishing Inc; 2011.p.106-113.
18. The University of Tennessee Health Science Center Nephrology [Internet]. Tennessee. Renal syndromes leading to abnormal kidney function [updated 2014 March 14] Available from: https://www.uthsc.edu/nephrology/documents/renal-syndromes-guide.pdf
19. Floege J, Feehally J. Introduction to Glomerular disease: Clinical presentations. In: Johnson RJ, Freehally J, Floege J, editors. Comprehensive Clinical Nephrology. 5th ed. Philadelphia. Elsevier Inc; 2015.p.184-97.
20. Deshpande SA, Kale K, Moulick ND. Clinico-histopathological correlation of Nephritic Syndrome in adults in urban population. International J. of Healthcare and Biomedical Research. 2015;3:34-45.
21. Polito MG, Ribeiro de Moura LA, Kirsztajn GM. An overview on frequency of renal biopsy diagnosis in Brazil: clinical and pathological patterns based on 9617 native kidney biopsies. Nephrol Dial Transplant. 2010;25:490–6
22. Naumovic R, , Pavlovic S, Stojkovic D, Basta-Jovanovic G, Nesic V. Renal biopsy registry from a single centre in Serbia: 20 years of experience. Nephrol Dial Transplant. 2009;24:877–85.
23. D'Agati VD. Renal Disease in Systemic Lupus Erythematosus, Mixed Connective Tissue Disease, Sjogren's Syndrome, and Rheumatoid Arthritis, Chapter 12. In: Jennette JC, Olson JL, Schwartz MM, Silva FG, editors. Hepinstall's Pathology of the Kidney, 6th ed. Lippincott Williams & Wilkins, Philadelphia. 2007;1:518-612.
24. Pesce F, Schena FP. World distribution of glomerular diseases: the role of renal biopsy registries. Nephrol Dial Transplant. 2013;28:334-6.