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Original Research Article

Histopathological Spectrum of Renal Biopsy in Adult Onset Nephrotic Range Proteinuria: An Experience at a Tertiary Care Centre

Savita Saharan[1*], Ranjana Solanki[2], Deepika Hemrajani[3], Madan Kumar Solanki[1]

[1]Resident, [2]Professor, [3]Assistant Professor,
Department of Pathology, SMS Medical College, Jaipur, Rajasthan, India.

ABSTRACT

Introduction: Adult onset nephrotic syndrome is broad clinical entity, needs further histological evaluation to reach a definite diagnosis. Hence, these patients should be investigated with renal biopsy. The aim of our study was to determine the histopathological spectrum of renal biopsy and to establish the clinic-pathological correlation in adults presenting with nephrotic range proteinuria.

Methods: 106 biopsies presenting with nephrotic range proteinuria were evaluated and 6 were excluded due to inadequacy. All the cases were subjected to Hematoxylin & Eosin stain and special stains like, Periodic Acid–Schiff, Methenamine-silver method (Jones) and Masson's Trichrome. IF core was processed separately.

Results: There were 63 male and 37 female patients with age range of 18-70 years. The most frequently affected age group was 18-27 years. 85 cases were of primary glomerular disease, 14 were of secondary glomerular disease and one case was of acute tubulo-interstitial nephritis. Overall, the most common pathological diagnosis was minimal change disease in 22%, followed by membranous glomerulonephritis in 13%. Most common secondary glomerular disease was Lupus Nephritis in 8%. IF modified the diagnosis in 41% cases while serology helped to arrive at diagnosis in 12% cases.

Conclusion: Our study emphasizes on the usefulness of combined approach including clinical data, serology, LM & IF, as results permit us to establish the specific diagnosis, which gives us a fair idea about selection of specific therapeutic regimen and likely prognosis of the disease.

Key words: Proteinuria, Adult Onset Nephrotic Syndrome, Renal Biopsy.

***Correspondence to:**

Dr. Savita Saharan,
C/O Sh. Vineet Prakash Tiwari,
5-A, Purendar Ji Ka Bagh,
Near Shanti Motors, MD Road,
Jaipur, Rajasthan, India.

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INTRODUCTION

Adult onset nephrotic syndrome (NS) is differ from paediatric NS in view that all patients require biopsy before planning the treatment, because for adults, the dangers of renal biopsy are minimum with skilled hands and avoids unnecessary steroid treatment.[1] Hence, histological examination remains the gold standard for the diagnosis of renal diseases. It helps in the classification of renal disorders and gives an insight into pathogenesis.

Proteinuria is a classical sign of renal injury.[2] It can be caused by direct injury to the kidney or by underlying systemic diseases. Nephrotic range proteinuria is defined as urinary protein excretion > 3.5 g/1.73 m^2/24 hours. The clinical and laboratory profiles alone do not give any clue about the underlying histological type, however they add to the diagnostic accuracy and also help in differential diagnosis when used in combination with LM and IF examination of renal biopsy.[3]

Glomerular diseases differ in epidemiology, etiology and natural history in different countries and their prevalence also varies with different socioeconomic conditions, race, age and indications for renal biopsy.[4] In India the most common pathological diagnosis of adult NS is Focal Segmental Glomerulosclerosis (FSGS), however there can be regional difference in the type of disease affecting our study population (north-western India).

Our study was aimed to determine the histopathological spectrum of renal biopsy in patients with adult onset nephrotic range proteinuria at a tertiary care centre in north-western India and to establish the clinico-pathological correlation in such patients.

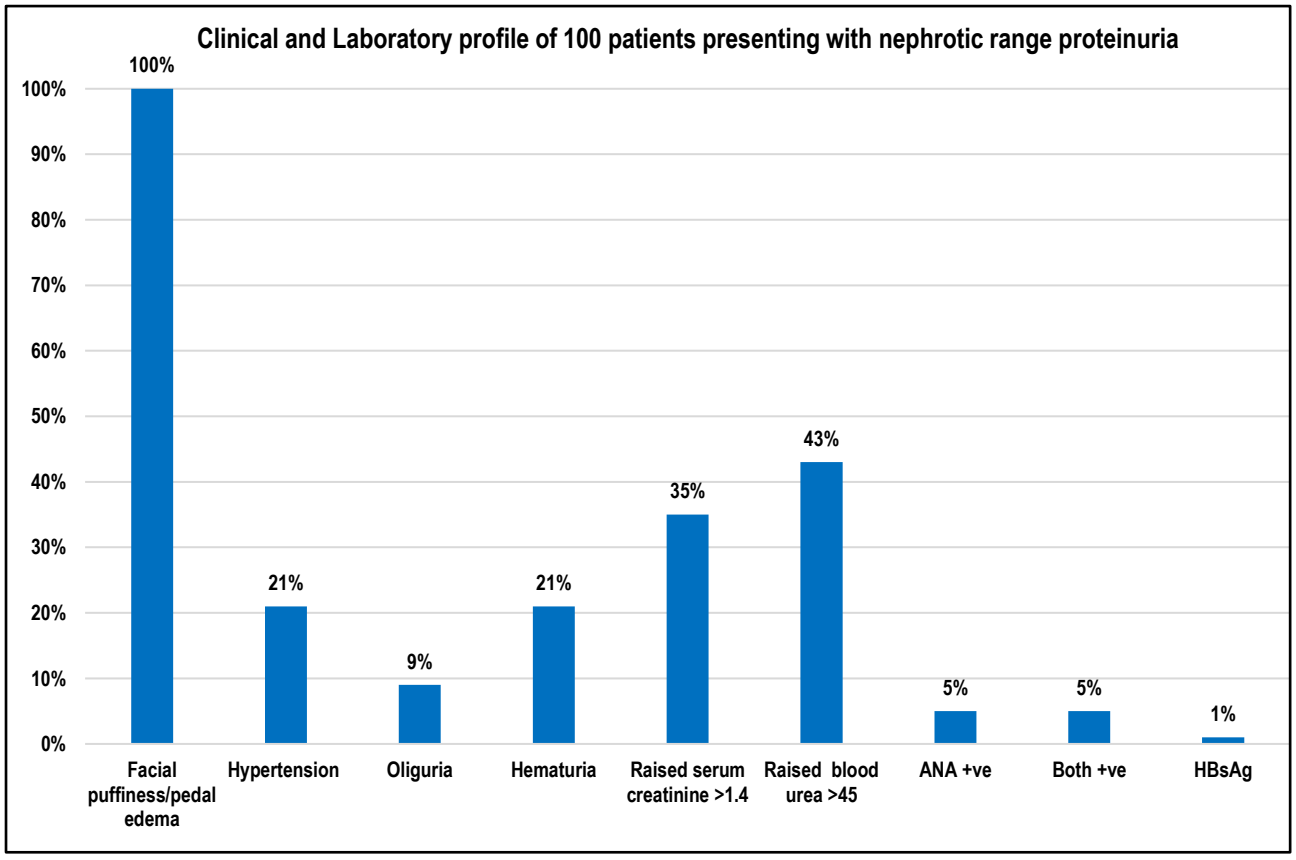
METHODS

The present prospective study was conducted at Department of Pathology, SMS Medical College & Hospitals, Jaipur over a period of two & half years. 100 adults (≥18 years of age) presenting with nephrotic range proteinuria were included in our study. Patients < 18 years of age and patients with absolute contraindication to renal biopsy i.e. bleeding diathesis and uncontrolled hypertension were excluded from the study.

All the cases were studied after detailed clinical work-up and investigated for routine urine and microscopic examination, 24 hours urinary protein, blood urea, serum creatinine, serum albumin, serum lipid profile, anti-HIV antibody, HBsAg, Anti-HCV antibody, ANA, Anti-dsDNA and ANCA serological tests were performed, if indicated. Informed consent from patients was taken. Two cores of renal tissue were obtained and systematically examined by light microscopy (LM) and immunofluoroscence (IF) study. The sections for LM examination were meticulously studied on Hematoxylin & Eosin (H&E) stain and special stains like Periodic Acid–Schiff (PAS), Methenamine-silver method (Jones) and Masson's Trichrome. Additional special stains were done wherever necessary. The second core was evaluated by Direct Immunofluorescence (DIF) techniques on cryostat sections with a panel of fluorochrome labelled antibodies to immunoglobulin G, M, A, complement component C3, C1q, kappa and lambda. Fibrinogen was used whenever indicated. The patients were categorized on basis of LM and IF findings and their correlation with serological and clinical findings was done wherever necessary.

Table 1: Age and Gender Distribution of 100 patients presenting with Nephrotic range proteinuria.

Age	Gender				Total	%
	Male	%	Female	%		
18-27	32	32%	19	19%	51	51%
28-37	12	12%	8	8%	20	20%
38-47	12	12%	5	5%	17	17%
48-57	4	4%	1	1%	5	5%
58-67	2	2%	4	4%	6	6%
>67	1	1%	0	0%	1	1%
Total	63	63%	37	37%	100	100%



RESULTS AND OBSERVATIONS

Out of 100 patients, there were 63 male and 37 female with male:female ratio 1.7:1. The age range was from 18 to 70 years with a mean of 31.5 years. The most frequently affected age group was18 to 27 years .The main demographic characteristics are shown in table 1.

All 100 patients in our study presented with oedema and nephrotic range proteinuria. Out of these 21% were hypertensive, 9% had oliguria, 35% had raised serum creatinine, 43% had raised blood urea and 21% had hematuria. ANA & Anti-dsDNA were positive in 5 patients. One patient was positive for HBsAg (Fig.1). None of the patients show anti-HCV antibody or anti-HIV antibody positivity. Mean 24 h urine protein excretion was 4.45 ± 1.21 g, mean serum albumin was 2.46 ± 0.55g/dL and mean serum cholesterol was 312.1± 119.29mg/dL.

We observed that out of 100 biopsies, most common pattern on LM examination was minimal change disease (MCD) (23%), followed by membranous glomerulonephritis (MGN) (14%),

Diffuse Proliferative Glomerulonephritis (DPGN) (13%) and Focal Segmental Glomerulosclerosis (FSGS) (10%).The distribution of cases based on LM & IF findings are tabulated in **Table 2.**

There were three cases with Crescentic glomerulonephritis morphology,of which two were labelled as Pauci Immune Crescentic Glomerulonephritis on IF study. One was p-ANCA positive and in the other ANCA status could not be assessed.

There were six biopsies showing Glomerular Organized Deposit (GODD), out of which, three were labelled as Renal Amyloidosis after staining for amyloid and PAS and two as Diabetic Nephropathy (DN) class III. In one biopsy, the nodules were strongly PAS positive, non argyrophilic, staining brown on silver stain and noncongophilic. There was no history of diabetes mellitus and on IF study, it was immunologically negative. We could not evaluate this biopsy by electronmicroscopy (EM), hence could not classify it further. Beside these, we encountered one case each of Chronic thrombotic microangiopathy (TMA) with advanced glomerulosclerosis, Chronic Ischemic Nephropathy and Acute Tubulo-interstitial Nephritis (ATIN).

Our results show that the glomerular diseases accounted up to 99% of the all renal biopsies presenting with nephrotic range proteinuria, while one case (1%) was of ATIN. Out of 99 cases of glomerular diseases, 85 (85.86%) presented with primary cause (PGD), while 14 (14.14%) had secondary causes (SGD).The spectrums of PGD and SGD are shown in **Table 3 & Table 4.**

In PGD most common lesion was MCD, seen in 22/85 (25.88%) cases, followed by MGN, seen in 13/85 (15.29%) cases, infection related glomerulonephritis (IRGN) 12/85 (14.12%) and FSGS 9/85 (10.58%). In SGD most common lesion was Lupus Nephritis (LN), seen in 8/14 (57.14%) cases, followed by Renal Amyloidosis in 3/14 (21.43%). Among LN cases, class IV LN accounted for the maximum number of cases, i.e. 4/8 (50%), followed by one case each of class I, II, III, V lesions.

The distribution of sex in various renal **diseases (Table 5)** shows male preponderance except LN and chronic TMA with Glomerulosclerosis. The distribution of histological diagnosis according to age (Table 6) shows that in patients < 40 years of age, MCD was the most common lesion, seen in 19/81 (23.46%) cases, while in patients > 40 years of age, it was MGN, in 5/19 (26.32%) cases.

Table 2: Distribution of cases based on light microscopy and IF findings

Morphological pattern on LM (n=100)	n(%)	IF finding	Final diagnosis	n(%)
NORMAL PATTERN	23	Negative	MCD	22 (95.65%)
	(23%)	Full house pattern	LN Class I	1 (4.35%)
MGN	14	CWG positivity of IgG& C3	MGN	13 (92.86%)
	(14%)	Full house pattern	LN Class V	1 (7.14%)
DPGN	13	CW lumpy bumpy deposits of IgG&C3	IRGN	9 (69.23%)
	(13%)	Full house pattern	LN class IV	3 (23.07%)
		CW & mesangium positivity of C3	C_3 Dominant GN	1 (7.69%)
FSGS	10	Focal ±to1+positivity of IgM& C3	FSGS	9 (90%)
	(10%)	CW & mesangium positivity of IgA	IgANclass IV	1 (10%)
Chronic GS	10	No Ig deposition to focal trace to 1+ IgM& C3 trapping	Chronic GS	8 (80%)
	(10%)	Full house pattern	LN class IV	1 (10%)
		CW & mesangium positivity of IgA & C3	IgAN	1 (10%)
MPGN	9 (9%)	CWG positivity of IgG&C3	MPGN	3 (33.33%)
		CW & mesangium positivity of C3	C_3 Dominant GN	6 (66.67%)
MesPGN	8 (8%)	CWG & mesangium positivity of IgA	IgA class IV	3 (37.5%)
		Full house pattern	LN class II	2 (25%)
			LN class III	
		CW lumpy bumpy deposits of IgG&C3	IRGN	2 (25%)
		CW & mesangium positivity of C3	C_3 Dominant GN	1 (12.5%)
GODD	6 (6%)	Negative	Renal amyloidosis	3 (50%)
		Negative	Diabetic N class III	2 (33.33%)
		Negative	GODD unclassified	1 (16.67%)
Crescentic GN	3 (3%)	CWG positivity of IgG & C3	MPGN	1 (33.33%)
		Negative	Pauci Immune GN	2 (66.67%)
FPGN	1 (1%)	CW lumpy bumpy deposits of IgG & C3	IRGN	1 (100%)
ATIN	1 (1%)	Negative	Acute TIN	1 (100%)
Chronic ischemic nephropathy	1 (1%)	Negative	Chronic ischemic nephropathy	1 (100%)
Chronic TMA with GS	1 (1%)	Trace positivity of IgG& C3.	Chronic TMA with GS	1 (100%)

GN-glomerulonephritis, MGN-membranous GN, FSGS- focal segmental glomerulosclerosis, MPGN- membranoproliferative GN, MesPGN- mesangioproliferative GN, GODD- glomerular organized deposits, DPGN- diffuse proliferative GN, FPGN- focal proliferative GN, ATIN- acute tubulointerstitial nephritis, TMA- thrombotic microangiopathy

Table 3: Distribution of primary glomerular diseases (n=85)

Primary Glomerular disease	No. of cases (n=85)	Percentage (100%)
MCD	22	25.88%
MGN	13	15.29%
IRGN	12	14.12%
FSGS	9	10.59%
C_3 dominant GN	8	9.41%
Chronic GS	8	9.41%
MPGN	4	4.71%
IgA nephropathy	5	5.88%
Pauci immune GN	2	2.35%
Chronic TMA with GS	1	1.18%
GODD unclassified	1	1.18%

Table 4: Distribution of secondary glomerular diseases (n=14)

Secondary Glomerular disease	No.of cases(n=14)	Percentage (100%)
LN	8	57.14%
Renal amyloidosis	3	21.43%
Diabetic nephropathy	2	14.29%
Chronic ischemic nephropathy	1	7.14%

Table 5: Distribution of sex in various histological diagnosis-

Diseases		Gender			
		F (n=37)	%	M (n=63)	%
MCD		8	21.62	14	22.22
MGN		3	8.11	10	15.87
IRGN		5	13.51	7	11.11
FSGS		3	8.11	6	9.52
C_3 dominant GN		3	8.11	5	7.94
Chronic GS		3	8.11	5	7.94
LN		7	18.92	1	1.59
IgA nephropathy		2	5.41	3	4.76
MPGN		1	2.70	3	4.76
Pauci immune GN		1	2.70	1	1.59
GODD	Renal amyloidosis	0	0	3	4.76
	Diabetic nephropathy	0	0	2	3.17
	Unclassified	0	0	1	1.59
Chronic ischemic nephropathy		0	0	1	1.59
Chronic TMA with GS		1	2.70	0	0.00
ATIN		0	0	1	1.59
Total		37	100	63	100

Table 6: Distribution of histological diagnosis according to age.

Disease		≤40 yrs (n=81)	%	>40 yrs (n=19)	%
MCD		19	23.46	3	15.79
MGN		8	9.88	5	26.32
IRGN		9	11.11	3	15.79
FSGS		8	9.88	1	5.26
C_3 dominant GN		7	8.64	1	5.26
Chronic GS		6	7.41	2	10.53
LN		8	9.88	0	0.00
IgA nephropathy		4	4.94	1	5.26
MPGN		4	4.94	0	0.00
Pauci immune GN		1	1.23	1	5.26
GODD	Renal amyloidosis	3	3.70	0	0.00
	Diabetic nephropathy	2	2.47	0	0.00
	Unclassified	0	0.0	1	5.26
Chronic ischemic nephropathy		0	0.0	1	5.26
Chronic TMA with GS		1	1.23	0	0.00
ATIN		1	1.23	0	0.00
Total		81	100.00	19	100.00

DISCUSSION

The main role of renal biopsy is to establish a specific diagnosis that helps the clinician to give diagnosis-specific therapy.[5] Ideal approach to renal biopsy includes LM, IF and EM examination. Although this is the routine approach in most laboratories of the developed countries but in developing countries most centres give diagnosis solely on LM examination which gives only morphological diagnosis.[6] Addition of IF and EM changes the diagnosis.

In our study,63% were males and 37% were females, indicating that chronic renal diseases are more common in males. The similar ratio was seen in study done by Tarik et al[7] with 62% male and 38% female.

The age range was from 18 to 70 years .The mean age of study population was 31.5 ± 13.4 years, which is comparable to study done by Rathi et al[8] having 31.5 ± 11 years. The most frequently affected age group was 18 to 27 years which is the same as that reported in study of Tarik et al.[7]

Out of 21 patients having hematuria, one patient of IRGN had macroscopic hematuria while rest all had microscopic hematuria. All 5 (100%) cases of IgAN and 66.67% cases of IRGN accounted as leading cause of hematuria.

Total 35% patients had serum creatinine >1.4 mg/dL. It was observed that serum creatinine was high in all 8(100%) patients with Chronic Glomerulosclerosis while all patients with MCD had normal serum creatinine. These results are also comparable to study done by Rathi et al.[8]

In our study most common lesion was MCD (22%), which is in concordance with many other studies.[9-14] However our result is at variance with many other recent studies.[8,15,16] where they suggest a shifting trend favouring FSGS as the most common cause of primary glomerular diseases. In our study FSGS was found in 9% cases. The reason for this discrepancy in findings may be due to smaller sample size in our study or missing the abnormal glomeruli during sampling and another important reason is unavailability of EM, where early FSGS presenting with mere foot process effacement, largely represents as MCD in the appropriate clinical setting.

The second most common lesion in our study was MGN (13%). Similarly in study by Rathi et al[8], Hassan et al[17], Sabir et al[18], Akhtar et al[14], MGN was the second most common lesion. While Zhou et al[19] found MGN as most common cause of PGD in his study.

In our study 12% cases were of IRGN. In a study done by Moroni et al[20] incidence of Infection-associated GN was 1.75%, as they found 50 out of 2862 biopsies fulfilling the inclusion criteria. These findings are at variance from our study possibly because our's is a tertiary care referral centre, where all poorly controlled infections are referred.

C3 Glomerulopathy (C3GP) has recently been described as a distinct entity. The most common presentation is with an acute nephritic syndrome. It's morphological picture is heterogeneous[21-23], as seen in our study, and none of the patterns is diagnostic of C3GP. An isolated C3 (i.e. >3+) or C3 dominant (i.e. >2 orders of intensity than any deposit of Ig) pattern on IF is the only feature to suggest an underlying C3GP on routine renal biopsies.[21-26] Undoubtedly, confirmatory tests like EM is essential to further classify these disorders into individual subtypes; however, it was not available in for study.[23-25,27]

Major entities miscalled as C3GP are Autoimmune Glomerulonephritis and IRGN. The differentiation of true IRGN from C3GP often cannot be made on the basis of morphology, clinical and lab data available at the time of biopsy. These patients require follow up clinically and serologically over several months. We found that eight (8%) cases were of C3GP. In a study done by Viswanathan et al[28] 0.7% cases of all renal biopsies were C3GP and 1.16% in another study by Mathur et al.[29] This discrepancy may be attributed to small sample size and lack of further work-up in such patients.

Although IgA nephropathy has been reported in all age groups but is most common in the second or third decade of life with a higher prevalence among males.[30] We found 5% patients of IgAN with a mean age of 29.4 years and male preponderance. A similar pattern was observed in study by Vanikar et al[31] and Siddappa et al.[30]

Amongst SGD, LN was the most common cause in our study, which is comparable to many other studies.[7,8,12,15-17,19] The second common cause of SGD was Renal Amyloidosis. In our country tuberculosis is common problem which often progresses to amyloidosis. Since the treatment of Renal amyloidosis is totally different from that of primary NS, hence emphasizes the need for renal biopsy in older patients with NS[19]. Other diseases which are on the rise in older patients of developed countries include cryoglobulinemia, monoclonal gammopathy induced deposit disease and collagenofibrotic glomerulopathy, which also emphasize the need for renal biopsy.

Though worldwide the most common cause of SGD is diabetes mellitus, we found only 2 (2%) patient having DN. Since in long standing type1 diabetic patients with complications like retinopathy or neuropathy, there is little doubt in diagnosis. Hence renal biopsy is rarely needed for evaluation.

We found that in Mesangioproliferative GN, DPGN, Crescentic GN and FPGN patterns of glomerular diseases, IF and serologic studies were very useful in the differential diagnosis of specific GN which led to specific categorization. In our study IF was indispensable in the final diagnosis of 41% cases and serology was useful in arriving at a final diagnosis in12% cases.

CONCLUSION

Our study demonstrates that clinical data, serological tests, in combination with IF and LM study are indispensable in the elucidation of final specific diagnosis of patients presenting with nephrotic range proteinuria. Hence, these should be employed routinely in the pathologic evaluation of renal biopsies. This gives a fair idea of selection of specific therapeutic regimen and likely prognosis of the disease.

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