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## Spectrum of renal biopsies; a three-year data from a tertiary care centre of eastern India

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### ABSTRACT

**Introduction:** Biopsy registries are important in studying the frequency of kidney diseases, their trends over time and allow comparison of data from different geographical regions.

**Objectives:** We report the spectrum of biopsy-proven glomerular and tubular diseases in a single centre in Eastern India.

**Patients and Methods:** Medical records of 1293 patients with biopsy-proven glomerular diseases (GDs) and tubular diseases over a period of three years (March 2013 to March 2016) were retrospectively analysed. All biopsy specimens were examined by the same pathologist with light and immunofluorescence microscopy. Electron microscopic (EM) analysis was performed only in selected cases. Histologic spectrum of various GDs was studied along with its correlation with the clinical and laboratory parameters.

**Results:** The clinical diagnosis was nephrotic syndrome (NS) in 820 (63.41%), rapidly progressive glomerulonephritis in 194 (15.2%), asymptomatic urinary abnormalities (AUA) in 108 (8%), acute kidney injury (AKI)/acute nephritic syndrome in 118 (9.3%), and macroscopic hematuria (MH) in 3 (0.43%) patients. Male: Female ratio was 0.95. Around 22.42% were < 18 years, 72.2% were between 18 to 59 years, and 5.3% were ≥ 60 years of age. The most common GD overall as well as primary glomerular disease (PGD) was minimal change disease (MCD) (21.6 and 83.44%). Secondary GD was present in 20.87%; most common being lupus nephritis (84.58%). Among the NS, the most common GD was MCD (33.17%), followed by FSGS (17.56%), membranous nephropathy (MN) (13.90%), lupus nephritis (LN) (9.39%), IgA-nephropathy (0.49%), and MPGN (7.32%). Thrombotic microangiopathy (TMA) constituted 24.6% of AKI presentation particularly common in pregnancy. Diffuse proliferative glomerulonephritis (DPGN) with severe renal dysfunction was present in 14% of elderly patients.

**Conclusion:** The spectrum of GD varies according to the area of study and changes over time. Some entities which have not been reported earlier from this region that are uncommon but are significant were pregnancy associated TMA, C3 glomerulopathy (C3G) and DPGN in adults.

### Implication for health policy/practice/research/medical education:

This study presents the pattern of kidney diseases based on biopsy findings. It will help in analyzing the changes in kidney diseases, especially glomerular diseases over time and comparing different disease patterns in other developing and developed countries. Understanding clinical and biopsy correlations will help in management of patients. Preventive and treatment strategies can further be planned for newly recognized disease entities.

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### Introduction

Renal biopsy registries provide the data on epidemiology of various renal pathologies and provide insight into the changing trends over time (1,2). These data also allow comparison of pattern of renal diseases between different

geographical regions across the world (1). In Japan the incidence of diabetic nephropathy is rising, while IgA nephropathy is showing a declining trend (3). The biopsy registries help in estimating the disease burden due to individual diseases and this may help primary and

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secondary prevention strategies. In India, population based data on chronic kidney disease (CKD) is not very robust however, a high overall prevalence of 17.2% has been reported (4). Among the causes of CKD, diabetic nephropathy (31%) has been reported as the commonest followed by CKD of unknown origin (16%) (5). Screening programs for urinary abnormalities and conducting the biopsy in early stages will help define the glomerular diseases (GDs) in the CKD with unknown etiology (CKDu) group. The developments in the understanding of pathogenesis of various kidney diseases resulted in changes and updating of classification systems applied to the reporting of renal biopsies (6,7). Additionally, some newer entities like C3 glomerulopathy have been defined (8). Despite an encouraging data on non-invasive biomarkers, renal biopsy is an irreplaceable investigation, essential for prognosis and treatment in many diseases at present (9). Though invasive, in the experience of most centers it has been found safe with very low risk of complication in both adults and children (10,11). Hence, renal biopsy registries are an essential tool for epidemiologic analysis as well as for enhancing and continuing our understanding of the differences in renal diseases across various regions and ethnicities (12).

### Objectives

There is data on spectrum of renal biopsies from various regions of India, however, from eastern India is scarce (13,14). Here, we present the observations and analysis of three years data of renal biopsies performed at the largest single center of Eastern India. The aim was to study the spectrum of renal biopsies and the corresponding clinical presentation. This study also elaborates our protocol of renal biopsy which may be of paramount importance in identifying GDs at early stage and thereby unraveling the CKDu group further.

### Patients and Methods

#### Study design

This is a retrospective observational single center study of the native kidney biopsies performed between March 2013 to March 2016 (n=1293) at Institute of Post-Graduate Medical Education and Research, Kolkata. Records of renal biopsies performed during this period were examined. For collection of data regarding the clinical and laboratory parameters of renal biopsy (Bx) requisitions containing age, gender and indication of biopsy were reviewed. The data obtained was analyzed and biopsy findings were compared with clinical and laboratory parameters.

The analysis of biopsy registry and its comparisons with other studies, heavily depends on the biopsy practices of the institute/geographical area. Hence we consider it prudent to detail the protocol of renal biopsy indications practiced at our institute. The indications of renal biopsy in childhood onset nephrotic syndrome adopted in our institute are the guidelines recommended by Kidney

Disease Improving Global Outcomes (KDIGO) (15). Additional indications in childhood onset nephrotic syndrome as well as in other clinical presentations have been elaborated in Table S1 (See online Supplementary file 1). Acute kidney injury (AKI) in pregnancy which is not associated with preeclampsia or other obvious cause is biopsied unless contraindicated to diagnose hemolytic uremic syndrome. There is no population based urinary screening program in this part of the country however, those incidentally detected to have asymptomatic urinary abnormalities (AUA) suggestive of glomerular etiology are conducted kidney biopsy as this would help in prognostication and treatment of the disease. The contraindications to biopsy are mentioned in Table S2.

#### Renal biopsy procedure

As an institutional protocol, two cores of renal tissue were obtained with automated biopsy gun (16G × 16 cm for adults ≥18 years old and 18 G × 16 cm for patients <18 years of age) under real-time ultrasonography guidance and subjected to light microscopy (LM) using Hematoxylin and eosin, periodic acid–Schiff (PAS) stain, methenamine silver stain and trichrome stain and immunofluorescence (IF).

IF staining was performed on 3- $\mu$ m cryostat sections using polyclonal fluorescein isothiocyanate-conjugated antibodies to IgG, IgM, IgA, C3, C1q, kappa and lambda light chains. The intensity of IF staining was graded on a scale of 0 to 3+. Interstitial fibrosis and tubular atrophy (IFTA) were graded as mild (<25%), moderate (25-50%), and severe (>50%) on LM. When thought necessary by the treating physician electron microscopy was done using the paraffin embedded tissue. Additional special stains were used whenever indicated. All biopsy samples were analyzed by a single nephropathologist. A second biopsy was performed in case a biopsy sample was inadequate for diagnosis and if exact diagnosis would have significant implications on the therapy and if the patients gave consent for a re-biopsy.

#### Ethical considerations

This is a retrospective study. The existing records of data available with department of nephrology, IPGMER, Kolkata were reviewed and analyzed. The research followed the tenets of the Declaration of Helsinki. Written informed consent at the time of renal biopsy was obtained.

#### Statistical analysis

Descriptive statistical analysis was performed. For categorical data, proportion is presented as percentage of total. Continuous normally distributed data are presented as means with corresponding standard deviations (SD).

### Results

We first describe the clinical presentation of patients undergoing kidney biopsy and the baseline characteristics

followed by the etiological diagnosis in each clinical presentation category. For further analysis of our biopsy data, kidney diseases have been classified as; glomerular pathologies including; (a) primary glomerular diseases (PGD) and (b) secondary glomerular diseases (SGD). Patients with evidence of extensive chronic glomerulosclerosis (CGS) were also classified into PGD or SGD depending on the findings on histology. If they were not classifiable as PGD or SGD, they have been included separately as CGS. Finally, we compare the data of this study with that of our previous data (16) and the data from neighbouring regions of our country (16-18) as well as other registries across the globe (19,20).

A total of 1293 biopsy-proven glomerular/tubular diseases were recorded over the period of study. The average glomerular yield was adequate ( $17.41 \pm 8.42$  glomeruli). Electron microscopy (EM) was performed in 5.3% of biopsies. The average age of the patients was  $29.7 \pm 15.24$  years. Male: Female ratio was 0.95 (631 males: 662 females).

The contribution of the various glomerular/tubular syndromes to the performance of kidney biopsy is presented in Figure 1. The demographic and baseline characteristics and frequency of each of these syndromes are described in Table 1. The most common clinical presentation was nephrotic syndrome (NS). The various types of histology and their proportion in each clinical syndrome are presented in Table 2. The most common cause of NS was minimal change disease (MCD) followed by focal segmental glomerulosclerosis (FSGS). The most common etiology of RPRF/RPGN (rapidly progressive renal failure/rapidly progressive glomerulonephritis) was necrotising crescentic GN (CresGN) seen in 32.99% ( $n = 64$ ); of them, 23.43% were anti-GBM disease and 18.75% were pauci-immune. The next common GD presenting as RPRF/RPGN was lupus nephritis (LN) (32.47%,  $n = 63$ ), of which class IV was in 71.42% of cases. Lupus nephritis was seen in 9.39% ( $n = 77$ ) of NS while 55.84% of the cases were either pure class V of LN or class V overlap with other LN classes. The distribution of various etiologies to GN with

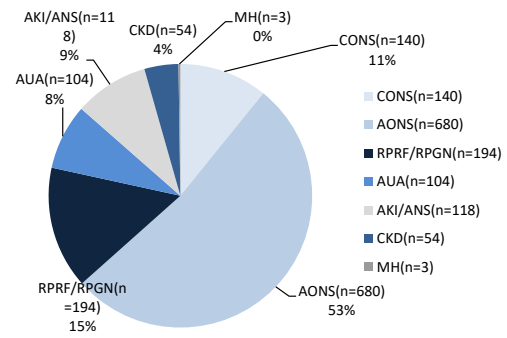


Figure 1. Spectrum of various clinical syndromes.

crescents has been shown in Table S3. As expected, the disease with the severest changes was necrotising CresGN, with an average of  $79.03 \pm 33.2\%$  crescents on biopsy. Four cases of MN showed crescents. Of these two had full house staining on immunofluorescence (IF) suggestive of secondary MN but has no clinical or serological evidence of a secondary disease. The variants of FSGS have been shown in Table S4. Membranoproliferative GN (MPGN) comprised 7.35% of the total biopsies.

In those presenting as AKI/AGN, acute tubular necrosis/acute tubulointerstitial nephritis (ATN/ATIN) and thrombotic microangiopathy (TMA) contributed to 24.58% each followed by lupus nephritis. In ATN/ATIN group 72.41% ( $n=21$ ) cases were none recovering AKI associated with snake bite. One case was diarrhoea associated non-recovering AKI. Rest of the cases were of undetermined etiology. Two cases had moderate chronicity (IFTA >50%). In our study 75.8% cases of TMA were presented as post-partum AKI. Around 17.2% of cases were associated with malignant hypertension. About 68.18% of patients in post-partum TMA had patchy cortical necrosis. Of, all TMA cases, one case had calcineurin inhibitor toxicity and one had post-diarrhoeal haemolytic uremic syndrome. The most common cause of AUA was IgAN contributing to almost half (42%) of the cases. IgAN was also the most common etiological

Table 1. Baseline characteristics of patients in each clinical syndrome ( $n = 1293$ )

Variable	CONS (n=140)	AONS (n=680)	RPRF/RPGN (n=194)	AKI/ANS (n=118)	AUA (n=104)	CKD (n=54)	MH (n=3)
Age (y)*	$10.5 \pm 5.0$	$31.8 \pm 14.4$	$33.3 \pm 15.1$	$31.0 \pm 14.9$	$29.9 \pm 12.0$	$36 \pm 16.0$	$25.3 \pm 12.5$
Gender (male)	61.4	50.7	36.6	38.1	49.5	51.8	66.6
Hypertension (% in each category)	0.07	41.3	77.8	75.6	31.8	90.8	0
Serum creatinine* (mg/dL)	$0.9 \pm 0.42$	$1.64 \pm 1.49$	$5.2 \pm 4.3$	$5.1 \pm 3.2$	$1.6 \pm 0.9$	$6.67 \pm 4.14$	$1.13 \pm 0.2$
Serum albumin* (g/dL)	$2.7 \pm 0.6$	$2.5 \pm 0.9$	$2.8 \pm 1.1$	$2.9 \pm 1.4$	$3.1 \pm 0.8$	$2.8 \pm 0.7$	$2.9 \pm 0.5$
Hemoglobin* (g/dL)	$12.4 \pm 2.8$	$10.9 \pm 3.6$	$8.8 \pm 2.7$	$11.1 \pm 2.2$	$11.5 \pm 1.5$	$7.9 \pm 2.9$	$12.7 \pm 0.8$
Hematuria (% in each category)	14.2	26.3	82.1	70.6	66.1	18.5	100
24 hour Urine Protein* (g/d)	$3.5 \pm 2.12$	$4.7 \pm 3.0$	$2.7 \pm 1.8$	$1.2 \pm 0.9$	$1.7 \pm 1.1$	$2.4 \pm 1.3$	$0.6 \pm 0.4$

\*Values indicate mean  $\pm$  standard deviation

RPRF, Rapidly progressive renal failure; RPGN, Rapidly progressive glomerulonephritis; AUA, Asymptomatic urinary abnormalities; ANS, Acute nephritic syndrome; AKI, Acute kidney injury; CKD, Chronic kidney disease; MH, Macroscopic hematuria.

**Table 2.** Spectrum of various glomerular histologies in each clinical syndrome

Biopsy Diagnosis n (% of total)	NS (n=820) (%)	RPRF/RPGN (n=194) (%)	AKI/ANS (n=118) (%)	AUA (n=104) (%)	CKD (n=54) (%)	MH (n=3)(%)
MCD/Normal (n=279; 21.58%)	272 (33.2)	0 (0)	5 (4.2)	2 (1.9)	0 (0)	0 (0)
FSGS (n=163; 12.61%)	144 (17.6)	3 (1.5)	6 (5.1)	8 (7.7)	2 (3.7)	0 (0)
MN (n=132; 10.21%)	114 (13.9)	7 (3.6)	3 (2.5)	6 (5.8)	2 (3.7)	0 (0)
LN (n=181; 14.00%)	77 (9.4)	63 (32.5)	19 (16.1)	21 (20.2)	1 (1.9)	0 (0)
IgAN/HSP (n=134; 10.36%)	59 (7.2)	10 (5.2)	3 (2.5)	42 (40.4)	17 (31.5)	3 (100)
MPGN (n=95; 7.35%)	60 (7.3)	14 (7.2)	9 (7.6)	5 (4.8)	7 (13.0)	0 (0)
DPGN (n=98; 7.58%)	49 (5.9)	32 (16.5)	5 (4.2)	11 (10.6)	1 (1.9)	0 (0)
CresGN (n=79;6.11 %)	4 (0.5)	64 (33.0)	7 (5.9)	0 (0)	4 (7.4)	0 (0)
PCD (n=29; 2.24%)	23 (2.8)	0 (0)	1 (0.8)	0 (0)	5 (9.3)	0 (0)
CGS (n=19; 1.47%)	5 (0.6)	0 (0)	0 (0)	0 (0)	14 (25.9)	0 (0)
NGS (n=4; 0.31%)	4 (0.5)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
MesPGN (n=16; 1.24%)	8 (1.0)	0 (0)	2 (1.7)	6 (5.8)	0 (0)	0 (0)
TMA (n=29; 2.24%)	0 (0)	0 (0)	29 (24.6)	0 (0)	0 (0)	0 (0)
ATN/ATIN (n=30; 2.32%)	0 (0)	0 (0)	29 (24.6)	1 (1.0)	0 (0)	0 (0)
C1qN (n=1; 0.08%)	1 (0.1)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Lithium toxicity (n=1; 0.08%)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.9)	0 (0)
DN (n=1; 0.08%)	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)
Collapsing glomerulopathy (n=1; 0.08%)	0 (0)	1 (0.5)	0 (0)	0 (0)	0 (0)	0 (0)
Hypertensive nephrosclerosis (n=1; 0.08%)	0 (0)	0 (0)	0 (0)	1 (1.0)	0 (0)	0 (0)

NS, Nephrotic syndrome; RPRF, Rapidly progressive renal failure; RPGN, Rapidly progressive glomerulonephritis; AUA, Asymptomatic urinary abnormalities; ANS, Acute nephritic syndrome; AKI, Acute kidney injury; CKD, Chronic kidney disease; MH, Macroscopic hematuria; MCD, Minimal change disease; FSGS, Focal segmental glomerulosclerosis; MN, Membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch–Schönlein purpura; MPGN, Membranoproliferative glomerulonephritis; DPGN, Diffuse proliferative glomerulonephritis; CresGN, Crescentic glomerulonephritis; FPGN, Focal proliferative glomerulonephritis; PCD, Plasma Cell Dyscrasia; CGS, Chronic glomerulosclerosis; NGS, Nodular glomerulosclerosis; LCDD, Light chain deposition disease; TMA, Thrombotic microangiopathy; DN, Diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, Mesangial proliferative glomerulonephritis; TBMD, Thin basement membrane disease; LN, Lupus nephritis; ATIN, Acute tubulointerstitial nephritis.

diagnosis (17%) in those presenting as CKD. In 14% cases of CKD no definitive diagnosis was possible. These patients have been classified as CGS. IgAN/ Henoch–Schönlein purpura was the diagnosis in all case of macroscopic hematuria (MH).

In diffuse proliferative glomerulonephritis (DPGN), the majority of the cases were adults especially >60 years. The most common presentation was NS (49.5%) although a significant number (32.9%) presented with RPRF with dialysis requirement. A small number of patients also presented as infection related glomerulonephritis with incomplete recovery of renal function. The different biopsy diagnosis in the monoclonal gammopathy group as a common disease in adults especially elderly (including plasma cell dyscrasias) has been shown in Table S5. We had also a case of idiopathic nodular glomerulosclerosis.

Glomerular diseases were also classified according to gender and age at presentation (<12 years, 12-17 years, 18-59 years, and ≥ 60 years) (Tables 3 and 4 respectively). The GDs were also studied based on serum creatinine values (Table 5). Serum creatinine ≥ 3 mg/dL was common in CresGN, IgAN and DPGN. MCD was the most common GD in the age groups of <12 and 12-17 years, whereas LN

was the most common GD in adults in the age group 18-59 years and MN was the commonest GD in the elderly patients.

The distribution of GD as PGD and SGD is presented in Table 6 and Figure 2. LN was the most common SGD. MCD was the most common GD followed by FSGS and IgAN.

## Discussion

Analysis of CKD trends, suggests that despite diabetes and hypertension being the most common causes of end-stage renal disease (ESRD), incidence of GDs contributing to ESRD is also rising (21). Biopsy registries serve many purposes as elaborated in introduction above.

The most frequent indication for performing renal biopsy in our study was similar to most single centre reports from across the world was NS (22-24) While single centre studies almost invariably have NS as the most common indication (25,26). This may largely be due to urinary screening strategies adopted by these nations. However, in Japan where population based urinary screening programs have been implemented, since chronic glomerulonephritis was the most common indication there (27). AUA was the

**Table 3.** Glomerular disease spectrum according to gender

Biopsy Diagnosis	Male (n=631)	female (n=662)	Sex ratio F:M	Sex ratio M:F
MCD/Normal	161 (25.5%)	118 (17.8%)	-	1.36:1
FSGS	112 (17.7%)	51 (7.7%)	-	2.19:1
MN	78 (12.36%)	54 (8.2%)	-	1.4:1
LN	20 (3.1%)	161 (24.3%)	8.05:1	-
IgAN/HSP	74 (11.7%)	60 (9.1%)	-	1.23:1
MPGN	43 (6.81%)	52 (7.8%)	1.20:1	-
DPGN	46 (7.3%)	52 (7.8%)	1.13:1	-
CresGN	32 (5.1%)	47 (7.1%)	1.46:1	-
PCD	17 (2.7%)	12 (1.8%)	-	1.41:1
CGS	8 (1.2%)	11 (1.6%)	1.3:1	-
NGS	3 (0.5%)	1 (0.2%)	-	3:1
MesPGN	8 (1.2%)	8 (1.2%)	-	1:1
TMA	7 (1.1%)	22 (3.3%)	3.14:1	-
ATN/ATIN	18 (2.9%)	12 (1.8%)	-	1.5:1
C1qN	1 (0.2%)	0	-	-
Lithium toxicity		1 (0.2%)	-	-
DN	1 (0.2%)	0	-	-
Collapsing glomerulopathy	1 (0.2%)	0	-	-
Hypertensive nephrosclerosis	1 (0.2%)	0	-	-

MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; MN, membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch-Schönlein purpura; MPGN, membranoproliferative glomerulonephritis; DPGN, diffuse proliferative glomerulonephritis; CresGN, crescentic glomerulonephritis; PCD, plasma cell dyscrasia; CGS, chronic glomerulosclerosis; NGS, nodular glomerulosclerosis; TMA, thrombotic microangiopathy; DN, diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, mesangial proliferative glomerulonephritis; LN, lupus nephritis; ATIN, acute tubulointerstitial nephritis.

**Table 4.** Glomerular disease spectrum according to the age of presentation

Biopsy Diagnosis	Age Group (y)							
	<12		12-17		18-59		>60	
MCD/normal	78	69.0%	65	36.7%	133	14.2%	3	4.3%
FSGS	10	8.8%	31	17.5%	115	12.3%	7	10.1%
MN	4	3.5%	8	4.5%	107	11.5%	13	18.8%
LN	2	1.8%	19	10.7%	159	17.0%	1	1.4%
IgAN/HSP	5	4.4%	9	5.1%	116	12.4%	4	5.8%
MPGN	4	3.5%	14	7.9%	69	7.4%	8	11.6%
DPGN	4	3.5%	10	5.6%	73	7.8%	10	14.5%
FPGN	0	0.0%	0	0.0%	1	0.1%	0	0.0%
CresGN	1	0.9%	9	5.1%	60	6.4%	9	13.0%
PCD	0	0.0%	0	0.0%	21	2.2%	8	11.6%
CGS	0	0.0%	3	1.7%	16	1.7%	0	0.0%
NGS	0	0.0%	0	0.0%	3	0.3%	1	1.4%
MesPGN	1	0.9%	4	2.3%	11	1.2%	0	0.0%
TMA	3	2.7%	1	0.6%	25	2.7%	0	0.0%
ATN/ATIN	0	0.0%	4	2.3%	22	2.4%	4	5.8%
C1qN	1	0.9%	0	0.0%	0	0.0%	0	0.0%
Lithium toxicity	0	0.0%	0	0.0%	1	0.1%	0	0.0%
DN	0	0.0%	0	0.0%	1	0.1%	0	0.0%
Collapsing glomerulopathy	0	0.0%	0	0.0%	0	0.0%	1	1.4%
Hypertensive nephrosclerosis	0	0.0%	0	0.0%	1	0.1%	0	0.0%

MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; MN, membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch-Schönlein purpura; MPGN, membranoproliferative glomerulonephritis; DPGN, diffuse proliferative glomerulonephritis; CresGN, crescentic glomerulonephritis; PCD, plasma cell dyscrasia; CGS, chronic glomerulosclerosis; NGS, nodular glomerulosclerosis; TMA, thrombotic microangiopathy; DN, diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, mesangial proliferative glomerulonephritis; LN, lupus nephritis; ATIN, acute tubulointerstitial nephritis.

**Table 5.** Spectrum of glomerular diseases and distribution in various serum creatinine (SCr) levels

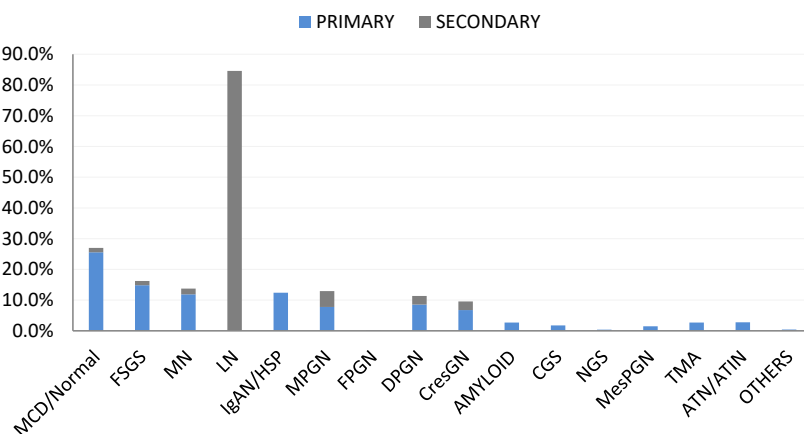
Biopsy Diagnosis	SCr <1.5 mg/dL		SCr 1.5-2.9 mg/dL		SCr >3.0 mg/dL	
	Count	%	Count	%	Count	%
MCD/Normal	245	34.5%	28	8.8%	6	2.3%
FSGS	90	12.7%	68	21.3%	5	1.9%
MN	107	15.0%	19	6.0%	6	2.3%
LN	93	13.1%	64	20.1%	24	9.1%
IgAN/HSP	58	8.2%	43	13.5%	33	12.5%
MPGN	51	7.2%	22	6.9%	22	8.4%
DPGN	36	4.9%	34	10.7%	28	10.6%
CresGN	3	0.4%	12	3.8%	64	24.3%
PCD	16	2.3%	5	1.6%	8	3.0%
CGS	0	0.0%	2	0.6%	17	6.5%
NGS	1	0.1%	2	0.6%	1	0.4%
MesPGN	9	1.3%	5	1.6%	2	0.8%
TMA	0	0.0%	3	0.9%	26	9.9%
ATN/ATIN	1	0.1%	10	3.1%	19	7.2%
C1qN	1	0.1%	0	0.0%	0	0.0%
Lithium toxicity	0	0.0%	0	0.0%	1	0.4%
DN	0	0.0%	1	0.3%	0	0.0%
Collapsing glomerulopathy	0	0.0%	1	0.3%	0	0.0%
Hypertensive Nephrosclerosis	0	0.0%	0	0.0%	1	0.4%

MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; MN, membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch-Schönlein purpura; MPGN, membranoproliferative glomerulonephritis; DPGN, diffuse proliferative glomerulonephritis; CresGN, crescentic glomerulonephritis; PCD, plasma cell dyscrasia; CGS, chronic glomerulosclerosis; NGS, nodular glomerulosclerosis; TMA, thrombotic microangiopathy; DN, diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, mesangial proliferative glomerulonephritis; LN, lupus nephritis; ATIN, acute tubulointerstitial nephritis.

most common indication of biopsy in studies from Italy and France (28):

According to hygiene hypothesis, the increased exposure to bacterial antigens in developing countries may reflect a different pattern of response of the immune system, which translates in a different exacerbation of particular GDs [higher prevalence of MPGN, in developing areas, higher prevalence of IgA-nephropathy (IgAN), and MCD, in more developed countries (26,29)]. India has a growing burden of problems of either extremes of economical classes and hence it is not possible to extrapolate the data from other countries to India directly (30,31). Prevalence

of MPGN in our data was lower than that reported from developing countries like Romania (32). As cited above in India CKDu as a significant prevalence and most if not all are likely to be due to GDs that went undetected in early stages. As is shown in Table 7, our centre has reported higher prevalence of IgAN compared to our own earlier data as well as other regions of India. This may be a reflection of rather more vigorous biopsy practice in our institute in an effort to define the etiology of GD even when the clinical presentation is that of AUA or CKD. In both of these clinical syndromes and those with MH, IgAN was the most frequent diagnosis. Biopsy in CKD

**Figure 2.** Primary and secondary glomerular diseases.

**Table 6.** Frequencies of primary and secondary glomerular diseases

Biopsy diagnosis	Total	Primary n=1079 (%)	Secondary n=214 (%)
MCD/Normal	279	276 (25.58)	3 (1.40)
FSGS	163	160 (14.83)	3 (1.40)
MN	132	128 (11.86)	4 (1.80)
LN	181	0 (0)	181 (84.58)
IgAN/HSP	134	134 (12.42)	0 (0)
MPGN	95	84 (7.78)	11 (5.14)
DPGN	98	92 (8.53)	6 (2.80)
CresGN	79	73 (6.76)	6 (2.80)
PCD	29	29 (2.69)	0 (0)
CGS	19	19 (1.76)	0 (0)
NGS	4	4 (0.37)	0 (0)
MesPGN	16	16 (1.48)	0 (0)
TMA	29	29 (2.69)	0 (0)
ATN/ATIN	30	30 (2.78)	0 (0)
C1qN	1	1 (0.09)	0 (0)
Lithium toxicity	1	1 (0.09)	0 (0)
DN	1	1 (0.09)	0 (0)
Collapsing Glomerulopathy	1	1 (0.09)	0 (0)
Hypertensive nephrosclerosis	1	1 (0.09)	0 (0)

is considered to be controversial (26) but in our study definitive diagnosis was not possible in only 14% of cases while in the rest of the cases (86%) a definite etiology was found. The implications of a definitive diagnosis go beyond therapeutics to prognostication of recurrence after transplant if the progresses to ESRD. A population based urinary screening for school children has been recently completed in our institute (unpublished data as yet). This may be the first step in identifying the GN early in their course in our region.

The frequent histological patterns related to primary NS in adults in our study are similar to other studies like MCD, FSGS and MN. However, a significant proportion of IgAN was also presented as NS similar to our previous report and another report from south India but unlike some reports from other countries (16,18,26,33). We did not find an increase in prevalence of FSGS compared to our previous report (16,23), however the gap in the two studies of our institute is too small to define the trends of GD in eastern India. Other studies from India and other countries have reported an increasing prevalence of FSGS (4,19). FSGS of not otherwise specified (NOS) was the most common variant as earlier. The prevalence

**Table 7.** Comparison of our study with biopsy-proven glomerular disease spectrum seen in other centers in India and neighboring countries

Variables	Our Study	IPGMER Kolkata <sup>16</sup>	TITS, Asarwa <sup>23</sup>	Karachi, Pakistan <sup>24</sup>	RAMAIAH, Bangalore <sup>22</sup>	PGIMS, Rohtak <sup>21</sup>
Duration	2013-2015	2010-2012	2008-2013	2003-2012	2008-2013	2012
No. of biopsies	1293	666	335	435	661	75
Age (y)	29.7±15.24	28±14.62	7.91±3.04	40	42.8	-
PGD	83.45	79.13	81.79	-	42.3	61.3
SGD	16.55	20.83	16.12	-	-	16
MCD/normal	21.58	20.12	6.93	5	14.52	8.6
FSGS	12.61	18.02	8.02	8	5.45	6.8
MN	10.21	12.01	3.28	12	6.81	25.8
LN	14.00	15.32	7.76	6	3.18	12.1
IgAN/HSP	10.36	8.1	6.2	1.8	2.72	5.2
MPGN	7.35	5.25	13.5	8	4.08	17.2
DPGN	7.50	4.95	9.55	1.5	3.03	-
CresGN	6.11	7.51	10.94	4	3.18	8.6
PCD	2.24	1.2	0.89	4	0.30	6.8
CGS	1.47	3	-	-	3.48	-
NGS	0.31	0.45	-	-	0.00	-
MesPGN	1.24	0.6	24.45	-	0.00	-
TMA	2.24	1.5	6.26	-	0.30	-
ATN/ATIN	2.32	-	-	19.5	0.15	-
C1qN	0.08	0.15	-	-	0.00	-
Lithium toxicity	0.08	-	-	-	0.00	-
DN	0.08	-	-	7	16.64	1
Collapsing glomerulopathy	0.08	-	-	-	0.00	-
Hypertensive Nephrosclerosis	0.08	-	1.19	-	-	-

PGD, primary glomerular diseases; SGD, secondary glomerular diseases; MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; MN, membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch-Schönlein purpura; MPGN, membranoproliferative glomerulonephritis; DPGN, diffuse proliferative glomerulonephritis; CresGN, crescentic glomerulonephritis; PCD, plasma cell dyscracia; CGS, chronic glomerulosclerosis; NGS, nodular glomerulosclerosis; TMA, thrombotic microangiopathy; DN, diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, mesangial proliferative glomerulonephritis; LN, lupus nephritis; ATIN, acute tubulointerstitial nephritis.

Values indicate % of total biopsies.

**Table 8.** Comparison of our study with biopsy-proven glomerular disease spectrum seen in other centers across the globe

Variables	Our Study	IPGMER Kolkata <sup>16</sup>	TITS, Asarwa <sup>23</sup>	Karachi, Pakistan <sup>24</sup>
Duration	2013-2015	1994-2011	2010	1994-2003
No. of biopsies	1293	10,472	3,869	104
Age (y)	29.7±15.24	46.6	41.3 ± 15.4	
PGD (%)	83.45	57.8	55.2	
SGD (%)	16.55	29		
MCD/Normal (%)	21.58	6.1		1.92
FSGS (%)	12.61	6.9	5.1	20.1
MN (%)	10.21	7.1	10.2	10.5
LN (%)	14.00	7.1	5.4	7.69
IgAN/HSP (%)	10.36	20.5	30.4	25
MPGN (%)	7.35	3.2	2.4	5.76
DPGN (%)	7.50	0.8	0.8	7.69
CresGN (%)	6.11	6.9	6.4	
PCD (%)	2.24	3.7	1.4	
CGS (%)	1.47	-		
NGS (%)	0.31	-		
MesPGN (%)	1.24	3.3		
TMA (%)	2.24	-	0.2	
ATN/ATIN (%)	2.32	3.3		
C1qN (%)	0.08	-		
Lithium toxicity (%)	0.08	-		
DN (%)	0.08	4.1	4.7	
Collapsing glomerulopathy (%)	0.08			
Hypertensive nephrosclerosis (%)	0.08		3.8	

PGD, primary glomerular diseases; SGD, secondary glomerular diseases; MCD, minimal change disease; FSGS, focal segmental glomerulosclerosis; MN, membranous nephropathy; IgAN, IgA nephropathy; HSP, Henoch–Schönlein purpura; MPGN, membranoproliferative glomerulonephritis; DPGN, diffuse proliferative glomerulonephritis; CresGN, crescentic glomerulonephritis; PCD, plasma cell dyscrasia; CGS, chronic glomerulosclerosis; NGS, nodular glomerulosclerosis; TMA, thrombotic microangiopathy; DN, diabetic nephropathy; C1qN, C1q nephropathy; MesPGN, mesangial proliferative glomerulonephritis; LN, lupus nephritis; ATIN, acute tubulointerstitial nephritis.

\*Please refer to the details in the actual article given in reference as the categories may be overlapping and the actual % may hence differ.

of mesangioproliferative GN (MesPGN) is much lower in our data (1.24%) compared to that in the other regional studies from India (18,21,34). It has been almost double in number compared to previous study from our institute (16). Unlike our previous report where no case was given the diagnosis as C3 glomerulopathy (C3G), 5 cases in this study had a diagnosis consistent with C3G, which some of the MesGN cases were C3G.

According to available data, AKI has been associated with increased prevalence of bleeding complications (35) and this may be the reason of hesitation in performing renal biopsy in such settings yet renal biopsy in AKI has varied diagnosis and in about a third of the cases the clinical presentation may not correlate with the diagnosis (36). This is particularly true in case post-partum AKI. While other studies from our country and other neighboring countries (as shown in Tables 7 and 8) have not reported a significant prevalence of TMA except in children we have found this entity in 29% of all AKI cases. This difference may be due to referral bias where pregnant patient with hospital delivery may be recognized early, referred to nephrologist and readily biopsied. Awareness amongst primary care physicians and obstetricians regarding this

disease is still not satisfactory and majority patients are referred late (unpublished data of our institute). This highlights the importance of early renal biopsy in the usually devastating post-partum AKI for early diagnosis, treatment and to decide regarding the prospects of live renal transplant. Also, renal biopsy in AKI can diagnose otherwise covert plasma cell dyscrasia as in one of our cases. Since plasma cell dyscrasias are considered in the differential diagnosis of elderly patients with renal dysfunction, most are diagnosed without kidney biopsy. This is the most likely reason for low prevalence of cast nephropathy compared to other histological diagnoses in this study. The other etiologies of AKI/ANS were similar to those found by Beaman et al (35).

The most common PGD in most registries is IgAN (37) however in our data the most common PGD was MCD followed by FSGS. As discussed above this may be due to subclinical/covert presentation of IgAN. The prevalence of SGD has not changed and correlated well with other biopsy registries with LN being the most common SGD. Diabetic nephropathy is not biopsied routinely hence it contributed only a small proportion of cases. Other important finding was DPGN in elderly (>60 years). Occasionally this may



be the only presentation of an occult infection. The overall prevalence of DPGN has also increased almost three fold compared to our previous report. This may be due to increasing trend of biopsy in adults with infection related glomerulonephritis. As the prognosis is guarded in this group renal biopsy may prove helpful in diagnosis (38).

A limitation of the study is that EM was not done in all cases. However, in all cases where a definitive diagnosis could not be reached with LM and IF, EM was considered. Yet, the possibility of a different diagnosis after EM remains. Another limitation is that this is a single centre hospital-based biopsy registry so subclinical presentation of disease in the population as less likely to be reflected entirely in our data. Comparisons with other studies may not reflect the true difference in prevalence due to racial/genetic/environmental/socioeconomic causes but also those due to the differences in indications for renal biopsy.

### Conclusion

Nephrotic syndrome due to MCD is the dominating biopsy finding in this region. The most common cause of AUA was IgAN. The most common glomerular etiology for CKD was IgAN followed by crescentic GN most probably due to late referral. Vigorous biopsy practices as well as population based urinary screening programs may prove extremely help in identifying the etiology of CKDu in early stages. Although the prevalence of TMA is low, most of the cases had unsatisfactory recovery, emphasizing the early renal biopsy especially in the setting of pregnancy associated AKI for its recognition. MPGN is not as common as found in some developing countries. As compared to the last report from our institute it is seen that the prevalence of DPGN has increased almost three fold. Prevention of infections in elderly and vaccination may help in controlling DPGN in elderly who often have moderate to severe renal dysfunction and usually have guarded prognosis. The prevalence of SGD correlated well with other biopsy registries.

### Limitations of the study

This investigation was retrospective and was conducted in a single center. Besides, the study was cross-sectional without respect to treatment or follow up to find the final outcome of patients. Thus larger studies with multi-center basis are suggested.

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### Authors' contribution

CM complied, gathered and reviewed the data. SSD analyzed and wrote the primary manuscript. VD organized and analyzed data. AR and RP gave the original idea and have been mentors. AAK reported the renal biopsies. MP,

AB, VB, UD, KB, TDS, SD and DS were involved in patient care, procedure of renal biopsies and interpretation of renal biopsies. All authors read, revised, and approved the final manuscript.

### Conflicts of interest

The authors declare that they have no conflict of interest.

### Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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### Supplementary Materials

Supplementary file 1 contains Tables S1-S5.

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