# Clinicopathological spectrum of biopsy proven glomerular diseases reported by an academic centre in Sri Lanka

HD Wijesinghe[1], DR Wijayaratne[2], FN Nuha[1], GG Ranaweera[1], AAH Priyani[1], C Ratnayake[3], VN Gunasekera[4], ES Wijewickrama[2], R Lanerolle[2], MVC de Silva[1]

*[1]Department of Pathology, [2] Department of Clinical Medicine, Faculty of Medicine, University of Colombo, Sri Lanka*

*[3] Medical Research Institute, Colombo, Sri Lanka, [4] Lady Ridgeway Hospital, Colombo, Sri Lanka*

WCN24 – AB – 2249

## 1. Objective

- To describe the clinicopathological spectrum of biopsy proven glomerular disease (GD) reported at an academic centre in Sri Lanka.

## 2. Methods

- Records of all native kidney biopsies (adult and paediatric) reported at the Department of Pathology, Faculty of Medicine, University of Colombo Sri Lanka from January 2017 to October 2023 for which both light microscopy (LM) and immunofluorescence (IF) descriptions were available were reviewed.

## 3. Results

- 468(75 paediatric; 393 adult) biopsies included
- Mean age =36.5 ± 1.8 years
- Male: Female ratio= 1:1.03.
- Past history:
  - Diabetes = 24.4% (n=114)
  - Hypertension = 47.3% (n=221)
  - Systemic lupus erythematosus = 10.3% (n=48)
- **Commonest indication for biopsy** - nephrotic range proteinuria/nephrotic syndrome(NRP/NS) (n=249, 53.2%).
- **50%(n=234) had proliferative GDs** most of which were IC mediated (n=181;77.6%).

Figure 1: Clinical presentation and morphological pattern on light microscopy

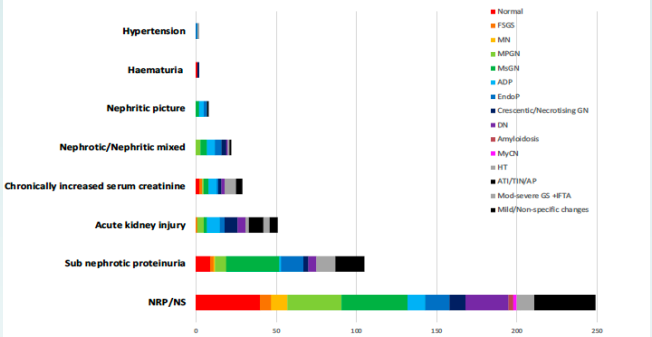


**Table 1: Morphological pattern on light microscopy and immunofluorescence pattern in proliferative glomerulonephritis**

| Morphological pattern | Immunofluorescence pattern | | | | | | | | | Total (Morphological pattern) |
|---|---|---|---|---|---|---|---|---|---|---|
| | Immune complex mediated | | | | C3 dominant | | Anti GBM | Mild/ trace IgM only | Neg IF | |
| | IgA[a] | LN | IRGN/?IRGN | NOS | IRGN/ ?IRGN | NOS | | | | |
| Membranoproliferative | 3 | 29 | 0 | 12 | 0/1 | 3 | 0 | 1 | 0 | 49 |
| Mesangioproliferative | 45[a] | 6 | 1/1 | 18 | 0 | 6 | 0 | 2 | 6 | 85 |
| Acute diffuse proliferative | 2[a] | 0 | 15/3 | 3 | 4[b]/4 | 2 | 0 | 0 | 0 | 33 |
| Endocapillary proliferative GN | 8[a] | 9 | 2[c]/5 | 1 | 2[b]/5 | 7 | 0 | 1 | 0 | 40 |
| Crescentic/ Necrotising GN | 3 | 1 | 2/3 | 9 | 0/1 | 0 | 2 | 0 | 6[c] | 27 |
| Total | 61 | 45 | 20/12 | 43 | 6/11 | 18 | 2 | 4 | 12 | 234 |

Most cases of NRP/NS showed non-IgA, non-lupus immune complex (IC) deposition.

The commonest cause of sub nephrotic range proteinuria was IgA nephropathy (n=39; 39%).

**Table 2: Clinical presentation and final diagnosis in cases with a definite/probable diagnosis**

| Clinical History | Normal LM and IF | MN | FSGS | IgA | IgA vasculitis | IC-NOS | LN | IRGN | PSGN | Possible IRGN | C3 DGN | anti GBM | Pauci-immune | DN | Amyloidosis | MyCN | ATI/ TIN/ AP | HT | CKD | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sub nephrotic proteinuria | 6 | 1 | 2 | 39 | 3 | 11 | 16 | 1 | 1 | 4 | 5 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 7 | 100 |
| Haematuria only | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| Nephritic picture | 1 | | 0 | 1 | 1 | 1 | 0 | 0 | 2 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 |
| NRP/NS | 36 | 11 | 7 | 34 | 0 | 44 | 29 | 4 | 2 | 7 | 11 | 2 | 1 | 25 | 3 | 2 | 0 | 2 | 5 | 226 |
| Nephrotic/ Nephritic mixed | 1 | 0 | 0 | 3 | 0 | 0 | 2 | 3 | 1 | 9 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 21 |
| Chronically increased serum creatinine | 3 | 0 | 2 | 3 | 0 | 1 | 1 | 5 | 0 | 1 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 1 | 4 | 25 |
| Acute kidney injury | 0 | 0 | 1 | 4 | 0 | 8 | 3 | 5 | 2 | 5 | 3 | 0 | 2 | 4 | 0 | 0 | 7 | 2** | 1 | 47 |
| Young hypertension | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 |
| Total | 48 | 12 | 12 | 84 | 4 | 65 | 51 | 18 | 8 | 28 | 22 | 2 | 6 | 35 | 3 | 2 | 7 | 7 | 17 | 431 |

Abbreviations: NRP/NS – nephrotic range proteinuria/nephrotic syndrome; LM – light microscopy; IF – immunofluorescence; MN – membranous nephropathy; FSGS – focal segmental glomerulosclerosis; IgA - IgA nephropathy; HSP – Henoch-Schoenlein purpura; IC NOS – immune complex mediated glomerulonephritis - not otherwise specified, LN – lupus nephritis; IRGN– post infectious glomerulonephritis; PSGN – post-streptococcal glomerulonephritis; C3 DGN – C3 dominant glomerulonephritis, anti GBM – anti glomerular basement membrane disease; DN – diabetic nephropathy; MyCN – myeloma cast nephropathy; HT – hypertensive renal changes; ATI, TIN. AP – acute tubular injury, tubulointerstitial nephritis, acute pyelonephritis; HT – hypertensive renal changes; CKD – chronic kidney disease

## 4. Conclusions

- The indication for kidney biopsy in more than half the patients was nephrotic range proteinuria.
- A diagnosis was not reached in a significant number, including cases that were normal on LM with negative IF, cases with IC deposition but no identified cause and cases with mild and/or non-specific changes.
- Further serological, microbiological and pathological evaluation and follow up data would be useful to further categorise cases.