

Histopathological Patterns of Glomerulonephritis in Renal Biopsies: A 5-Year Retrospective Study

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Abstract

Introduction: Glomerulonephritis (GN) is a major cause of chronic kidney disease worldwide, with marked geographic variability in histopathological patterns. Periodic regional evaluation of biopsy-proven GN is essential to understand epidemiologic trends and optimize management strategies.

Methods: This retrospective observational study included all native renal biopsies performed between January 2020 and December 2024 at a tertiary care center. Biopsies demonstrating glomerulonephritis were analyzed. Clinical data, laboratory parameters, and histopathological findings from light microscopy, immunofluorescence, and electron microscopy (where indicated) were reviewed. GN was classified into primary and secondary categories. Statistical analysis was performed using appropriate tests, with $p < 0.05$ considered significant.

Results: Of 312 native renal biopsies, 268 (85.9%) were diagnosed as GN. The mean age was 36.8 ± 14.7 years, with male predominance (60.4%). Nephrotic syndrome was the most common presentation (50.0%). Primary GN accounted for 64.9% of cases, while secondary GN comprised 35.1%. IgA nephropathy (19.4%) was the most frequent diagnosis, followed by focal segmental glomerulosclerosis (17.2%) and membranous nephropathy (14.2%). Lupus nephritis (15.3%) was the leading secondary cause. Rapidly progressive renal failure was strongly associated with pauci-immune crescentic GN and proliferative lupus nephritis ($p < 0.001$). Crescent formation was observed in 21.6% of cases, and moderate-to-severe interstitial fibrosis and tubular atrophy correlated with elevated serum creatinine ($p = 0.003$). An increasing trend in focal segmental glomerulosclerosis was noted over the study period.

Conclusion: IgA nephropathy and focal segmental glomerulosclerosis predominate among primary GN, whereas lupus nephritis remains the leading secondary cause. Continuous regional surveillance is necessary to monitor evolving trends and guide evidence-based nephrology practice.

Keywords: Glomerulonephritis, IgA nephropathy, Focal segmental glomerulosclerosis, Lupus nephritis, Membranous nephropathy, Crescentic glomerulonephritis.

Introduction and Background

Glomerulonephritis (GN) encompasses a heterogeneous group of immune-mediated disorders characterized by inflammation and structural injury of the renal glomeruli, frequently leading to significant morbidity and progression to chronic kidney disease (CKD). Globally, glomerular diseases remain among the leading causes of end-stage renal disease (ESRD), particularly in younger populations, thereby imposing a substantial clinical and socioeconomic burden [1,2]. Despite advances in immunopathology and molecular nephrology, renal biopsy continues to represent the gold standard for definitive diagnosis, prognostication, and therapeutic stratification in glomerular disorders [3].

The spectrum of GN demonstrates considerable geographic and temporal variability. Differences in ethnicity, environmental exposures, infection prevalence, socioeconomic conditions, and referral patterns influence the observed histopathological distribution across regions [4,5]. For example, IgA nephropathy is reported as the most common primary glomerulonephritis in many parts of East Asia and Europe, whereas focal segmental glomerulosclerosis (FSGS) has shown a rising incidence in North America and parts of Africa [6,7]. Similarly, secondary glomerular diseases such as lupus nephritis continue to represent a major proportion of biopsy-proven GN in younger female populations, particularly in developing countries [8]. These variations underscore the importance of region-specific epidemiological data derived from renal biopsy registries.

Renal biopsy evaluation integrates light microscopy (LM), immunofluorescence (IF), and, where available, electron microscopy (EM). Each modality contributes complementary diagnostic

information. LM provides architectural and cellular detail, allowing assessment of mesangial hypercellularity, endocapillary proliferation, segmental sclerosis, crescents, and chronicity markers such as interstitial fibrosis and tubular atrophy. IF facilitates identification of immune complex deposition patterns and complement activation, enabling differentiation between immune complex-mediated and pauci-immune processes. EM further refines diagnosis through localization of electron-dense deposits and ultrastructural assessment of podocyte injury [3,9]. Together, these tools enable precise classification according to established clinicopathologic frameworks, including the Oxford classification for IgA nephropathy and the ISN/RPS classification for lupus nephritis.

Beyond diagnostic categorization, histopathological patterns carry substantial prognostic implications. The degree of crescent formation, segmental sclerosis, interstitial fibrosis, and chronic vascular changes correlates with renal outcomes independent of clinical parameters [10]. In IgA nephropathy, for instance, the MEST-C scoring system has demonstrated reproducible association with disease progression [11]. Similarly, activity and chronicity indices in lupus nephritis guide therapeutic intensity and predict response to immunosuppressive therapy [12]. Therefore, understanding the local distribution of these patterns is essential for optimizing clinical decision-making and resource allocation.

In India and other low- and middle-income countries, data on biopsy-proven GN remain limited and often fragmented. Existing reports suggest shifting trends over recent decades, with increasing recognition of FSGS and persistent burden of infection-related and immune complex-mediated GN

[5,13]. However, institutional variations are considerable, and periodic reassessment is warranted to identify evolving epidemiologic patterns. Retrospective analyses of renal biopsy archives offer a pragmatic and methodologically robust approach to delineate these trends over defined time intervals.

A five-year retrospective evaluation provides sufficient duration to observe temporal fluctuations while maintaining consistency in diagnostic criteria and laboratory practices. Such a study allows characterization of demographic distribution, clinicopathologic correlation, and proportional frequency of primary versus secondary glomerular diseases. Additionally, it facilitates comparison with national and international registries, thereby contextualizing regional findings within the global nephrology landscape.

Given the pivotal role of histopathology in guiding management and predicting outcomes in GN, systematic documentation of biopsy patterns remains indispensable. The present study aims to analyze the histopathological spectrum of glomerulonephritis in native renal biopsies over five years at a tertiary care center, with emphasis on demographic distribution and clinicopathologic correlation. By providing contemporary regional data, this work seeks to contribute to the evolving epidemiological understanding of glomerular diseases and support evidence-based nephrology practice.

Materials and Methods

Study Design and Setting

This retrospective observational study was conducted in the Departments of Pathology at Government Medical College, Jammu. All native renal biopsies received over five years (January 2020 to December 2024) were reviewed. The study design and reporting were aligned with the

Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations to ensure methodological transparency and completeness [14].

Study Population

All consecutive native renal biopsies processed during the study period were screened for eligibility. Biopsies demonstrating histopathological evidence of glomerulonephritis were included. Adequacy of the specimen was defined by the presence of sufficient cortical tissue for diagnostic interpretation, preferably containing at least ten glomeruli on light microscopy or considered adequate by the reporting renal pathologist.

Exclusion criteria comprised transplant kidney biopsies, specimens showing isolated tubulointerstitial or vascular lesions without primary glomerular involvement, markedly inadequate tissue samples, and cases lacking essential clinical data. All eligible cases within the defined timeframe were included to minimize selection bias.

Clinical Data Collection

Clinical and laboratory information at the time of biopsy was retrieved from electronic medical records and biopsy requisition forms using a standardized data abstraction format. Variables recorded included age, sex, indication for biopsy, serum creatinine, estimated glomerular filtration rate (eGFR), quantitative proteinuria, urine microscopy findings, complement levels (C3, C4), autoimmune markers (ANA, anti-dsDNA, ANCA where indicated), and viral serology (HBsAg, anti-HCV, HIV).

Clinical syndromes were defined using established nephrology criteria. Nephrotic syndrome was defined as proteinuria greater than 3.5 g/day accompanied by hypoalbuminemia and edema. Nephritic

syndrome was defined by hematuria with variable proteinuria and hypertension. Rapidly progressive glomerulonephritis was defined as a rapid decline in renal function over weeks to months with crescent formation on biopsy [3,10].

Renal Biopsy Processing and Histopathological Evaluation

Percutaneous renal biopsies were performed under real-time ultrasound guidance using automated biopsy devices. Tissue cores were divided for light microscopy, immunofluorescence, and electron microscopy, where available.

For light microscopy, specimens were fixed in 10% neutral buffered formalin, routinely processed, and embedded in paraffin. Sections of 3–4 μm thickness were stained with hematoxylin and eosin, periodic acid–Schiff, Jones methenamine silver, and Masson's trichrome stains. Histological assessment included evaluation of the total number of glomeruli; presence of global or segmental sclerosis; mesangial and endocapillary proliferation; basement membrane thickening; crescents (cellular, fibrocellular, fibrous); interstitial fibrosis and tubular atrophy; and vascular changes. Chronicity parameters were graded semi-quantitatively.

Immunofluorescence studies were performed on frozen sections using antibodies against IgG, IgA, IgM, C3, C1q, and kappa and lambda light chains. Staining intensity was graded on a semi-quantitative scale (0 to 3+), and the pattern and distribution of deposits were documented. Electron microscopy was undertaken in selected cases, particularly where minimal change disease, early membranous nephropathy, or diagnostic uncertainty required ultrastructural confirmation [9].

Histopathological Classification

Glomerulonephritis was categorized into primary and secondary forms based on combined clinical, light microscopic, immunofluorescence, and ultrastructural findings. Primary glomerular diseases included minimal change disease, focal segmental glomerulosclerosis, IgA nephropathy, membranous nephropathy, and membranoproliferative glomerulonephritis. Secondary glomerular diseases included lupus nephritis, diabetic nephropathy with superimposed immune-mediated injury, infection-related glomerulonephritis, amyloidosis, and pauci-immune crescentic glomerulonephritis.

Lupus nephritis was classified according to the International Society of Nephrology/Renal Pathology Society (ISN/RPS) classification system [12]. IgA nephropathy cases were assessed using the Oxford MEST-C scoring system, where complete histologic parameters were available [11]. All biopsies were independently reviewed by two renal pathologists to reduce interobserver variability. In cases of discrepancy, a consensus diagnosis was established following joint review.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee of Government Medical College, Jammu. As the investigation involved retrospective analysis of archived biopsy specimens and medical records, the requirement for informed consent was waived. All patient identifiers were removed before data analysis to maintain confidentiality and comply with ethical standards.

Statistical Analysis

Data were analysed using IBM SPSS Statistics 25. Continuous variables were

expressed as mean \pm standard deviation or median (interquartile range), while categorical variables were summarized as frequencies and percentages. Associations

were assessed using Chi-square or Fisher's exact test as appropriate. A p-value <0.05 was considered statistically significant.

Results

Table 1: Distribution of various demographic and etiological variables.

| Variable | Value |
|---|-------------------|
| Mean age (years) | 36.8 \pm 14.7 |
| Age range (years) | 12–72 |
| Male, n (%) | 162 (60.4) |
| Female, n (%) | 106 (39.6) |
| Male: Female ratio | 1.5:1 |
| Median serum creatinine (mg/dL) | 1.9 (IQR 1.2–3.4) |
| Median proteinuria (g/day) | 3.8 (IQR 2.1–6.2) |
| Nephrotic syndrome, n (%) | 134 (50.0) |
| Nephritic syndrome, n (%) | 62 (23.1) |
| Rapidly progressive renal failure, n (%) | 38 (14.2) |
| Asymptomatic urinary abnormalities, n (%) | 21 (7.8) |
| Unexplained renal dysfunction, n (%) | 13 (4.9) |

The cohort demonstrated a predominance of young to middle-aged adults, with a clear male preponderance. Nephrotic syndrome constituted half of all biopsy indications, reflecting its central role in prompting histological evaluation. The median proteinuria exceeded nephrotic-range thresholds, while renal dysfunction at presentation was moderate but variable, indicating a heterogeneous clinical spectrum.

Table 2. Overall Histopathological Spectrum of Glomerulonephritis (n = 268)

| Category | Diagnosis | n (%) |
|-------------------------|--|-----------|
| Primary GN (64.9%) | IgA nephropathy | 52 (19.4) |
| | Focal segmental glomerulosclerosis | 46 (17.2) |
| | Membranous nephropathy | 38 (14.2) |
| | Minimal change disease | 24 (9.0) |
| | Membranoproliferative GN | 14 (5.2) |
| Secondary GN (35.1%) | Lupus nephritis | 41 (15.3) |
| | Infection-related GN | 19 (7.1) |
| | Diabetic nephropathy (immune-mediated) | 16 (6.0) |
| | Pauci-immune crescentic GN | 11 (4.1) |
| | Amyloidosis | 7 (2.6) |

Primary glomerulonephritis accounted for nearly two-thirds of cases. IgA nephropathy emerged as the most frequent diagnosis, followed closely by focal segmental glomerulosclerosis. Among secondary causes, lupus nephritis predominated. The proportional distribution reflects patterns commonly reported in contemporary tertiary-care biopsy series, with immune-complex-mediated diseases forming the bulk of cases.

Table 3. Age-wise Distribution of Major Glomerulonephritis Subtypes

| Diagnosis | ≤20 yrs | 21–40 yrs | 41–60 yrs | >60 yrs | Total |
|------------------------|---------|-----------|-----------|---------|-------|
| IgA nephropathy | 6 | 29 | 14 | 3 | 52 |
| FSGS | 5 | 24 | 13 | 4 | 46 |
| Membranous nephropathy | 1 | 9 | 19 | 9 | 38 |
| Minimal change disease | 7 | 11 | 5 | 1 | 24 |
| Lupus nephritis | 4 | 23 | 11 | 3 | 41 |

IgA nephropathy and FSGS were most prevalent in the 21–40 year age group, consistent with their known epidemiological predilection for young adults. Membranous nephropathy showed increasing frequency with advancing age. Minimal change disease demonstrated a higher representation in younger patients, whereas lupus nephritis predominantly affected women in the reproductive age group.

Table 4. Clinicopathological Correlation of Major Diagnoses

| Diagnosis | Nephrotic (%) | Nephritic (%) | RPGN (%) |
|--|---------------|---------------|----------|
| IgA nephropathy | 30.8 | 42.3 | 11.5 |
| FSGS | 63.0 | 10.9 | 8.7 |
| Membranous nephropathy | 68.4 | 5.3 | 2.6 |
| Minimal change disease | 79.2 | 4.2 | 0 |
| Lupus nephritis | 48.8 | 24.4 | 39.0 |
| Pauci-immune GN | 18.2 | 9.1 | 72.7 |
| p < 0.001 for the association between clinical presentation and histological diagnosis | | | |

Nephrotic syndrome was strongly associated with minimal change disease, membranous nephropathy, and FSGS. Nephritic presentations were most frequently observed in IgA nephropathy. Rapidly progressive renal failure showed a significant association with pauci-immune crescentic GN and proliferative lupus nephritis. These correlations were statistically significant, reinforcing the predictive value of clinicopathologic integration.

Table 5. Chronicity Markers and Crescent Formation

| Parameter | n (%) |
|--|-----------|
| Any crescent formation | 58 (21.6) |
| Cellular crescents | 31 (11.6) |
| Fibro cellular/Fibrous crescents | 27 (10.1) |
| IFTA >25% | 72 (26.9) |
| IFTA >50% | 29 (10.8) |
| Moderate-to-severe IFTA significantly correlated with elevated serum creatinine at presentation (p = 0.003). | |

Approximately one-fifth of biopsies demonstrated crescent formation, highlighting a substantial burden of aggressive disease. Chronicity markers, particularly interstitial fibrosis and tubular atrophy, were observed in over one-quarter of cases and were significantly associated with impaired renal function at presentation. These findings underscore the prognostic relevance of chronic histological injury.

Discussion

This five-year retrospective analysis delineates the contemporary histopathological spectrum of biopsy-proven glomerulonephritis in a tertiary care setting. The findings demonstrate that primary glomerulonephritis constitutes the majority of cases, with IgA nephropathy emerging as the most frequent diagnosis, followed closely by focal segmental glomerulosclerosis and membranous nephropathy. Among secondary causes, lupus nephritis represented the predominant entity. The observed distribution reflects evolving global trends while also highlighting region-specific characteristics.

The predominance of IgA nephropathy in our cohort aligns with reports from several Asian and European registries, where it represents the leading primary glomerular disease [6,15]. However, the proportion in our series remains lower than that reported in East Asian populations, suggesting potential differences in genetic susceptibility, environmental exposures, and biopsy practices. Focal segmental glomerulosclerosis constituted the second most common primary glomerulonephritis

and demonstrated a rising trend over the study period. This increase parallels observations from North American and African cohorts, where FSGS has shown progressive expansion in incidence, potentially related to obesity, metabolic factors, and improved recognition of podocytopathies [7,16].

Membranous nephropathy accounted for a substantial proportion of nephrotic presentations, particularly in older individuals, consistent with established epidemiological patterns [17]. The age-associated distribution observed in our study reinforces the need for age-stratified diagnostic suspicion when evaluating nephrotic syndrome in adults. Minimal change disease, although less frequent overall, remained an important cause of nephrotic syndrome, particularly in younger patients.

Secondary glomerulonephritis comprised over one-third of cases, with lupus nephritis as the leading diagnosis. The strong female predominance and peak incidence in reproductive age groups are concordant with known disease demographics [8,12]. Class IV lupus nephritis was the most frequent

histological subtype, reflecting the biopsy indication pattern in symptomatic and proliferative disease. The substantial burden of proliferative lupus nephritis underscores the ongoing need for early detection and aggressive immunomodulatory therapy in this population.

Infection-related glomerulonephritis demonstrated a gradual decline over the five-year interval. This observation may reflect improved infection control measures and earlier antimicrobial intervention, though residual burden persists. Comparable trends have been documented in certain developing regions undergoing epidemiological transition [18]. Nevertheless, infection-related disease continues to contribute significantly to secondary GN in resource-limited settings.

Clinicopathological correlation in the present study revealed expected but clinically meaningful associations. Nephrotic syndrome was predominantly linked to minimal change disease, membranous nephropathy, and FSGS, whereas nephritic presentations were more common in IgA nephropathy and infection-related GN. Rapidly progressive renal failure strongly correlated with pauci-immune crescentic GN and proliferative lupus nephritis. These findings reinforce the diagnostic utility of integrating clinical syndromes with histological evaluation and are consistent with established pathological paradigms [10,19].

Chronicity markers, particularly interstitial fibrosis and tubular atrophy (IFTA), were observed in over one-quarter of biopsies and showed a significant association with elevated serum creatinine at presentation. The prognostic importance of chronic tubulointerstitial injury is well recognized and often supersedes glomerular morphology in predicting long-term renal

outcomes [20]. Crescent formation was present in approximately one-fifth of cases, emphasizing the prevalence of aggressive immune-mediated injury within the cohort.

The rising proportion of FSGS observed over the study period merits attention. Whether this reflects a true epidemiological shift, increased metabolic comorbidity, referral bias, or heightened diagnostic recognition remains uncertain. Longitudinal registry-based studies are necessary to clarify temporal causation. Nonetheless, the trend mirrors patterns described in multicenter analyses across diverse populations [16,21].

The strengths of this study include comprehensive histopathological evaluation incorporating light microscopy, immunofluorescence, and electron microscopy when indicated, dual pathologist review to minimize interobserver variability, and systematic clinicopathologic correlation. The five-year duration allowed assessment of temporal patterns while maintaining consistency in diagnostic protocols.

Certain limitations warrant consideration. The retrospective design inherently limits causal inference and may introduce documentation bias. As a single-center study, referral patterns may influence disease distribution, potentially limiting generalizability. Additionally, long-term renal outcomes were not analyzed, precluding prognostic modeling. Despite these constraints, the dataset provides meaningful regional insight into the evolving spectrum of glomerulonephritis.

In conclusion, this five-year biopsy-based study demonstrates that IgA nephropathy and FSGS are the predominant primary glomerular diseases, while lupus nephritis remains the leading secondary cause in our

setting. The observed clinicopathological correlations and temporal trends are consistent with global patterns yet retain distinct regional characteristics. Continued surveillance through institutional and national renal biopsy registries is essential to monitor epidemiological shifts, refine diagnostic strategies, and guide resource allocation in nephrology practice.

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