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Correlates of Clinical Presentations and Histopathological Findings of Glomerular Disease Patients having Proteinuria

Gadhave Ram Panditrao[1], Vidya Nand[2]

[1]Junior Resident, Department of General Medicine, [2]Professor, Department of Nephrology,
Shri Ram Murti Smarak Institute of Medical Sciences, Bareilly, India.

Corresponding Author: Gadhave Ram Panditrao

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ABSTRACT

Background: Glomerular diseases (GD) are a major cause of chronic kidney disease and end-stage renal failure, commonly presenting with proteinuria, edema, hypertension, and renal dysfunction. Renal biopsy plays a crucial role in identifying the underlying histopathological subtype and guiding disease-specific management.
Objectives: To evaluate the prevalence, clinicopathological correlation, and histopathological spectrum of glomerular diseases in patients presenting with proteinuria.
Methodology: This hospital-based cross-sectional observational study was conducted over (2024-25) at SRMS Institute of Medical Sciences, Bareilly, Uttar Pradesh. A total 80 patients undergoing renal biopsy over the 18-month study period were included for this study using a complete enumeration approach. Relevant clinical history and a provisional diagnosis were documented for each patient. Prior to performing the renal biopsy, all patients underwent a series of investigations.
Results: Majority of participants belonged to the 31–39 years age group. There was a male preponderance in the study cohort (61% males). The mean proteinuria levels (3.78 ± 0.93 g/day) varied significantly among groups ($p < 0.001$). Edema was nearly universal (94%); Hypertension was significantly associated with diagnosis ($p < 0.001$). Oliguria and hematuria were also significantly associated with the diagnostic category ($p < 0.001$ each). Total cholesterol levels showed a significant difference as well ($p < 0.001$), with the highest values recorded in membranous nephropathy (334.5 mg/dL) and FSGS (305.7 mg/dL), consistent with hyperlipidemia commonly associated with nephrotic presentations.
Conclusions: Glomerular diseases in proteinuric adults predominantly affected young to middle-aged males, with IgA nephropathy being the most common histopathological diagnosis. Significant associations were observed between clinical features, biochemical parameters, and histopathological subtypes. Renal biopsy remains essential for accurate diagnosis and appropriate management of glomerular diseases.
Keywords: Proteinuria, glomerular disease, nephropathy, renal biopsy, IgA nephropathy.

INTRODUCTION

Glomerular disease (GD), an important cause of end-stage kidney disease, imposes a huge morbidity/ mortality burden on the health services in developing countries. The GD diagnostic challenge for pathologists / nephrologists has been simplified by the advances in renal biopsy, light microscopy

(LM) and immunofluorescence. GD histological diagnosis classification can meaningfully separate and categorize these patients to make an informed therapeutic prognosis allowing clinicians to assign lesion-specific therapy. [1] Tropical and temperate countries vary regarding various aspects of GD (etiology, histologic spectrum, prevalence, epidemiology, natural history, and indications for renal biopsy), affecting the results.[2]

On urinalysis, severe or ongoing proteinuria is an important sign of glomerulonephritis, and the commonest abnormal finding / disease manifestation indicating renal injury / glomerular dysfunction. For halting the kidney disease progression, nephrologists use the proteinuria degree to develop therapeutic strategies, monitor treatment response, and outcome prediction. For studying heavy proteinuria, accompanied with edema, renal biopsy is frequently used to select suitable therapy, detect various histopathological features, and get an idea about its prognosis. Still, the pathophysiology of proteinuria-induced GD progression is unknown. [3-9] Individuals having kidney-related morbidity, with substantial proteinuria and glomerular disorders may experience gradual renal impairment, cardiovascular problems, and poor quality of life.[10]

GD encompasses a diverse array of conditions, ranging from primary glomerulonephritis to secondary involvement in systemic diseases such as diabetes mellitus, and lupus nephritis. The disruption of the glomerular filtration barrier results in proteinuria.[11]

GD patients commonly clinically manifest as hypertension, proteinuria, edema, and hematuria. The GD progression and renal outcomes often correlate with persistence and severity of proteinuria. Timely diagnosis and intervention are facilitated by understanding the clinical nuances of these presentations.[12]

Renal biopsy / histopathological examination facilitates accurate diagnosis GD and evaluation of the extent of glomerular damage for developing its rational treatment. There is a global trend of increasing focal segmental glomerulosclerosis (FSGS) incidence with considerable heterogeneity in the clinical spectrum and histopathological patterns. [13-15] This study aims to unravel the complexities / mechanisms of GD in patients with proteinuria, providing insights into their accurate diagnosis and effective management, with following objectives: To evaluate the prevalence, clinicopathological correlation, and histopathological spectrum of glomerular diseases in patients presenting with proteinuria.

MATERIALS & METHODS

This hospital-based cross-sectional observational study was conducted over a period of 18 months (April 2024 to September 2025) in the Department of General Medicine at SRMS Institute of Medical Sciences, Bareilly, Uttar Pradesh. Patients having proteinuria >1 g/24 h with or without evidence of hypertension, deranged renal function or active sediment on urine microscopy, aged 18-80 years were included in the study. Patients suffering from chronic liver disease, chronic kidney disease, coagulopathy, single kidney, shrunken kidney, congestive cardiac failure and autosomal dominant polycystic kidney disease were excluded.

The total 80 patients undergoing renal biopsy over the 18-month study period were included for this study using a complete enumeration approach. No predetermination of sample size was done. A structured proforma for data collection was used to capture key parameters from patients: clinical history, physical examination, blood pressure monitoring, edema grading, complete hemogram, coagulation profile, urine routine and microscopy, 24-hour urine protein and creatinine, serum creatinine, urea, electrolytes, lipid profile, ultrasound abdomen. Renal biopsy was performed under ultrasound guidance using a 16-gauge automated biopsy gun, post-procedure monitoring for 24 hours including vitals,

hematuria, hematocrit, and ultrasound screening for complications.

All biopsies were analyzed under light microscopy and, where indicated, immunofluorescence. Cases were classified into primary and secondary glomerular diseases based on histological diagnosis. Relevant clinical history and a provisional diagnosis were documented for each patient. Prior to performing the renal biopsy, all patients underwent a series of investigations including complete hemogram, coagulation profile, urine routine and microscopy, 24-hour urinary protein and creatinine estimation, creatinine clearance, renal function tests, serum electrolytes, lipid profile, hepatitis B surface antigen, anti-HCV antibody, HIV antibody, and abdominal ultrasound. ANA testing was performed when clinically indicated. Percutaneous renal biopsy was carried out under ultrasound guidance using a 16-gauge automated biopsy gun.

All procedures were performed under strict aseptic conditions. Post-biopsy, patients were closely monitored for 24 hours for any complications, with regular assessment of pulse, blood pressure, urine for hematuria, hematocrit, and follow-up ultrasound. The indication for biopsy and the histological diagnosis was recorded for each case. Glomerular diseases were further classified into primary and secondary types. Final diagnosis was based on a combination of clinical features and histopathological findings. Before commencing the study, written informed consent was obtained from all participants. Data collection was carried out using a structured proforma, only patients who met the predefined inclusion criteria were selected for the study.

Ethical consideration: Ethical approval was obtained from the appropriate ethics committee before proceeding with the research. Ethical clearance certificate dated 06/06/2024 with Ref. No./SRMSIMS/ECC/ 2024/40.

Statistical Analysis

The collected data was entered into Microsoft Excel 2019 and analyzed using SPSS software (version 21.0). Descriptive statistics was used to summarize the demographic and clinical characteristics of the participants. Continuous variables were presented as means ± standard deviations and categorical variables were shown in frequencies and percentages along with presented using tables, bar diagrams, and pie charts where appropriate.[16] Chi-square test was used to assess association between clinical and histopathological variables. ANOVA test was used to assess the mean difference of continuous parameters between more than three groups. P-value <0.05 was considered statistically significant.

RESULT

Figure 1 shows that the age of participants ranged from 22-66 years, with the majority belonging to the 31–39 years age group. There was a male preponderance in the study cohort (61% males and 39% females).

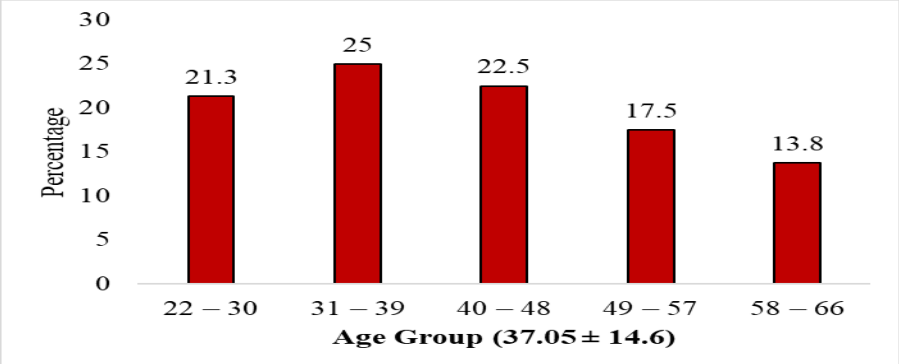

Figure 1. Age Distribution of the Patients (n = 80)

Edema was the most common clinical manifestation (94%), followed by frothy urine (46%), oliguria (27.5%), hematuria (21%), reduced appetite (20%), and abdominal pain (10%). Hypertension was the most common comorbidities (76%), followed by diabetes mellitus (11%) and coronary artery disease (2.5%). Mean proteinuria was 3.78 ± 0.93 g/day and serum creatinine was 1.98 ± 0.86 mg/dL. The lipid profile showed high total cholesterol, LDL, and triglycerides with low HDL levels. Coagulation parameters (PT, INR, and APTT) were within normal ranges in most participants (Table 1).

Table 1: Biochemical and Coagulation Parameters in the Study Population (n = 80)

Parameter	Mean ± SD	Median	Range (Min–Max)
Proteinuria (g/24 h)	3.78 ± 0.93	3.68	2.38 – 7.33
Serum Creatinine (mg/dL)	1.98 ± 0.86	2.2	1.20 – 3.50
Total Cholesterol (mg/dL)	299.74 ± 55.15	313.3	183.70 – 393.30
LDL Cholesterol (mg/dL)	183.53 ± 36.64	179.2	114.20 – 277.10
HDL Cholesterol (mg/dL)	34.61 ± 7.10	34	8.70 – 55.10
Triglycerides (mg/dL)	270.30 ± 73.78	268.6	134.60 – 433.70
Prothrombin Time (sec)	13.44 ± 1.59	13.6	9.92 – 16.55
INR	1.18 ± 0.14	1.17	0.80 – 1.61
APTT (sec)	33.79 ± 4.81	32.96	25.79 – 48.97

HBsAg was positive in 7.5% and Anti-HCV in 5% of patients, while most were negative for both markers. Mean right and left kidney lengths were 10.74 ± 0.84 cm and 10.96 ± 0.89 cm, respectively, indicating normal kidney size in most patients. Proteinuria was the most common indication for biopsy (42.5%), followed by nephritic syndrome (30%) and AKI/unexplained renal failure (27.5%). IgA nephropathy was the most frequent diagnosis (31.3%), followed by lupus nephritis (16.3%), focal segmental glomerulosclerosis (15%), membranous nephropathy (13.8%), and diabetic nephropathy (11.3%). MPGN/C3GN accounted for 6.3%, while minimal change disease, renal amyloidosis, and acute tubular necrosis were less common. A significant positive correlation was found between 24-hour proteinuria and serum creatinine ($r = 0.606$, $p < 0.001$), suggesting worsening renal function with increasing proteinuria. Total cholesterol also showed significant correlations with both proteinuria and serum creatinine ($p < 0.001$) (Table 2).

Table 2: Correlation between Biochemical Parameters (n = 80)

Variables		Correlation coefficient (r)	p-value
Proteinuria (g/24h)	Serum Creatinine (mg/dL)	0.606	< 0.001
Proteinuria (g/24h)	Total Cholesterol (mg/dL)	0.399	< 0.001
Serum Creatinine (mg/dL)	Total Cholesterol (mg/dL)	–0.464	< 0.001

Note: Karl-Pearson correlation coefficient applied.

Edema was present in nearly all patients (94%) and showed no significant association with diagnosis ($p = 0.441$). Hypertension was significantly associated with the diagnostic category ($p < 0.001$), particularly in lupus nephritis and diabetic nephropathy. Oliguria and hematuria were also significantly associated with diagnosis ($p < 0.001$ each), being more common in lupus nephritis and MPGN/C3GN. Frothy urine showed a borderline significant association ($p = 0.048$), occurring more frequently in membranous nephropathy, MPGN, and minimal change disease (Table 3).

Table 3: Clinico-Pathological association between Clinical Features and Histopathological Diagnosis (n = 80)

Clinical Feature	Edema present, n (%)	Hypertension, n (%)	Oliguria, n (%)	Hematuria, n (%)	Frothy urine, n (%)
Diabetic Nephropathy (n=9)	8 (88.9%)	8 (88.9%)	4 (44.4%)	3 (33.3%)	4 (44.4%)
FSGS (n=12)	11 (91.7%)	10 (83.3%)	7 (58.3%)	5 (41.7%)	6 (50.0%)
IgA Nephropathy (n=25)	24 (96.0%)	16 (64.0%)	7 (28.0%)	9 (36.0%)	8 (32.0%)
Lupus Nephritis (n=13)	12 (92.3%)	12 (92.3%)	10 (76.9%)	7 (53.8%)	5 (38.5%)
Membranous Nephropathy (n=11)	10 (90.9%)	9 (81.8%)	5 (45.5%)	3 (27.3%)	6 (54.5%)
MPGN / C3GN (n=5)	5 (100%)	3 (60.0%)	5 (100%)	2 (40.0%)	4 (80.0%)
Minimal change disease (n=2)	2 (100%)	2 (100%)	1 (50.0%)	0 (0%)	1 (50.0%)
Amyloidosis (n=2)	2 (100%)	2 (100%)	2 (100%)	1 (50.0%)	1 (50.0%)
ATN (n=1)	1 (100%)	1 (100%)	1 (100%)	0 (0%)	1 (100%)
Total (n=80)	**75 (93.8%)**	**62 (77.5%)**	**22 (27.5%)**	**17 (21.3%)**	**37 (46.3%)**
p-value	0.441	< 0.001	< 0.001	< 0.001	0.042

Note: Fisher's exact test applied (cell frequency less than 5).

Table 4 shows biochemical parameters differed significantly across histopathological diagnoses ($p < 0.001$). The highest proteinuria was seen in amyloidosis and MPGN/C3GN, while serum creatinine was highest in amyloidosis and lupus nephritis, indicating greater renal impairment. Total cholesterol levels were highest in membranous nephropathy and FSGS, consistent with nephrotic syndrome–related hyperlipidemia.

Table 4: Comparison of Biochemical Parameters across Histopathological Diagnoses (n = 80)

Histopathological Diagnosis	Proteinuria (g/24 h)	Serum Creatinine (mg/dL)	Total Cholesterol (mg/dL)
Diabetic Nephropathy	3.85 ± 0.65	1.82 ± 0.55	258.4 ± 36.9
FSGS	4.10 ± 0.82	2.05 ± 0.63	305.7 ± 28.2
IgA Nephropathy	4.25 ± 0.91	2.26 ± 0.81	210.6 ± 24.4
Lupus Nephritis	4.60 ± 0.77	3.10 ± 0.72	271.9 ± 22.5
Membranous Nephropathy	3.95 ± 0.48	2.15 ± 0.36	334.5 ± 18.1
MPGN / C3GN	4.70 ± 1.05	2.95 ± 0.81	268.2 ± 27.6
Amyloidosis	5.10 ± 1.42	3.48 ± 0.10	255.0 ± 18.5
Minimal change disease	3.40 ± 0.55	1.60 ± 0.35	290.0 ± 25.0
ATN	3.20 ± 0.00	2.90 ± 0.00	250.0 ± 0.00
p-value	**< 0.001**	**< 0.001**	**< 0.001**

Note: ANOVA test applied for p value

DISCUSSION

The mean age of the study population was 37.05±14.6 years, indicating that most patients were young to middle-aged adults. These findings align with Sarkar et al who reported a similar mean age of 36.8 years among Indian adults with nephrotic syndrome.[17] Comparable results were also observed by Mundi et al, where 60% of patients were between 20–50 years.[18] A recent study on north Indian patients also identified a comparable pattern, suggesting that glomerular disease predominantly affects younger adults in developing countries, likely due to infections and socioeconomic stressors.[19] This age trend may differ in Western cohorts, where degenerative or metabolic glomerulopathies predominate in older individuals.[20]

In this study, males accounted for 61.3% of the cases, confirming a clear male preponderance. This pattern mirrors findings from Ganesh et al and Elavarasan et al (2019), where males constituted 58–

64% of biopsy-proven glomerular diseases.[21,22] Similar trends were found globally that IgA nephropathy and FSGS had higher male frequency, linked to hormonal and immunologic differences.[23] In the present study, edema (94%) was the most frequent symptom, followed by frothy urine (46%) and oliguria (27.5%). Similar findings were reported by Rathi et al [2] and Mundi et al[18] where edema was the predominant manifestation. Amatya & Pant reported edema in 94% of nephrotic presentations in a Nepalese cohort, confirming this trend across regions.[24] Variability in hematuria frequency (21% in this study) could be explained by differences in the proportion of IgA nephropathy cases, where hematuria is characteristic.[21] Hypertension was the most prevalent co-morbidity (77.5%), followed by diabetes mellitus (10%). Similar rates were reported by Ganesh et al and Sarkar et al.[17,21] Yap et al emphasized that hypertension accelerates renal injury in glomerulonephritis and worsens proteinuria outcomes.[24] The modest diabetes rate reflects biopsy selection bias, as many diabetics with classic nephropathy patterns are often not biopsied.[25]

The mean proteinuria (3.78 g/day) and creatinine (1.98 mg/dL) values in this study were consistent with those reported by Sarkar et al and Elavarasan et al[17,22] Hyperlipidemia, with total cholesterol averaging 299.7 mg/dL, mirrors nephrotic biochemical patterns seen in previous studies. [26,27] These hyperlipidemic state results from hepatic overproduction of lipoproteins as compensation for hypoalbuminemia.[27] Coagulation parameters were normal in most cases indicating the absence of nephrotic coagulopathy in early-stage disease.[17]

HBsAg (7.5%) and Anti-HCV (5%) positivity aligns with prior regional data (4–9%) reported by Varshney et al[14] These findings reaffirm the role of viral infections as secondary causes of glomerulonephritis, particularly membranous and membranoproliferative subtypes. Screening for viral markers remains vital in biopsy workup to avoid misclassification of infection-related GN as primary disease.[29] Proteinuria and nephritic syndrome were the most common biopsy indications (42.5% and 30%), consistent with previous studies which established patterns in nephrology practice where these syndromes prompt histological evaluation to identify underlying glomerular pathology. [30-32]

Koubar et al confirmed that AKI/unexplained renal failure mains the most cost-effective and prognostically relevant biopsy criterion globally.[33] In the present study, IgA nephropathy (31.3%) was the most common histological diagnosis, followed by lupus nephritis (16.3%), FSGS (15%), and membranous nephropathy (13.8%). This aligns with Ganesh et al study where IgA nephropathy predominated.[21] More recent data from Chugh and Shakhuja show IgAN as a leading cause of primary glomerulonephritis worldwide, supporting the global shift from membranous GN to IgA dominance.[34] Regional variations persist and Nand and Kumari noted higher FSGS in Western and urban Indian cohorts due to obesity and hypertension.[32] The present study found a significant positive correlation between 24-hour proteinuria and serum creatinine ($r = 0.606$, $p < 0.001$), indicating that higher protein excretion is associated with declining renal function. This supports earlier findings that proteinuria is both a marker and driver of renal damage, leading to tubulointerstitial fibrosis and nephron loss. Similar trends were reported by Erkan and Abbate et al, who emphasized proteinuria pathogenic role in CKD progression.[35, 11]

A positive correlation between proteinuria and total cholesterol ($r = 0.399$) suggests that nephrotic-range protein loss stimulates hepatic lipid synthesis, consistent with Politano et al[10] Conversely, the negative correlation between serum creatinine and cholesterol ($r = –0.464$) may reflect metabolic decline in advanced renal disease, as described by Wingo and Clapp.[36] In this

study, edema was the most frequent clinical manifestation (93.8%) across all histopathological types, reflecting its universal association with proteinuria. This finding corresponds with earlier studies by Sarkar et al and Mundi et al, who reported edema as a predominant presenting feature in more than 90% of nephrotic patients. [17,18] Hypertension was significantly associated with specific diagnoses ($p < 0.001$), particularly lupus nephritis and diabetic nephropathy, suggesting vascular and immune-mediated mechanisms contributing to renal injury. This observation is consistent with findings by Koubar et al, who highlighted endothelial dysfunction and glomerular hyperfiltration as major pathways linking hypertension to glomerulopathies.[33] Moreover, oliguria and hematuria showed significant associations with lupus nephritis and MPGN/C3GN, paralleling reports by Ganesh et al (2018), who found these symptoms indicative of active proliferative lesions.[21] The borderline relationship of frothy urine ($p = 0.048$), more common in membranous nephropathy and MPGN, corresponds with Politano et al, who described it as a clinical indicator of nephrotic-range proteinuria.[10] This study further found significant variation in biochemical parameters across histopathological categories ($p < 0.001$). Proteinuria levels were highest in amyloidosis (5.10 g/24 h) and MPGN/C3GN (4.70 g/24 h), reflecting severe glomerular barrier damage and nephrotic-range protein loss. Similar observations were made by Elavarasan et al, who reported marked proteinuria in FSGS and amyloidosis due to extensive podocyte injury.[22] Elevated serum creatinine in amyloidosis (3.48 mg/dL) and lupus nephritis (3.10 mg/dL) in this study indicates advanced renal dysfunction, consistent with Ganesh et al, who found lupus nephritis associated with higher creatinine due to immune-complex–mediated glomerular damage.[21] The highest cholesterol levels in membranous nephropathy (334.5 mg/dL) and FSGS (305.7 mg/dL) align with findings from Politano et al, attributing hyperlipidemia to hepatic compensation for hypoalbuminemia in nephrotic syndromes.[10]

CONCLUSION

The majority of patients were young to middle-aged adults with a male predominance. IgA nephropathy was the most common histopathological diagnosis, followed by lupus nephritis, focal segmental glomerulosclerosis (FSGS), and membranous nephropathy, reflecting the evolving pattern of glomerular diseases observed in both national and global studies. Edema and hypertension were the most frequent clinical manifestations, while proteinuria showed a significant positive correlation with serum creatinine, indicating an association between increasing protein loss and worsening renal function. Biochemical parameters varied significantly across histopathological subtypes. Proteinuria and serum creatinine levels were highest in amyloidosis and lupus nephritis, suggesting greater renal impairment in these conditions, whereas hypercholesterolemia was most pronounced in membranous nephropathy and FSGS, consistent with nephrotic presentations. Significant associations of hypertension, hematuria, and oliguria with specific histopathological diagnoses highlight the importance of clinical and biochemical evaluation in predicting underlying glomerular pathology. These findings emphasize the crucial role of renal biopsy in the accurate diagnosis and management of glomerular diseases in patients presenting with proteinuria.

Recommendations:

Routine renal biopsy should be considered in patients presenting with unexplained proteinuria or nephrotic/nephritic features to establish an accurate diagnosis and guide therapy. Future multi-centric studies and long-term follow-up are recommended to validate these associations and assess treatment outcomes across histological subtypes.

Declaration by Authors
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