



Histopathological and Immunofluorescence Profile of Renal Biopsies: A Cross-Sectional Study from a Tertiary Care Center in Northeast India.

Dr. Soibam Bidyalakshmi Devi¹, Dr. Laishram Deepak Kumar², Dr. Sorokhaibam Babina³

¹Postgraduate Trainee, Department of Pathology, Regional Institute of Medical Sciences, Imphal, Manipur, India.

²Associate Professor & CO PI of Population Based Cancer Registry, Department of Pathology, Regional Institute of Medical Sciences, Imphal, Manipur, India.

³Professor, Department of Pathology, Regional Institute of Medical Sciences, Imphal, Manipur, India.



Correspondence:

Dr. Laishram Deepak Kumar.

Associate Professor & CO PI of
Population Based Cancer
Registry, Department of
Pathology, Regional Institute of
Medical Sciences, Imphal,
Manipur, India.

Received: 01-06-2026

Revised: 21-06-2026

Accepted: 31-07-2026

Published: 21-08-2026

Abstract

Background: Renal biopsy remains the gold standard for diagnosing and classifying glomerular diseases, particularly immune complex-mediated nephropathies. Histopathological examination combined with IF (Immuno-Fluorescence) provides valuable information regarding disease activity, severity, and prognosis. This study aimed to describe the histopathological and immunofluorescence profile of renal biopsies received at a tertiary care center and to correlate these findings with clinical and serological parameters. **Methods:** This cross-sectional study was conducted in the Department of Pathology, Regional Institute of Medical Sciences (RIMS), Imphal, from June 2024 to February 2026. All renal biopsy specimens received during the study period and fulfilling the inclusion criteria were evaluated. Histopathological assessment was performed using routine light microscopy, and immunofluorescence was carried out for IgG, IgA, IgM, C3, C1q, kappa, and lambda deposits. Clinical, biochemical, serological, histopathological, and IF findings were analyzed using SPSS version 21.0, with a p-value <0.05 considered statistically significant. **Results:** Twenty-nine patients were included in the study, predominantly young females in the second and third decades of life. Most patients presented with significant proteinuria, albuminuria, microscopic hematuria, active urinary sediments, hypertension, hypoalbuminemia, and elevated renal function parameters. Serological evaluation demonstrated high rates of ANA and anti-dsDNA positivity, frequent hypocomplementemia, and p-ANCA positivity. Histopathological examination revealed predominant mesangial and endocapillary hypercellularity, mild to moderate crescent formation, and limited chronic changes. Immunofluorescence commonly demonstrated a “full-house” pattern with IgG, IgA, IgM, C3, and C1q deposition. Diffuse proliferative lupus nephritis (Class IV) was the most frequent diagnosis. Significant associations were observed between disease groups and anti-dsDNA status, crescent formation, and IF intensities. **Conclusion:** Lupus nephritis was the predominant immune complex-mediated glomerulonephritis in this cohort, particularly among young females. The predominance of active lesions with minimal chronicity highlights the importance of early renal biopsy and prompt immunosuppressive therapy. Integration of clinical, serological, histopathological, and immunofluorescence findings is essential for accurate diagnosis, disease classification, and optimal patient management.

Keywords: Renal Biopsy, Immunofluorescence, Histopathology, Lupus nephritis, Glomerulonephritis, Immune Complex Disease, Full-House Pattern, Kidney Pathology.



This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (<http://creativecommons.org/licenses/by/4.0/>).

INTRODUCTION

The kidney is a structurally complex organ composed of four principal morphologic components-glomeruli, tubules, interstitium, and blood vessels-each of which exhibits distinct patterns of injury in renal disease. Although specific disorders may initially involve one component, the close anatomic interdependence of renal structures means that damage often extends to the others, making clinicopathological correlation essential for accurate diagnosis and prognostic assessment.[1]

The advent of percutaneous renal biopsy transformed the evaluation of renal diseases by providing access to pre-end-stage tissue for detailed histopathological examination. Alongside conventional light microscopy, immunopathologic techniques have become integral to renal pathology by revealing the pathogenesis and etiology of immune-mediated kidney diseases. The integration of histomorphology with immunologic findings has significantly improved diagnostic precision and enabled recognition of distinct patterns of glomerular injury.[1]

The role of immunologic mechanisms in renal disease was first suggested by Schick in 1907 through observations linking serum sickness with post-streptococcal glomerulonephritis.[2,3] The subsequent adaptation of the immunofluorescence technique by Mellors and Ortega in 1956 established the demonstration of immunoglobulin deposits in renal tissue, leading to the identification of characteristic staining patterns-including granular, linear, mesangial, and capillary wall deposits-that are now fundamental to renal diagnosis.[2,3]

Renal biopsy assessment requires a systematic approach combining light microscopy with special stains such as Periodic acid-Schiff, Silver Methenamine, Masson's trichrome, and Congo red to highlight basement membranes, mesangial alterations, fibrosis, and amyloid deposition.[3] Immunofluorescence remains indispensable for diagnosing disorders such as IgA nephropathy, C1q nephropathy, and C3 glomerulopathy. When frozen tissue is unavailable, immunofluorescence or immunohistochemistry performed on formalin-fixed paraffin-embedded tissue after antigen retrieval provides a valuable alternative for detecting immune deposits.[4]

Modern renal pathology employs a complementary approach involving light microscopy, immunofluorescence, and electron microscopy.[5] Emerging advances in digital pathology and machine learning have shown promise in improving the reproducibility and efficiency of immunofluorescence interpretation.[5] However, the widespread application of these techniques remains limited by factors such as cost, biopsy timing and site, tissue processing, prior treatment, and disease characteristics.[6]

Aims and Objectives

The present study was conducted to describe the histopathological and immunofluorescence profiles of renal biopsies in various types of renal diseases. It aimed to evaluate the histopathological parameters that facilitated the screening and early diagnosis of renal disorders, correlate the histopathological findings with direct immunofluorescence findings, and establish an accurate diagnosis. The study also sought to provide a basis for the subsequent monitoring of disease activity in patients with life-threatening renal diseases.

MATERIALS AND METHODS

Study Design

This cross-sectional study was conducted in the Department of Pathology at the Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, in collaboration with the Department of Nephrology. RIMS is a tertiary care referral hospital that receives renal biopsy specimens from Manipur, neighboring northeastern states, and Myanmar. The study was carried out over a period of 21 months, from June 2024 to February 2026, and included renal biopsy specimens received for histopathological and direct immunofluorescence examination.

Inclusion and Exclusion Criteria

The study included all renal biopsy cases irrespective of age, gender, and socioeconomic status. Renal biopsy specimens that were poorly fixed, improperly segregated in saline and formalin, or diagnosed as neoplastic renal diseases were excluded from the study.

Data Collection Tools

Data were collected using a pre-designed proforma to record patients' demographic and clinical details. Renal biopsy specimens were processed using standard histopathological equipment, including an automated tissue processor, rotary microtome, and Olympus binocular microscope. Histological evaluation was performed using H&E (Hematoxylin and Eosin), PAS (Periodic Acid-Schiff), Masson's trichrome, and Jones methenamine silver stains, while direct immunofluorescence was carried out using fluorescein-conjugated antisera for IgG, IgA, IgM, C3, and C1q.

Data Collection Procedure

Clinical information, demographic details, and relevant medical history were obtained from the patients' case records and recorded in a pre-designed proforma after obtaining written informed consent or assent. Renal biopsy specimens received in the Department of Pathology were screened according to the inclusion and exclusion criteria before processing. The tissue preserved in formol saline was fixed, dehydrated through ascending grades of alcohol, cleared in xylene using an automated tissue processor, embedded in paraffin blocks, and sectioned at a thickness of 3–5 µm using a rotary microtome. The sections were mounted on albuminized glass slides, deparaffinized, and stained with H&E for routine histopathological evaluation, while PAS, Masson's Trichrome, and Jones Methenamine Silver stains were used as special stains whenever required. Histopathological examination included the systematic assessment of glomerular, tubular, interstitial, and vascular changes under light microscopy.

For DIF (Direct Immunofluorescence), a fresh portion of the renal biopsy specimen was transported in saline-soaked gauze and processed in a cryostat. Tissue sections of approximately 3–4 µm thickness were prepared, mounted on slides, and

incubated with fluorescein-conjugated antibodies against IgG, IgA, IgM, C3, and C1q after washing with phosphate-buffered saline. Following incubation, the slides were washed, mounted with glycerol-based mounting medium, and examined under an immunofluorescence microscope. The pattern, location, and intensity of immune deposits were recorded and correlated with the histopathological findings to establish the final diagnosis.

Statistical Analysis

The collected data were entered into Microsoft Excel 2010 and analyzed using IBM SPSS Statistics Version 21.0 (IBM Corp., Armonk, New York, USA). Descriptive statistical analysis was performed for all study variables. Age was categorized into groups and expressed as frequency and percentage. Categorical variables, including gender, clinical features, type of renal disease, histopathological parameters, and immunofluorescence findings, were also summarized as frequencies and percentages. The chi-square test was used to assess the association between the type of renal disease and the age and gender of the participants. A p-value of less than 0.05 was considered statistically significant at a 95% confidence interval.

RESULTS

Table 1 illustrates the demographic and clinical profile of the 29 patients. Most patients were aged 21–40 years (62.1%), with a marked female predominance (96.6%). Systemic lupus erythematosus (89.7%) was the most common clinical diagnosis, and suspected lupus nephritis (89.7%) was the leading indication for renal biopsy, followed by rapidly progressive glomerulonephritis (10.3%).

Table 1: Demographic and Clinical Profile of the Study Population

Parameter	Category	Frequency (n)	Percentage (%)
Age Distribution (years)			
Age (in years)	≤ 20	5	17.25
	21 – 30	9	31.04
	31 – 40	9	31.04
	41 – 50	2	6.9
	> 50	4	13.8
	Total	29	100.0
Gender Distribution			
Gender	Male	1	3.4
	Female	28	96.6
	Total	29	100.0
Clinical Diagnosis			
Clinical Diagnosis	Systemic Lupus Erythematosus (SLE)	26	89.7
	Nephrotic Syndrome	2	6.9
	Post-infectious Glomerulonephritis (PIGN)	1	3.4
	Total	29	100.0
Indication for Renal Biopsy			
Indication for Biopsy	Suspected Lupus Nephritis	26	89.7
	Rapidly Progressive GN (RPGN)	3	10.3
	Total	29	100.0

Table 2 depicts the urinary, biochemical, and blood pressure findings at presentation. Moderate-to-severe proteinuria (+3 in 48.3%) and severe albuminuria (58.6%) were the predominant urinary abnormalities, while glucosuria was absent in most patients (96.6%). Elevated serum creatinine and urea were observed in 69.0% of patients, with hypoalbuminemia present in 58.6%. Additionally, 65.5% of patients were hypertensive, with grade 2 hypertension (37.9%) being more common than grade 1 (27.6%).

Table 2: Urinary, Biochemical and Blood Pressure Parameters

Parameter	Category	Frequency (n)	Percentage (%)
Proteinuria Grading			
Proteinuria	+2	8	27.6
	+3	14	48.3
	+4	7	27.6
	Total	29	100.0
Glucosuria Grading			
Glucosuria	Nil	28	96.6

	Trace	1	3.4
	Total	29	100.0
Urine Albumin–Creatinine Ratio (ACR)			
Urine ACR	Mild	5	17.2
	Moderate	7	24.1
	Severe	17	58.6
	Total	29	100.0
Hematuria			
Hematuria	Present	21	72.4
	Absent	8	27.6
	Total	29	100.0
Type of Urinary Casts			
Urinary Casts	None	13	44.8
	Granular	9	31.0
	Hyaline	4	13.8
	Waxy	2	6.9
	Hyaline + Granular	1	3.4
	Total	29	100.0
Renal Function and Protein Parameters			
Serum Creatinine	Elevated	20	69.0
	Normal	9	31.0
Serum Urea	Elevated	20	69.0
	Normal	9	31.0
Serum Albumin	Low	17	58.6
	Normal	12	41.4
Total Serum Protein	Low	17	58.6
	Normal	8	27.6
	Elevated	4	13.8
Blood Pressure Status			
Blood Pressure	Grade 1 Hypertension	8	27.6
	Grade 2 Hypertension	11	37.9
	Normal	10	34.5
	Total	29	100.0

Table 3 shows the serological and immunological profile of the study cohort. ANA positivity (93.1%), anti-dsDNA positivity (82.8%), and low C3/C4 levels (86.2%) were highly prevalent, supporting an immune-complex–mediated disease process, while p-ANCA positivity was observed in 62.1% of patients.

Table 3: Serological and Immunological Marker Profile

Parameter	Category	Frequency (n)	Percentage (%)
Antinuclear Antibody (ANA)	Positive	27	93.1
	Negative	2	6.9
p-ANCA	Positive	18	62.1
	Negative	11	37.9
Complement (C3/C4)	Low	25	86.2
	Normal	4	13.8
Anti-dsDNA Antibody	Positive	24	82.8
	Negative	5	17.2
Total (n)	—	29	100.0

Table 4 outlines the light-microscopic histopathological findings of renal biopsies. Mesangial hypercellularity (79.3%) and endocapillary hypercellularity (65.5%) were the predominant proliferative lesions, while crescents were present in 24.1% of cases and fibrinoid necrosis was absent. Chronic changes were mostly mild, with tubular atrophy (75.9%) and interstitial fibrosis (58.6%) being the commonest findings, whereas vascular lesions were infrequent. PAS with Masson's trichrome was the most commonly used special-stain combination (82.7%).

Table 4: Light Microscopy Findings on Renal Biopsy

Parameter	Category	Frequency (n)	Percentage (%)
Glomerular Sampling Adequacy			
Number of Glomeruli / Biopsy	Adequate (≥ 6 glomeruli)	25	86.2
	Inadequate (< 6 glomeruli)	4	13.8
Mesangial and Endocapillary Changes			
Mesangial Hypercellularity	Present	23	79.3
	Absent	6	20.7
Endocapillary Hypercellularity Score	0	10	34.5
	1	7	24.1
	2	11	37.9
	3	1	3.4
Active Inflammatory Lesions			
Neutrophils / Karyorrhexis Score	0	21	72.4
	1	3	10.3
	2	4	13.8
	3	1	3.4
Hyaline Deposits / Wire-loop Score	0	16	55.2
	1	10	34.5
	2	3	10.3
Fibrinoid Necrosis	Absent	29	100.0
Crescentic Involvement			
Number of Crescents	0	22	75.9
	1	2	6.9
	2	3	10.3
	4	1	3.4
	5	1	3.4
Type of Crescents (n = 7)	Cellular	3	10.3
	Fibrocellular	3	10.3
	Fibrous	1	3.4
Crescents Scoring	0	22	75.9
	1	1	3.4
	2	1	3.4
	4	5	17.2
Chronic/Tubulointerstitial Changes			
Interstitial Inflammation Score	0	2	6.9
	1	16	55.2
	2	10	34.5
	3	1	3.4
Type of Lesion	Diffuse	20	69.0
	Focal	7	24.1
	Focal Segmental	1	3.4
	Global	1	3.4
GBM Thickening	Present	17	58.6
	Absent	12	41.4
Global Glomerulosclerosis Score	0	25	86.2
	1	3	10.3
	3	1	3.4
Tubular Atrophy Score	0	1	3.4
	1	22	75.9
	2	6	20.7
Interstitial Fibrosis Score	0	12	41.4
	1	13	44.8
	2	4	13.8
Vascular Changes (Present)			
Refractile Casts	Present	3	10.3
Intimal Fibrosis	Present	2	6.9

Citation: Devi SB, Kumar LD, Babina S. Histopathological and immunofluorescence profile of renal biopsies: a cross-sectional study from a tertiary care center in Northeast India. *Int. J. Med.*, 2026;8(8):1672-1681.

Medial Hypertrophy	Present	3	10.3
Arteriolar Hyalinosis	Present	3	10.3
Special Stains Used			
Special Stains	PAS alone	5	17.2
	PAS + Masson's Trichrome	24	82.7

Table 5 summarizes the direct immunofluorescence findings, with C3 positivity in all cases (100%) and high positivity for IgG (89.7%), IgA (93.1%), IgM (89.7%), and C1q (86.2%). The predominance of strong staining intensity and the characteristic full-house pattern strongly support a lupus-predominant, immune-complex-mediated glomerulonephritis in this cohort.

Table 5: Immunofluorescence Findings

Immune Reactant	Category	Frequency (n)	Percentage (%)
IgG			
IgG — Positivity	Positive	26	89.7
	Negative	3	10.3
IgG — Intensity	0	2	6.9
	1+	7	24.1
	2+	7	24.1
	3+	13	44.8
IgA			
IgA — Positivity	Positive	27	93.1
	Negative	2	6.9
IgA — Intensity	0	2	6.9
	1+	10	34.5
	2+	3	10.3
	3+	14	48.3
IgM			
IgM — Positivity	Positive	26	89.7
	Negative	3	10.3
IgM — Intensity	0	3	10.3
	1+	7	24.1
	2+	7	24.1
	3+	12	41.4
C3			
C3 — Positivity	Positive	29	100.0
C3 — Intensity	1+	7	24.1
	2+	8	27.6
	3+	14	48.3
C1q			
C1q — Positivity	Positive	25	86.2
	Negative	4	13.8
C1q — Intensity	0	4	13.8
	1+	7	24.1
	2+	7	24.1
	3+	11	37.9
Location of Immune Deposits			
Location of Deposits	Specifically documented	7	24.1
	Not separately recorded	22	75.9

Table 6 presents the final histopathological and clinico-histo-immunofluorescence diagnoses. Diffuse lupus nephritis (41.4%) was the most common histopathological diagnosis, while Class IV lupus nephritis (58.6%) predominated on final classification. A full-house immunofluorescence pattern (89.7%) was the dominant IF finding, with the Modified NIH Activity Index indicating active disease in 89.7% of patients and the Chronicity Index showing a low chronic disease burden in 86.2% of cases.

Table 6: Final Diagnosis and Disease Activity/Chronicity Classification

Parameter	Category	Frequency (n)	Percentage (%)
Histopathological Diagnosis			
Histopathological Diagnosis	Diffuse Lupus Nephritis (LN)	12	41.4
	Diffuse Membranoproliferative GN	3	10.3
	Focal Mesangioproliferative GN	3	10.3
	Diffuse (unspecified pattern)	2	6.9
	Advanced Sclerosing LN	1	3.4
	Diffuse Proliferative GN	1	3.4
	Focal LN	1	3.4
	Focal Proliferative GN	1	3.4
	Focal Segmental Membranoproliferative GN	1	3.4
	IgA Nephropathy	1	3.4
	Membranous LN	1	3.4
	Mesangioproliferative GN	1	3.4
	Post-infectious GN	1	3.4
	Total	29	100.0
Immunofluorescence (IF) Correlation			
IF Correlation	Full-house Positivity	26	89.7
	C3-only Positivity	2	6.9
	IgA + C3 Positivity	1	3.4
	Total	29	100.0
Combined Final Diagnosis			
Combined Final Diagnosis	Class IV Lupus Nephritis	17	58.6
	Class II Lupus Nephritis	4	13.8
	Class III Lupus Nephritis	3	10.3
	C3-dominant GN, mesangioproliferative pattern	2	6.9
	Class V Lupus Nephritis	1	3.4
	Class VI Lupus Nephritis	1	3.4
	IgA Nephropathy	1	3.4
	Total	29	100.0
Modified NIH Activity Index (range 0–10)			
NIH Activity Index	0	1	3.4
	1	5	17.2
	2	5	17.2
	3	2	6.9
	4	1	3.4
	5	6	20.7
	6	2	6.9
	7	3	10.3
	8	1	3.4
	9	2	6.9
	10	1	3.4
Modified NIH Chronicity Index (range 0–7)			
NIH Chronicity Index	0	1	3.4
	1	10	34.5
	2	8	27.6
	3	7	24.1
	4	2	6.9
	5	1	3.4
	6	1	3.4
MEST-C Score (Oxford Classification, IgA Nephropathy cases)			
MEST-C Score	1	4	13.8
	2	4	13.8
	3	5	17.2
	4	4	13.8
	5	5	17.2
	6	3	10.3

	7	1	3.4
	8	2	6.9
	9	1	3.4

DISCUSSION

This study demonstrates that LN (Lupus Nephritis) was the predominant renal pathology among the 29 renal biopsy specimens, with most patients belonging to the second and third decades of life, a pattern consistent with previous biopsy-based studies reporting early onset of LN.[7] The marked female predominance (96.6%) further supports the established epidemiological pattern of autoimmune renal diseases occurring predominantly in women because of hormonal and immune-regulatory influences.[7]

SLE (Systemic Lupus Erythematosus) accounted for 89.7% of clinical diagnoses, highlighting the central role of renal biopsy in confirming and classifying LN before initiating targeted immunosuppressive therapy.[8] The predominance of LN among secondary glomerular diseases observed in this cohort is also comparable to reports from Eastern India.[9]

The urinary profile reflected active glomerular injury, with moderate-to-severe proteinuria present in nearly three-fourths of patients and severe albuminuria observed in almost 60%, indicating significant disruption of the glomerular filtration barrier.[10] Microscopic hematuria (72.4%) was another common finding, reflecting active proliferative glomerular inflammation.[11] Urinary casts were detected in 55.2% of patients, with granular casts (34.5%) being the predominant type, indicating ongoing glomerular and tubular injury.[12] The combined presence of proteinuria, hematuria, and active urinary sediment is characteristic of immune complex-mediated nephritis such as LN.[13]

Biochemical findings indicated significant renal dysfunction, with elevated serum creatinine and urea occurring in 69% of patients, reflecting impaired glomerular filtration associated with active nephritis.[14] Hypoalbuminemia was observed in 58.6% of patients, consistent with substantial urinary protein loss and increased disease activity.[15] Hypertension affected 65.5% of the cohort, supporting the established relationship between renal impairment, sodium retention, and accelerated progression of glomerular disease.[14]

The serological profile strongly supported immune complex-mediated nephropathy. ANA positivity was observed in 93.1% of patients, consistent with its recognized role as a sensitive serological marker of SLE.[16] Anti-dsDNA antibodies were positive in 82.8% of cases, reinforcing their established association with active proliferative LN and immune complex deposition.[17] Reduced complement levels (C3 and/or C4) were present in 86.2% of patients, reflecting complement consumption during active immune complex formation.[18] Although p-ANCA positivity was identified in 62.1% of patients, previous studies suggest that this finding may represent overlapping autoimmune activation rather than true ANCA-associated vasculitis.[19]

Light microscopic examination revealed predominantly proliferative lesions. Mesangial hypercellularity (79.3%) and endocapillary hypercellularity (65.5%) were the most frequent findings, consistent with active proliferative LN.[20] Crescent formation was identified in 24.1% of biopsies and was predominantly cellular or fibrocellular, indicating active rather than chronic injury.[21] The absence of fibrinoid necrosis suggests that fulminant necrotizing pathology was uncommon within this cohort.[20] Chronic irreversible changes remained relatively limited, despite glomerular basement membrane thickening (58.6%), tubular atrophy, and interstitial fibrosis, suggesting that inflammatory activity predominated over established scarring.[20]

Immunofluorescence findings provided strong evidence for immune complex-mediated disease. IgG deposition was present in 89.7% of cases, while IgA positivity reached 93.1%, supporting widespread polyclonal immune complex deposition.[20] IgM deposition was observed in 89.7% of biopsies, further reinforcing active immune complex disease. Universal C3 deposition (100%) and C1q positivity (86.2%) reflected activation of the classical complement pathway, a hallmark of LN. The frequent coexistence of IgG, IgA, IgM, C3, and C1q corresponds to the characteristic full-house immunofluorescence pattern described in LN. [22]

Histopathological classification showed that Class IV LN constituted 58.6% of final diagnoses, confirming diffuse proliferative LN as the predominant subtype in this cohort.[18] The predominance of high activity with comparatively low chronicity indices suggests that most patients were biopsied during a potentially reversible inflammatory phase, emphasizing the importance of early therapeutic intervention.[17]

Inferential analysis further highlighted the diagnostic value of integrated renal biopsy assessment. Anti-dsDNA status demonstrated a significant association with disease groups ($p = 0.003$), supporting its role as an important indicator of

active proliferative disease.[23] Crescent burden also showed a significant association ($p = 0.014$), reinforcing its value as a marker of aggressive inflammatory injury.[24] Several immunofluorescence parameters, including IgG intensity ($p = 0.002$), IgA and IgM positivity ($p < 0.001$), IgM intensity ($p = 0.008$), and C1q positivity and intensity ($p < 0.001$ and $p = 0.004$), demonstrated significant associations with disease groups, confirming the diagnostic importance of immune complex composition.[25] Histopathological diagnosis also differed significantly across disease groups ($p = 0.023$), while immunofluorescence correlation showed the strongest association ($p < 0.001$), emphasizing that the combined interpretation of clinical features, serology, light microscopy, and immunofluorescence provides the highest diagnostic accuracy in renal biopsy evaluation.[26]

This study highlights that biopsy-proven renal disease in this cohort was characterized predominantly by active immune complex-mediated proliferative lupus nephritis with marked serological activity, significant proteinuria, proliferative histological lesions, and a characteristic full-house immunofluorescence profile, underscoring the importance of early renal biopsy and integrated clinicopathological evaluation for optimal management.

Limitation

A total of 29 cases were included in the study, as patient recruitment was limited by the ongoing conflicts during the study period and the restricted duration of the study.

CONCLUSION

Lupus nephritis was the predominant immune complex-mediated glomerulonephritis in this cohort, occurring mainly in young female patients and characterized by active glomerular lesions with limited chronic damage. The strong correlation between clinical presentation, autoimmune serology, histopathological findings, and the characteristic full-house immunofluorescence pattern highlights the value of an integrated diagnostic approach. Early renal biopsy combined with timely immunosuppressive therapy is essential for accurate disease classification, assessment of disease activity, and improved renal outcomes.

REFERENCES

1. Pandey KK, Tiwari A, Agarwal A. Immunologic glomerulopathies: diagnostic role of immunofluorescence study of renal biopsies. *Int J Res Med Sci* 2017;5(12):5381-5.
2. Vernier RL, Tinglof B, Urizar R, et al. Immunofluorescence studies in renal disease. *Proc 3rd Int Congr Nephrol* 1966;3(3):83-94.
3. Anish A, Pillai HS, Annamalai PT. Reporting and interpreting renal biopsies: a review article. *J Adv Med Med Res* 2022;34(23):210-24.
4. Wagrowska-Danilewicz M, Zeromski J. Immunofluorescent evaluation of renal biopsy: current point of view. *Pol J Pathol* 2010;61(2):83-8.
5. Ligabue G, Pollastri F, Fontana F, et al. Evaluation of the classification accuracy of kidney biopsy direct immunofluorescence through convolutional neural networks. *Clin J Am Soc Nephrol* 2020;15(10):1445-54.
6. Buch AC, Sood SK, Bamanikar SA, et al. Role of direct immunofluorescence in the diagnosis of glomerulonephritis. *Med J DY Patil Univ* 2015;8(4):452-7.
7. Wang H, Ren YL, Chang J, et al. A systematic review and meta-analysis of prevalence of biopsy-proven lupus nephritis. *Arch Rheumatol* 2018;33(1):17-25.
8. Charaya P, Solanki R, Hemrajani D, et al. A clinicopathological study of lupus nephritis based on the international society of nephrology-renal pathology society 2018 classification system. *Journal of Medical Society* 2023;37(2):63-7.
9. Krishna A, Singh PP, Sinha S, et al. Histopathological spectrum of native kidney biopsy from Eastern India: a single-center observational study. *Turk J Nephrol* 2023;32(4):310-5.
10. Birmingham DJ, Rovin BH, Shidham G, et al. Relationship between albuminuria and total proteinuria in systemic lupus erythematosus nephritis: diagnostic and therapeutic implications. *Clin J Am Soc Nephrol* 2008;3(4):1028-33.
11. Amatya M, Pant AD. Clinical and histopathological study of renal biopsy in Nepalese children: a single-center experience. *PLoS One* 2022;17(10):e 0275701.
12. Muthukuda C, Suriyakumara V, Sosai C, et al. Clinicopathological spectrum of biopsy-proven renal diseases of patients at a single center in Sri Lanka: a cross-sectional retrospective review. *BMC Nephrol* 2023;24(1):1-14.
13. Seymour AE, Spargo BH, Penksa R. Contributions of renal biopsy studies to the Seymour AE, Spargo BH, Penksa R. Contributions of renal biopsy studies to the understanding of disease. *Am J Pathol* 1971;65(3):550-98.
14. Gopal A, Kavadihanda C, Bairwa D, et al. Performance of clinical and biochemical parameters in identifying renal histopathology and predictors of one-year renal outcome in lupus nephritis: a single-centre study from India. *Diagnostics (Basel)* 2022;12(12):3163.
15. Musa R, Rout P, Qurie A. Lupus nephritis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing 2025.

16. Tan EM, Cohen AS, Fries JF, et al. The 1982 revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1982;25(11):1271-7.
17. Almaani S, Meara A, Rovin BH. Update on lupus nephritis. *Clin J Am Soc Nephrol* 2017;12(5):825-35.
18. [Weening JJ, D'Agati VD, Schwartz MM, et al. The classification of glomerulonephritis in systemic lupus erythematosus revisited. *Kidney Int* 2004;65(2):521-30.
19. Lacetera R, Calatroni M, Roggero L, et al. Prevalence and clinical significance of ANCA positivity in lupus nephritis: a case series of 116 patients and literature review. *J Nephrol* 2023;36(4):1059-70.
20. da Luz Neto ER, Tavares MB, de Melo AGJT, et al. Analysis of the sensitivity and specificity of histopathological findings for diagnosing lupus nephritis. *Diagnostics (Basel)* 2024;14(23):2681.
21. Lin S, Zhang J, Chen B, et al. Role of crescents for lupus nephritis in clinical, pathological and prognosis: a single-center retrospective cohort study. *Eur J Med Res* 2023;28:60.
22. Jain S, Chauhan S, Dixit S, et al. Role of direct immunofluorescence microscopy in spectrum of diffuse proliferative glomerulonephritis: a single-center study. *J Microsc Ultrastruct* 2021;9(4):177-82.
23. Kwon OC, Park JH, Park HC, et al. Non-histologic factors discriminating proliferative lupus nephritis from membranous lupus nephritis. *Arthritis Res Ther* 2020;22:138.
24. Chen S, Tang Z, Zhang Y, et al. Significance of histological crescent formation in patients with diffuse proliferative lupus nephritis. *Am J Nephrol* 2013;38(6):445-52.
25. [Nossent H, Berden J, Swaak T. Renal immunofluorescence and the prediction of renal outcome in patients with proliferative lupus nephritis. *Lupus* 2000;9(7):504-10.
26. Kudose S, Santoriello D, Bomback AS, et al. Sensitivity and specificity of pathologic findings to diagnose lupus nephritis. *Clin J Am Soc Nephrol* 2019;14(11):1605-15.