

## A Study to Assess the Predictors of Poor Renal Responses in Lupus Nephritis in a Teaching Hospital in North Bengal

Srijana Pradhan<sup>1</sup>, Abhed Biswas<sup>2</sup>, Pasang Lahmu Sherpa<sup>3</sup>, Saptadeep Misra<sup>4</sup>

<sup>1</sup>Junior Resident, MBBS, Department of Medicine, North Bengal Medical College and Hospital, Siliguri, West Bengal 734012

<sup>2</sup>Assistant Professor, MD Medicine, Department of Medicine, North Bengal Medical College and Hospital, Siliguri, West Bengal 734012

<sup>3</sup>Associate Professor, MD Medicine, Department of Medicine, North Bengal Medical College and Hospital, Siliguri, West Bengal 734012

<sup>4</sup>MBBS, MD (General Medicine), Post Graduate Trainee, Department of General Medicine, North Bengal Medical College and Hospital, Siliguri, West Bengal 734012

Received: 01-08-2025 / Revised: 16-09-2025 / Accepted: 28-10-2025

Corresponding Author: Dr. Saptadeep Misra

Conflict of interest: Nil

### Abstract

**Introduction:** Complement activation, inflammatory cytokine release, autoantibody formation (especially anti-dsDNA antibodies), and immunological dysregulation all play intricate roles in the pathophysiology of lupus nephritis.

**Aims:** This study aims to evaluate the demographic, socioeconomic, clinical, and laboratory profiles of patients with systemic lupus erythematosus (SLE) presenting with lupus nephritis and to identify potential predictors of poor renal response among these patients in a tertiary care teaching institute in North Bengal.

**Materials & Methods:** This observational, ambispective cohort, non-interventional study was conducted at the Rheumatology OPD, Medicine OPD, and IPD of the Department of Medicine, North Bengal Medical College and Hospital, Darjeeling. The study was carried out over 18 months (1st July 2023 to 31st December 2024) and included a total of 60 patients.

**Result:** In our study of 60 patients, those with good outcomes had similar baseline SLEDAI scores ( $10.75 \pm 5.58$ ) compared to patients with poor outcomes ( $11.78 \pm 5.56$ ;  $p = 0.6106$ ). At 3 months, disease activity remained comparable between the good ( $3.57 \pm 7.22$ ) and poor outcome groups ( $4.45 \pm 8.44$ ;  $p = 0.3678$ ). By 6 months, patients with good outcomes showed significantly lower disease activity ( $2.27 \pm 4.29$ ) than those with poor outcomes ( $4.24 \pm 8.67$ ;  $p < 0.0001$ ).

**Conclusion:** We concluded that poor renal outcomes were more commonly linked to patients with lupus nephritis who had higher disease activity scores, persistent anti-dsDNA elevation, lower complement C4 levels, impaired renal function (elevated urea and creatinine, reduced eGFR), and significantly higher 24-hour proteinuria.

**Keywords:** Lupus Nephritis, Predictors, Proteinuria, And Immunosuppressive Therapy.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

### Introduction

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease characterized by systemic inflammation affecting multiple organs and tissues [1]. It is a complex disorder with a wide spectrum of clinical manifestations, ranging from mild symptoms such as fatigue and skin rashes to severe organ damage. SLE primarily affects women of childbearing age and has a multifactorial etiology involving genetic, environmental, and hormonal factors [2]. Due to its diverse presentation and unpredictable course, SLE poses significant challenges in diagnosis and management. One of the most severe complications of SLE is lupus nephritis (LN), a form of kidney inflammation

resulting from autoimmune-mediated damage. Lupus nephritis affects nearly 40-60% of SLE patients and is a major contributor to morbidity and mortality [3]. It is classified into different histological types based on the International Society of Nephrology/Renal Pathology Society (ISN/RPS) classification, which helps guide treatment strategies [4]. Without timely intervention, lupus nephritis can progress to end-stage renal disease (ESRD), necessitating dialysis or kidney transplantation [5]. The pathogenesis of lupus nephritis involves a complex interplay of immune dysregulation, autoantibody production (particularly anti-dsDNA antibodies), complement

activation, and inflammatory cytokine release [6]. These immune-mediated processes lead to glomerular and tubulointerstitial injury, manifesting clinically as proteinuria, hematuria, and declining renal function. Renal biopsy is considered the gold standard for diagnosing and assessing the severity of renal involvement. However, it is an invasive procedure and may not always be feasible in patients with severe manifestations such as hematological or central nervous system involvement. Therefore, several studies have been conducted to identify novel biomarkers that can predict the renal involvement. Neutrophil gelatinase-associated lipocalin (NGAL) might predict the renal involvement in SLE before nephritis development, but it cannot predict the severity, staging, or treatment strategy. Understanding the mechanisms underlying lupus nephritis and refining therapeutic strategies is crucial in reducing its burden and enhancing the quality of life for affected individuals. Despite the advances in immunosuppressive therapies, managing lupus nephritis remains challenging due to relapses, drug toxicity and variable patient responses to treatment. This study aims to evaluate the demographic, socioeconomic, clinical, and laboratory profiles of patients with systemic lupus erythematosus (SLE) presenting with lupus nephritis and to identify potential predictors of poor renal response among these patients in a tertiary care teaching institute in North Bengal.

### Materials and Methods

**Type of Study:** Observational, ambispective cohort, non-interventional, hospital based study.

**Place of Study:** Department of Medicine, North Bengal Medical College and Hospital, Darjeeling.

**Study Duration:** 18 months from 1st July 2023 to 31st Dec 2024.

**Sample Size:** 60 patients.

**Inclusion Criteria:** All patients who fulfill the 2019 classification criteria for SLE and had a biopsy proven diagnosis of lupus nephritis classified according to the International Society of Nephrology/ Renal Pathology Society (ISN/RPS) 2003 lupus nephritis classification system

### Exclusion Criteria

- Participants unwilling to give formal or written consent
- Patients with preexisting disease like Diabetes which can cause proteinuria

### Study Variables

- Demographic features (gender, age at the time of diagnosis of SLE and lupus nephritis)
- Socioeconomic status according to Modified Kuppuswamy Scale
- Medication (drugs at the time of starting LN induction treatment, induction regimen (immunosuppressant, glucocorticoid pulses, adjunctive therapy)
- Comorbidities (diabetes, hypertension)
- Disease features (SLE classification criteria, LN histological class, serum immunological profile)
- Disease activity (SLE disease activity index 2000, SLEDAI-2K score)

**Statistical Analysis:** Data were entered into Excel and analyzed using SPSS and GraphPad Prism. Numerical variables were summarized as means  $\pm$  standard deviations, while categorical variables were presented as counts and percentages.

Independent groups were compared using two-sample t-tests, and paired t-tests were applied for correlated (paired) data. Comparisons of categorical variables were performed using chi-square tests, with Fisher's exact test applied when sample sizes were small. A p-value of  $\leq 0.05$  was considered statistically significant.

### Result

**Table 1: Association between Age in Years, Gender and SES Class: Outcome**

|              |        | Good Outcome | Poor Outcome | Total       | p-value |
|--------------|--------|--------------|--------------|-------------|---------|
| Age in Years | < 20   | 18 (35.3%)   | 2 (22.2%)    | 20 (33.3%)  | 0.0696  |
|              | 20–30  | 25 (49.0%)   | 4 (44.4%)    | 29 (48.3%)  |         |
|              | 31–40  | 3 (5.9%)     | 3 (33.3%)    | 6 (10.0%)   |         |
|              | > 40   | 5 (9.8%)     | 0 (0.0%)     | 5 (8.3%)    |         |
|              | Total  | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |
| Gender       | Female | 50 (98.0%)   | 9 (100.0%)   | 59 (98.3%)  | 0.6718  |
|              | Male   | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    |         |
|              | Total  | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |
| SES Class    | II     | 9 (17.6%)    | 2 (22.2%)    | 11 (18.3%)  | 0.7001  |
|              | III    | 28 (54.9%)   | 5 (55.6%)    | 33 (55.0%)  |         |
|              | IV     | 12 (23.5%)   | 1 (11.1%)    | 13 (21.7%)  |         |
|              | V      | 2 (3.9%)     | 1 (11.1%)    | 3 (5.0%)    |         |
|              | Total  | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |

**Table 2: Association between 2nd Immunosuppressant used, comorbidities and LN histological class: Outcome**

|             |             | Good Outcome | Poor Outcome | Total       | p-value |
|-------------|-------------|--------------|--------------|-------------|---------|
| 2nd IS Used | CYC         | 39 (76.5%)   | 7 (77.8%)    | 46 (76.7%)  | 0.7007  |
|             | CYC, MMF    | 1 (2.0%)     | 1 (11.1%)    | 2 (3.3%)    |         |
|             | MMF         | 5 (9.8%)     | 1 (11.1%)    | 6 (10.0%)   |         |
|             | RTX         | 2 (3.9%)     | 0 (0.0%)     | 2 (3.3%)    |         |
|             | RTX and MMF | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    |         |
|             | Nil         | 3 (5.9%)     | 0 (0.0%)     | 3 (5.0%)    |         |
|             | Total       | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |
| Comorbidity | CKD         | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    | 0.2563  |
|             | MASLD       | 2 (3.9%)     | 0 (0.0%)     | 2 (3.3%)    |         |
|             | NIL         | 46 (90.2%)   | 8 (88.9%)    | 54 (90.0%)  |         |
|             | PTB         | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    |         |
|             | RA          | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    |         |
|             | RPGN        | 0 (0.0%)     | 1 (11.1%)    | 1 (1.7%)    |         |
|             | Total       | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |
| LN Class    | II          | 3 (5.9%)     | 0 (0.0%)     | 3 (5.0%)    | 0.1805  |
|             | III + V     | 0 (0.0%)     | 1 (11.1%)    | 1 (1.7%)    |         |
|             | IV          | 35 (68.6%)   | 5 (55.6%)    | 40 (66.7%)  |         |
|             | IV and V    | 2 (3.9%)     | 0 (0.0%)     | 2 (3.3%)    |         |
|             | IV + V      | 1 (2.0%)     | 0 (0.0%)     | 1 (1.7%)    |         |
|             | V           | 10 (19.6%)   | 3 (33.3%)    | 13 (21.7%)  |         |
|             | Total       | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |

**Table 3: Association between ANA patterns: Outcome**

| ANA Pattern   | Good Outcome | Poor Outcome | Total       | p-value |
|---------------|--------------|--------------|-------------|---------|
| Coarse        | 10 (19.6%)   | 2 (22.2%)    | 12 (20.0%)  | 0.8582  |
| Fine Speckled | 19 (37.3%)   | 4 (44.4%)    | 23 (38.3%)  |         |
| Homogeneous   | 22 (43.1%)   | 3 (33.3%)    | 25 (41.7%)  |         |
| Total         | 51 (100.0%)  | 9 (100.0%)   | 60 (100.0%) |         |

**Table 4: Distribution of mean SLEDAI score at 0, 3, 6months: Outcome**

|                            |              | Number | Mean    | SD   | Minimum | Maximum | Median | p-value |
|----------------------------|--------------|--------|---------|------|---------|---------|--------|---------|
| SLEDAI score at (0 months) | Good Outcome | 51     | 10.7451 | 5.58 | 1       | 23      | 10     | 0.6106  |
|                            | Poor Outcome | 9      | 11.7778 | 5.56 | 4       | 21      | 11     |         |
| SLEDAI score at 3 months   | Good Outcome | 49     | 7.2245  | 3.57 | 1       | 14      | 8      | 0.3678  |
|                            | Poor Outcome | 9      | 8.4444  | 4.45 | 4       | 18      | 8      |         |
| SLEDAI score at 6 months   | Good Outcome | 49     | 4.2857  | 2.27 | 1       | 11      | 4      | <0.0001 |
|                            | Poor Outcome | 9      | 8.6667  | 4.24 | 2       | 14      | 10     |         |

**Table 5: Distribution of mean antids DNA at 0, 3, 6months: Outcome**

|                |              | Number | Mean     | SD       | Minimum | Maximum | Median | p-value |
|----------------|--------------|--------|----------|----------|---------|---------|--------|---------|
| Antids DNA (0) | Good Outcome | 51     | 226.1727 | 315.2923 | 2.2     | 1309    | 92     | 0.2655  |
|                | Poor Outcome | 9      | 362.6222 | 442.0843 | 6.2     | 1246    | 287    |         |
| Antids DNA (3) | Good Outcome | 49     | 132.1427 | 163.8042 | 1.48    | 682     | 78     | 0.0493  |
|                | Poor Outcome | 9      | 285.2111 | 384.3782 | 2       | 1056    | 124    |         |
| Antids DNA (6) | Good Outcome | 49     | 99.0741  | 127.6124 | 1.2     | 540     | 59     | 0.0157  |
|                | Poor Outcome | 9      | 265.9556 | 375.5164 | 3       | 998     | 110    |         |

**Table 6: Distribution of mean C3 and C4 AT 0, 3, 6months: Outcome**

|        |              | Number | Mean    | SD   | Minimum | Maximum | Median | p-value |
|--------|--------------|--------|---------|------|---------|---------|--------|---------|
| C3 (0) | Good Outcome | 51     | 44.0898 | 19.5 | 14      | 94      | 42     | 0.0682  |
|        | Poor Outcome | 9      | 60.7    | 45.2 | 14      | 158     | 50     |         |
| C3 (3) | Good Outcome | 49     | 64.1904 | 25.9 | 24      | 165     | 61     | 0.5353  |
|        | Poor Outcome | 9      | 70.7333 | 42.6 | 23      | 147     | 59     |         |
| C3 (6) | Good Outcome | 49     | 84.6551 | 31.8 | 20.4    | 194     | 86.7   | 0.2305  |
|        | Poor Outcome | 9      | 70.1444 | 39.7 | 15      | 127     | 81     |         |
| C4 (0) | Good Outcome | 51     | 9.1512  | 5.97 | 0.34    | 28.6    | 8.05   | 0.0105  |
|        | Poor Outcome | 9      | 16.2511 | 13.3 | 4       | 41      | 11.46  |         |
| C4 (3) | Good Outcome | 49     | 17.3129 | 9.03 | 4.6     | 43      | 14.2   | 0.0374  |
|        | Poor Outcome | 9      | 26.2778 | 21.2 | 5.7     | 78      | 18     |         |
| C4 (6) | Good Outcome | 49     | 26.3347 | 14.2 | 5.2     | 98      | 25     | 0.5847  |
|        | Poor Outcome | 9      | 23.6056 | 9.89 | 6       | 35      | 24.2   |         |

**Table 7: Distribution of mean urea at 0, 3, 6months: Outcome**

|          |              | Number | Mean    | SD      | Minimum | Maximum | Median | p-value |
|----------|--------------|--------|---------|---------|---------|---------|--------|---------|
| Urea (0) | Good Outcome | 51     | 51.3137 | 41.4152 | 10      | 196     | 37     | 0.1837  |
|          | Poor Outcome | 9      | 73.1667 | 62.534  | 30      | 218     | 43     |         |
| Urea (3) | Good Outcome | 49     | 35.1224 | 20.8633 | 12      | 104     | 28     | 0.1112  |
|          | Poor Outcome | 9      | 48.3333 | 30.5655 | 26      | 125     | 46     |         |
| Urea (6) | Good Outcome | 49     | 27.6531 | 15.8687 | 10      | 88      | 24     | 0.5635  |
|          | Poor Outcome | 9      | 31.2222 | 22.281  | 17      | 88      | 24     |         |

**Table 8: Distribution of mean creatinine at 0, 3, 6months: Outcome**

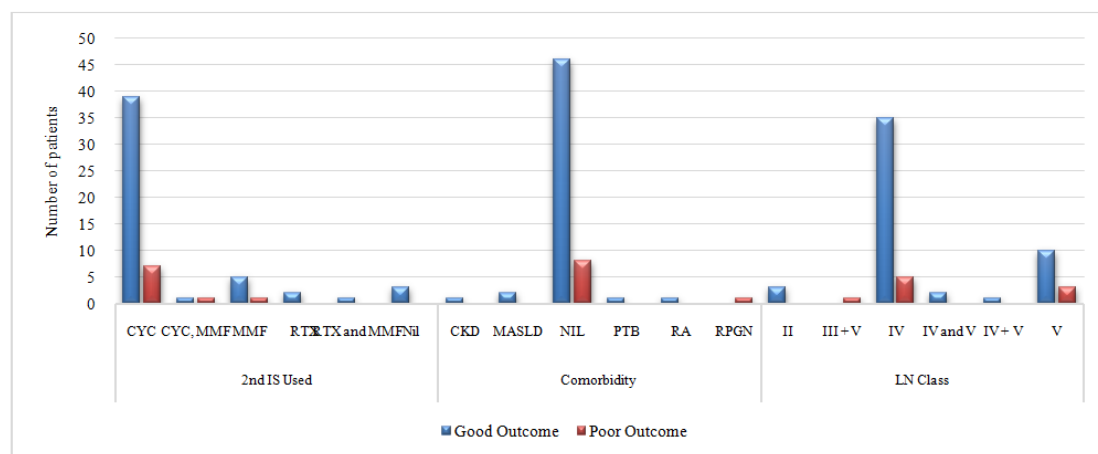
|                |              | Number | Mean   | SD     | Minimum | Maximum | Median | p-value |
|----------------|--------------|--------|--------|--------|---------|---------|--------|---------|
| Creatinine (3) | Good Outcome | 51     | 1.19   | 0.7918 | 0.4     | 3.5     | 0.8    | 0.0722  |
|                | Poor Outcome | 9      | 1.9778 | 2.5193 | 0.5     | 8.5     | 1.3    |         |
| Creatinine (3) | Good Outcome | 49     | 0.8857 | 0.4103 | 0.4     | 1.9     | 0.8    | 0.052   |
|                | Poor Outcome | 9      | 1.4222 | 1.6962 | 0.4     | 5.8     | 0.8    |         |
| Creatinine (3) | Good Outcome | 49     | 0.7755 | 0.2983 | 0.4     | 1.6     | 0.7    | 0.0572  |
|                | Poor Outcome | 9      | 1.1    | 0.976  | 0.4     | 3.6     | 0.8    |         |

**Table 9: Distribution of mean eGFR at 0, 3, 6 months: Outcome**

|           |              | Number | Mean  | SD    | Minimum | Maximum | Median | p-value |
|-----------|--------------|--------|-------|-------|---------|---------|--------|---------|
| E GFR (0) | Good Outcome | 50     | 89.2  | 43.25 | 18      | 148     | 107    | 0.418   |
|           | Poor Outcome | 9      | 76.11 | 50.31 | 6       | 135     | 58     |         |
| E GFR (3) | Good Outcome | 49     | 101   | 34.74 | 38      | 142     | 111    | 0.403   |
|           | Poor Outcome | 9      | 89.67 | 49.38 | 9       | 148     | 105    |         |
| E GFR (6) | Good Outcome | 49     | 108.7 | 30.06 | 45      | 158     | 111    | 0.28    |
|           | Poor Outcome | 9      | 96.11 | 40.58 | 17      | 138     | 110    |         |

**Table 10: Distribution of mean 24 hr proteinuria at 0, 3, 6months: Outcome**

|                       | Number | Mean      | SD        | Minimum | Maximum | Median |
|-----------------------|--------|-----------|-----------|---------|---------|--------|
| 24 hr proteinuria (0) | 60     | 2312.6078 | 3974.6054 | 144     | 26771   | 1395.5 |
| 24 hr proteinuria (3) | 58     | 807.4003  | 908.3352  | 72.7    | 5060    | 555.5  |
| 24 hr proteinuria (6) | 58     | 347.8628  | 526.1569  | 12.5    | 2289    | 133.75 |



**Figure 1: IS Use vs. LN Class**

In our study of 60 patients, age appeared to have a potential influence on outcomes, though the association was not statistically significant ( $p = 0.0696$ ). Among the 51 patients with good outcomes, the majority (25 patients; 49.0%) were aged 20–30 years, while in the poor outcome group (9 patients), the highest proportion (3 patients; 33.3%) were aged 31–40 years. Gender showed no significant association with outcomes ( $p = 0.6718$ ); 59 out of 60 patients were female, and only one male had a good outcome. Socioeconomic status (SES) also did not significantly affect outcomes ( $p = 0.7001$ ). Most patients in both good (28 patients; 54.9%) and poor (5 patients; 55.6%) outcome groups belonged to SES Class III. In our study of 60 patients, the choice of second immunosuppressant (IS) did not show a statistically significant association with outcomes ( $p = 0.7007$ ). Cyclophosphamide (CYC) was the most commonly used agent in both good (39 patients; 76.5%) and poor (7 patients; 77.8%) outcome groups. Comorbidities were also not significantly associated with outcomes ( $p = 0.2563$ ). Most patients had no comorbid conditions, seen in 46 patients (90.2%) with good outcomes and 8 patients (88.9%) with poor outcomes. Similarly, lupus nephritis (LN) class was not significantly linked to outcomes ( $p = 0.1805$ ). Class IV was the most frequent, observed in 35 patients (68.6%) with good outcomes and 5 patients (55.6%) with poor outcomes. In our study of 60 patients, the ANA pattern was not significantly associated with clinical outcomes ( $p = 0.8582$ ). The homogeneous pattern was the most common overall, seen in 22 patients (43.1%) with good outcomes and 3 patients (33.3%) with poor outcomes. The fine speckled pattern was observed in 19 patients (37.3%) with good outcomes and 4 patients (44.4%) with poor outcomes. The coarse pattern was the least common, found in 10 patients (19.6%) with good outcomes and 2 patients (22.2%) with poor outcomes. In our study of 60 patients, those with good outcomes had similar baseline SLEDAI

scores ( $10.75 \pm 5.58$ ) compared to patients with poor outcomes ( $11.78 \pm 5.56$ ;  $p = 0.6106$ ). At 3 months, disease activity remained comparable between the good ( $3.57 \pm 7.22$ ) and poor outcome groups ( $4.45 \pm 8.44$ ;  $p = 0.3678$ ). By 6 months, patients with good outcomes showed significantly lower disease activity ( $2.27 \pm 4.29$ ) than those with poor outcomes ( $4.24 \pm 8.67$ ;  $p < 0.0001$ ). In our study, baseline Anti-dsDNA levels were higher in the poor outcome group ( $362.62 \pm 442.08$ ) compared to the good outcome group ( $226.17 \pm 315.29$ ), but this difference was not statistically significant ( $p = 0.2655$ ). At 3 months, Anti-dsDNA levels decreased in both groups but remained significantly higher in patients with poor outcomes ( $285.21 \pm 384.38$ ) than in those with good outcomes ( $132.14 \pm 163.80$ ;  $p = 0.0493$ ). By 6 months, the difference was more pronounced, with poor outcome patients showing significantly elevated levels ( $265.96 \pm 375.52$ ) compared to the good outcome group ( $99.07 \pm 127.61$ ;  $p = 0.0157$ ). In our study, baseline C3 levels were higher in the poor outcome group ( $60.7 \pm 45.23$ ) compared to the good outcome group ( $44.09 \pm 19.54$ ), though this was not statistically significant ( $p = 0.0682$ ). At 3 and 6 months, C3 levels increased in both groups with no significant differences ( $p = 0.5353$  and  $0.2305$ , respectively). Conversely, baseline C4 levels were significantly lower in the good outcome group ( $9.15 \pm 5.97$ ) compared to the poor outcome group ( $16.25 \pm 13.33$ ;  $p = 0.0105$ ). This significant difference persisted at 3 months (Good:  $17.31 \pm 9.03$ ; Poor:  $26.28 \pm 21.23$ ;  $p = 0.0374$ ) but was not significant by 6 months ( $p = 0.5847$ ). In our study, baseline urea levels were higher in the poor outcome group ( $73.17 \pm 62.53$ ) compared to the good outcome group ( $51.31 \pm 41.42$ ), but this difference was not statistically significant ( $p = 0.1837$ ). At 3 months, urea levels decreased in both groups, remaining higher in the poor outcome group ( $48.33 \pm 30.57$ ) than in the good outcome group ( $35.12 \pm 20.86$ ;  $p = 0.1112$ ). By 6 months, urea levels further declined and were similar

between groups (Good:  $27.65 \pm 15.87$ ; Poor:  $31.22 \pm 22.28$ ;  $p = 0.5635$ ). In our study, baseline creatinine levels tended to be higher in the poor outcome group ( $1.98 \pm 2.52$ ) compared to the good outcome group ( $1.19 \pm 0.79$ ), although this was not statistically significant ( $p = 0.0722$ ). At 3 months, creatinine levels decreased in both groups but remained higher in the poor outcome group ( $1.42 \pm 1.70$ ) versus the good outcome group ( $0.89 \pm 0.41$ ;  $p = 0.052$ ). By 6 months, the difference narrowed with creatinine levels of  $0.78 \pm 0.30$  in the good outcome group and  $1.10 \pm 0.98$  in the poor outcome group ( $p = 0.0572$ ), no significant difference. In our study, estimated glomerular filtration rate (eGFR) at baseline was slightly higher in the good outcome group ( $89.2 \pm 43.25$ ) compared to the poor outcome group ( $76.11 \pm 50.31$ ), but the difference was not significant ( $p = 0.418$ ). At 3 and 6 months, eGFR increased in both groups, remaining higher in the good outcome group ( $101.02 \pm 34.74$  and  $108.67 \pm 30.06$ , respectively) than in the poor outcome group ( $89.67 \pm 49.38$  and  $96.11 \pm 40.58$ ), though differences were not statistically significant ( $p = 0.4034$  and  $0.2804$ ). In our study, baseline 24-hour proteinuria levels were significantly higher in the poor outcome group ( $5969.81 \pm 8026.37$  mg) compared to the good outcome group ( $1667.22 \pm 2345.42$  mg;  $p = 0.0021$ ). At 3 months, proteinuria decreased in both groups but remained significantly elevated in the poor outcome group ( $1916.15 \pm 985.06$  mg) versus the good outcome group ( $603.75 \pm 738.39$  mg;  $p < 0.0001$ ). By 6 months, the difference persisted, with the poor outcome group showing much higher proteinuria ( $1356.22 \pm 584.06$  mg) compared to the good outcome group ( $162.65 \pm 215.01$  mg;  $p < 0.0001$ ).

## Discussion

We found that a higher proportion of patients with good outcomes (25 out of 51; 49.0%) were in the 20–30 year age group, whereas in the poor outcome group, the highest proportion (3 out of 9; 33.3%) belonged to the 31–40 year group, indicating a trend toward poorer response with increasing age ( $p = 0.0696$ ).

The gender distribution was highly skewed toward females, with 59 out of 60 patients (98.3%) being female; the only male patient (1.7%) achieved a good outcome ( $p = 0.6718$ ). Most patients in both groups belonged to socioeconomic class III (28 out of 51 [54.9%] in the good outcome group and 5 out of 9 [55.6%] in the poor outcome group), showing no major variation in socioeconomic background between the two outcome groups ( $p = 0.7001$ ). In similar study Kaur R et al [7] (2023), in their large retrospective cohort, found that the majority of patients treated successfully with intralesional MMR were in the 21–40-year range, with no significant gender difference in clearance rates.

We observed that CYC was the most frequently used second-line immunosuppressant in both groups—39 out of 51 (76.5%) patients with good outcomes and 7 out of 9 (77.8%) with poor outcomes ( $p = 0.7007$ ). Most patients (54 out of 60; 90.0%) had no comorbidities, while isolated cases of CKD, MASLD, PTB, and RA were seen only among good outcomes, and one patient with poor outcome had RPGN ( $p = 0.2563$ ). Class IV lupus nephritis was the most common histological type, seen in 35 out of 51 (68.6%) good outcome cases and 5 out of 9 (55.6%) poor outcome cases, followed by Class V (19.6% vs 33.3%), with no significant association between LN class and treatment outcome ( $p = 0.1805$ ). In other study by Graili P et al.[8] (2021) demonstrated that CYC-based regimens yielded remission rates above 70%, irrespective of sex or baseline renal function, consistent with our observations of comparable outcomes across CYC-treated groups

We showed that the most common ANA pattern overall was homogeneous, observed in 22 out of 51 (43.1%) patients with good outcomes and 3 out of 9 (33.3%) with poor outcomes. The fine speckled pattern was the next most frequent, seen in 19 out of 51 (37.3%) good outcome cases and 4 out of 9 (44.4%) poor outcome cases, followed by the coarse pattern in 10 out of 51 (19.6%) and 2 out of 9 (22.2%) cases, respectively. The distribution of ANA patterns showed no statistically significant association with treatment outcome ( $p = 0.8582$ ). In similar study by Rathi et al. [9] (2018) noted that the homogeneous ANA pattern was the most frequent among Indian LN patients (41%), followed by speckled patterns (35%), and found no significant correlation between ANA pattern and renal outcome.

We found that the mean baseline SLEDAI score was comparable between the good and poor outcome groups ( $10.75 \pm 5.58$  vs.  $11.78 \pm 5.56$ ;  $p = 0.6106$ ). At 3 months, the mean SLEDAI score showed a greater reduction in the good outcome group ( $7.22 \pm 3.57$ ) compared to the poor outcome group ( $8.44 \pm 4.45$ ), though the difference was not statistically significant ( $p = 0.3678$ ). However, by 6 months, patients with good outcomes demonstrated a markedly lower mean SLEDAI score ( $4.29 \pm 2.27$ ) compared to those with poor outcomes ( $8.67 \pm 4.24$ ), and this difference was statistically significant ( $p < 0.0001$ ). In similar study by Tawfik Yet al [10] (2015) observed that patients achieving clinical remission exhibited a significantly greater decline in SLEDAI scores over 6 months of follow-up.

We observed that the mean anti-dsDNA level at baseline was higher in the poor outcome group ( $362.62 \pm 442.08$  IU/mL) compared to the good outcome group ( $226.17 \pm 315.29$  IU/mL), though the difference was not statistically significant ( $p =$

0.2655). At 3 months, anti-dsDNA levels showed a significant decline in the good outcome group ( $132.14 \pm 163.80$  IU/mL) compared to the poor outcome group ( $285.21 \pm 384.38$  IU/mL) ( $p = 0.0493$ ). By 6 months, this difference became more pronounced, with mean levels of  $99.07 \pm 127.61$  IU/mL in the good outcome group versus  $265.96 \pm 375.52$  IU/mL in the poor outcome group ( $p = 0.0157$ ).

We found that mean baseline C3 levels were slightly lower in the good outcome group ( $44.09 \pm 19.54$  mg/dL) compared to the poor outcome group ( $60.70 \pm 45.23$  mg/dL), though the difference was not statistically significant ( $p = 0.0682$ ). At 3 months, C3 levels improved in both groups ( $64.19 \pm 25.94$  vs.  $70.73 \pm 42.65$  mg/dL;  $p = 0.5353$ ), and by 6 months, mean C3 values were higher in the good outcome group ( $84.66 \pm 31.76$  mg/dL) than in the poor outcome group ( $70.14 \pm 39.65$  mg/dL), again without statistical significance ( $p = 0.2305$ ). For C4, baseline levels were significantly lower in the good outcome group ( $9.15 \pm 5.97$  mg/dL) than in the poor outcome group ( $16.25 \pm 13.33$  mg/dL;  $p = 0.0105$ ). At 3 months, C4 levels improved in both groups, remaining higher in those with poor outcomes ( $17.31 \pm 9.03$  vs.  $26.28 \pm 21.23$  mg/dL;  $p = 0.0374$ ). By 6 months, C4 levels were comparable between the groups ( $26.33 \pm 14.22$  vs.  $23.61 \pm 9.89$  mg/dL;  $p = 0.5847$ ). In other study by Sciascia et al. [11] (2021) observed that hypocomplementemia at baseline often reflects immune complex deposition and complement consumption, with subsequent recovery paralleling treatment

We found that the mean baseline serum urea level was higher in the poor outcome group ( $73.17 \pm 62.53$  mg/dL) compared to the good outcome group ( $51.31 \pm 41.42$  mg/dL), though this difference was not statistically significant ( $p = 0.1837$ ). At 3 months, urea levels showed a declining trend in both groups, with mean values of  $35.12 \pm 20.86$  mg/dL in the good outcome group and  $48.33 \pm 30.57$  mg/dL in the poor outcome group ( $p = 0.1112$ ). By 6 months, further improvement was noted, with mean urea levels of  $27.65 \pm 15.87$  mg/dL in patients with good outcomes and  $31.22 \pm 22.28$  mg/dL in those with poor outcomes ( $p = 0.5635$ ). We showed that the mean baseline serum creatinine level was higher in the poor outcome group ( $1.98 \pm 2.52$  mg/dL) compared to the good outcome group ( $1.19 \pm 0.79$  mg/dL), though the difference did not reach statistical significance ( $p = 0.0722$ ). At 3 months, creatinine levels improved in both groups, with lower mean values in the good outcome group ( $0.89 \pm 0.41$  mg/dL) compared to the poor outcome group ( $1.42 \pm 1.70$  mg/dL), again showing a near-significant difference ( $p = 0.052$ ). By 6 months, further improvement was observed, with mean creatinine levels of  $0.78 \pm 0.30$  mg/dL in

the good outcome group and  $1.10 \pm 0.98$  mg/dL in the poor outcome group ( $p = 0.0572$ ).

We found that the mean baseline estimated glomerular filtration rate (eGFR) was higher in the good outcome group ( $89.2 \pm 43.25$  mL/min/1.73 m<sup>2</sup>) compared to the poor outcome group ( $76.11 \pm 50.31$  mL/min/1.73 m<sup>2</sup>), though the difference was not statistically significant ( $p = 0.418$ ). At 3 months, eGFR improved in both groups, with mean values of  $101.02 \pm 34.74$  mL/min/1.73 m<sup>2</sup> in the good outcome group and  $89.67 \pm 49.38$  mL/min/1.73 m<sup>2</sup> in the poor outcome group ( $p = 0.4034$ ). By 6 months, further improvement was noted, with mean eGFR of  $108.67 \pm 30.06$  mL/min/1.73 m<sup>2</sup> in the good outcome group and  $96.11 \pm 40.58$  mL/min/1.73 m<sup>2</sup> in the poor outcome group ( $p = 0.2804$ ). In similar study by Tektonidou et al. [12] (2020) found that also observed that baseline eGFR strongly predicts long-term renal outcome, with lower eGFR at presentation correlating with chronic renal damage and poorer therapeutic

We showed that at baseline (0 month), the mean 24-hour proteinuria was significantly lower in the good outcome group ( $1667.22 \pm 2345.42$  mg/24 hr) compared to the poor outcome group ( $5969.81 \pm 8026.37$  mg/24 hr), with a statistically significant difference ( $p = 0.0021$ ). At 3 months, proteinuria markedly declined in the good outcome group ( $603.75 \pm 738.39$  mg/24 hr) but remained substantially higher in the poor outcome group ( $1916.15 \pm 985.06$  mg/24 hr), showing a highly significant difference ( $p < 0.0001$ ). Similarly, at 6 months, mean proteinuria further reduced to  $162.65 \pm 215.01$  mg/24 hr in the good outcome group, while persisting at elevated levels in the poor outcome group ( $1356.22 \pm 584.06$  mg/24 hr), again demonstrating a highly significant difference ( $p < 0.0001$ ).

## Conclusion

We concluded that poor renal outcomes were more commonly linked to patients with lupus nephritis who had higher disease activity scores, persistent anti-dsDNA elevation, lower complement C4 levels, impaired renal function (elevated urea and creatinine, reduced eGFR), and significantly higher 24-hour proteinuria. Proteinuria was the most reliable and statistically significant predictive factor across all time points, while ANA pattern, lupus class, age, gender, socioeconomic level, and comorbidities did not exhibit any meaningful correlations. These results highlight the significance of closely monitoring certain serological and renal markers, as well as proteinuria, during follow-up. By employing these variables to identify high-risk patients early on, treatment intensity may be guided and long-term renal outcomes in lupus nephritis may be improved.

## Reference

1. Grammatikos AP, Tsokos GC. Immunodeficiency and autoimmunity: lessons from systemic lupus erythematosus. *Trends in molecular medicine*. 2012 Feb 1;18(2):101-8.
2. Choi J, Kim ST, Craft J. The pathogenesis of systemic lupus erythematosus—an update. *Current opinion in immunology*. 2012 Dec 1; 24(6):651-7.
3. Yu F, Haas M, Glasscock R, Zhao MH. Redefining lupus nephritis: clinical implications of pathophysiologic subtypes. *Nature Reviews Nephrology*. 2017 Aug; 13(8): 483-95.
4. Weening JJ, D'agati VD, Schwartz MM, Seshan SV, Alpers CE, Appel GB, Balow JE, Bruijn JA, Cook T, Ferrario F, Fogo AB. The classification of glomerulonephritis in systemic lupus erythematosus revisited. *Kidney international*. 2004 Feb 1;65(2):521-30.
5. Almaani S, Meara A, Rovin BH. Update on lupus nephritis. *Clinical Journal of the American Society of Nephrology*. 2017 May 1; 12(5): 825-35.
6. Davidson A. What is damaging the kidney in lupus nephritis?. *Nature Reviews Rheumatology*. 2016 Mar;12(3):143-53.
7. Kaur R, Sood S, Agrawal I, Sharma B. Retrospective Analysis of Treatment of Cutaneous Warts with Measles, Mumps, and Rubella Immunotherapy Over 8 Years. *Turkish Journal of Dermatology*. 2023 Apr 1;17(2):64-8.
8. Graili P, Ieraci L, Hosseinkhah N, Argent-Katwala M. Artificial intelligence in outcomes research: a systematic scoping review. *Expert Review of Pharmacoeconomics & Outcomes Research*. 2021 Jul 4;21(4):601-23.
9. Rathi M, Goyal A, Jaryal A, Sharma A, Gupta PK, Dhir V, et al. Clinicopathological spectrum of lupus nephritis: An experience from a tertiary care center in northern India. *Indian J Nephrol*. 2018;28(1):28-34.
10. Tawfik Y, Shaat RM, El-Bassiony SR, Hawas S, Effat N. Urinary and serum neutrophil gelatinase-associated lipocalin as a biomarker in Egyptian systemic lupus erythematosus patients: Relation to lupus nephritis and disease activity. *The Egyptian Rheumatologist*. 2015 Oct 1;37(4):S25-31.
11. Sciascia S, Radin M, Yazdany J, et al. Complement as a biomarker for systemic lupus erythematosus. *Expert Rev Clin Immunol*. 2021; 17(6):563–575.
12. Tektonidou MG, Dasgupta A, Ward MM. Risk of end-stage renal disease in patients with lupus nephritis, 1971–2015: A systematic review and Bayesian meta-analysis. *Arthritis Rheumatol*. 2016;68(6):1432–1441.