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Urine KIM-1 as Predictor of Renal Histopathology and Treatment Response in Lupus Nephritis Patients

Mamun Chowdhury Raju[1], Rana Mokarram Hossain[2], A.H. Hamid Ahmed[3], Muhammad Nazrul Islam[4], Md. Omar Faroque[5], Md Bedar Uddin[6], S.M. Remin Rafi[7], Anjuman Ara Daisy[8], Nur Jahan[9], Noureen Amin[10]

Abstract

Introduction: Lupus nephritis is one of the most severe complications of systemic lupus erythematosus (SLE) and typically develops within five years of an SLE diagnosis in 50%-60% of patients. Lupus nephritis is a major predictor of prognosis and a significant contributor to morbidity and mortality in SLE. Renal biopsy is the gold standard for diagnosing lupus nephritis patients. However, its invasive nature and associated risks limit its use, highlighting the need for non-invasive biomarker like Kidney Injury Molecule-1 (KIM-1). Present study aimed to assess urine KIM-1 as predictors of renal histopathology and treatment response in lupus nephritis patients.

Materials & Methods: This prospective observational study was conducted in the Nephrology department at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from March 2023 to August 2024, involving adult patients (age ≥ 18 years) diagnosed with SLE and lupus nephritis. Using convenience sampling, 52 respondents were enrolled. Ethical approval was obtained from the Institutional Review Board (IRB) of BSMMU. Renal biopsies and urine samples were collected for analysis, and patients were followed up at six months.

Results: The study population had a mean age of 28.1 years. The female predominance was 92.3%. The mean BMI was 24.2 kg/m². Hypertension was present in 46.2% of respondents, anemia in 30.8%, edema in 59.6%, and proteinuria in 48.1% (++), and 34.6% (+++). Histologically, 57.7% were classified as Class IV lupus nephritis, and 17.3% as Class III. Hydroxychloroquine was administered to 100% of respondents, cyclophosphamide with corticosteroids to 61.5%, MMF with corticosteroids to 23.1%, and corticosteroids alone to 15.4%. Complete response was achieved by 67.3%, partial response by 28.8%, and no response by 3.8%. At baseline, Class IV lupus nephritis patients had the highest mean urine KIM-1 levels (672.7 ± 504.0 ng/mL), which decreased after six months but remained highest (231.6 ± 191.4 ng/mL). The proliferative group had significantly higher mean urine KIM-1 levels than the non-proliferative group at both baseline and six months. Mean Urine KIM-1 level was significantly ($p < 0.05$) lower in respondents with a complete response compared to those with partial or no response, both at baseline and after six months. Statistically significant differences in mean serum creatinine, serum albumin, and urinary PCR levels were observed between response groups.

Conclusion: Urine KIM-1 levels significantly correlate with disease severity and renal function markers, highlighting its clinical utility. Future research should validate urine KIM-1 as a biomarker and explore its potential in guiding treatment decisions.

Affiliation:

[1]Medical Officer, OSD, DGHS, Dhaka, Bangladesh

[2]Professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

[3]Professor& Chairman, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

[4]Professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

[5]Associate professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

[6]Medical Officer, 250 Bedded General Hospital, Jashore, Bangladesh.

[7]Medical Officer, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh.

[8]Medical Officer, OSD, DGHS, Dhaka, Bangladesh

[9]Medical Officer, Department of Nephrology, National Institute of kidney diseases and Urology, Dhaka, Bangladesh

[10]Research Assistant, Department of Nephrology, National Institute of kidney diseases and Urology, Dhaka, Bangladesh

***Corresponding author:**
Dr. Mamun Chowdhury Raju, Medical Officer, OSD, DGHS, Dhaka, Bangladesh.

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Keywords: Urine KIM-1, Systemic Lupus Erythematosus, Lupus Nephritis.

Introduction

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disorder that impacts various organs, with lupus nephritis being one of its most severe complications [1]. lupus nephritis typically manifests within five years of an SLE diagnosis in 50%-60% of patients [2-4] Even with intensive immunosuppressive treatment, lupus nephritis remains the primary indicator of prognosis and a significant contributor to morbidity and mortality in SLE [5]. Severe lupus nephritis can lead to end-stage kidney disease in 10%-26% of cases [6]. Early detection and management are essential for improving prognosis, but these remain difficult to achieve at present [7-9]. Renal biopsy remains the gold standard method for diagnosing, guiding treatment, and predicting prognosis in lupus nephritis patients. However, the invasive nature of renal biopsies and the risks involved have limited its use [10,11]. Clinical findings and biochemical profile are not enough to accurately predict the histopathologic involvement of lupus nephritis. Conventional biomarkers, such as proteinuria, anti-dsDNA antibody, and complement levels, lack sensitivity and specificity in predicting renal activity in lupus nephritis patients [12]. Therefore, there is an urgent need for non-invasive biomarkers to reflect renal activity, predict renal prognosis, and ultimately guide the treatment of lupus nephritis.

Kidney Injury Molecule-1 (KIM-1) is a transmembrane protein, which features Ig-like and mucin domains in its ectodomain. Prior studies have shown KIM-1 to play a role in tubulointerstitial injury [13]. The expression of tubular KIM-1 is specific to ongoing tubular cell injury and its urinary concentrations may reflect this expression [13-15] Elevated levels of KIM-1 have been observed in patients with acute ischemia, toxic kidney injury, multiple cystic kidney disease, and renal cell carcinoma, as well as in those with chronic kidney disease, including lupus nephritis [16-18]. Research indicates that urine KIM-1 levels are significantly elevated in SLE patients with active lupus nephritis compared to those with inactive lupus nephritis [19,20]. Additionally, urine KIM-1 levels correlate with disease activity and the histological severity of lupus nephritis. However, there is limited data on the impact of urine KIM-1 levels on therapy response, its correlation with renal pathology, or its effectiveness as a predictor of renal function. Present study aimed to assess urine KIM-1 as predictors of renal histopathology and treatment response in lupus nephritis patients.

Materials & Methods

This prospective observational study was conducted in the department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka from March 2023 to August 2024, among adult (age ≥ 18 years) patients diagnosed with SLE with lupus nephritis. Patients with malignancy, active infection, primary glomerulonephritis, other causes of glomerulopathies (infections such as HIV, hepatitis B or C virus, malignancy, drugs), autoimmune diseases other than SLE, inadequate kidney biopsy specimen (<8 scorable glomeruli), pregnant and lactating woman or unwilling to give written consent for the study, were excluded. Using convenience sampling technique, 52 respondents were enrolled in the study as per selection criteria. Before starting the study, formal ethical approval was taken from the Institutional review board (IRB) of BSMMU. At first the natural history, pathophysiology, relevant investigations, current treatment options and outcome of lupus nephritis were explained. Then renal biopsy was done after taking informed written consent. A fresh urine sample from the patient was collected and sent for urinary total protein analysis, while another urine sample was preserved at -80°C for the measurement of urine KIM-1. Urine KIM-1 was measured using a commercially available KIM-1 kit. Additional hematological and biochemical tests on blood and urine were conducted before performing the renal biopsy. Treatment was given according to KDIGO guidelines. All the patients were followed-up at 6th month during the study period. Regular contact with the study population was maintained over the cell phone to minimize drop-out of study subjects as well as early detection & management of adverse drug responses. Data was collected in a pre-tested questionnaire by taking history, examining the subjects clinically, reviewing laboratory findings and outcomes.

Statistical analysis was performed using Windows® based software program Statistical Packages for Social Sciences 25 (SPSS-25) (Chicago, IL, USA). After collection, all the data were checked and cleaned. Quantitative data were expressed as percentage, mean and standard deviation and qualitative data were expressed as frequency distribution and percentage. To determine statistical significance, Chi-squared test, Unpaired t-test, Mann–Whitney U test, Kruskal Wallis test and ANOVA test were considered according to applicability. P value of < 0.05 was considered statistically significant.

Results

Mean age for the study population was 28.1 ± 6.6 years (Table 1). Study population was female predominant at 92.3%. Mean BMI was 24.2 ± 2.6 kg/m2. During admission, hypertension was present among 46.2% respondents, anemia was present among 30.8% respondents, oedema (+) was present among 59.6% respondents and proteinuria (++) was present among 48.1% respondents, followed by 34.6% respondents having proteinuria (+++). For histological classification of lupus nephritis, 57.7% respondents were Class IV, followed by 17.3% respondents being Class III.

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Table 1: Descriptive statistics of the study population. (n = 52)

Characteristics			Data
Age (in years)			28.1 ± 6.6
Gender	Male		4 (7.7%)
	Female		48 (92.3%)
BMI (kg/m²)			24.2 ± 2.6
Clinical presentation at admission	Hypertension		24 (46.2%)
	Anemia		16 (30.8%)
	Oedema	+	31 (59.6%)
		++	5 (9.6%)
	Proteinuria	+	7 (13.5%)
		++	25 (48.1%)
		+++	18 (34.6%)
		++++	2 (3.8%)
Histological classification of lupus nephritis	Class I		0
	Class II		8 (15.4%)
	Class III		9 (17.3%)
	Class IV		30 (57.7%)
	Class V		5 (9.6%)
	Class VI		0
Biochemical profile	RBC (/HPF)	0-5	25 (48.1%)
		>5	27 (51.9%)
	WBC (/HPF)	<5	39 (75.0%)
		>5	13 (25.0%)
	Urinary PCR (g/g)	<3.5	39 (75.0%)
		>3.5	13 (25.0%)
	Serum Albumin (g/g)	<35	41 (78.8%)
		>35	11 (21.2%)
	Serum Creatinine (mg/dl)	>1.2	19 (36.5%)
		≤1.2	33 (63.5%)
	eGFR (%)	15-30	2 (3.8%)
		31-45	9 (17.3%)
		46-60	10 (19.2%)
		61-90	11 (21.2%)
		>90	20 (38.5%)

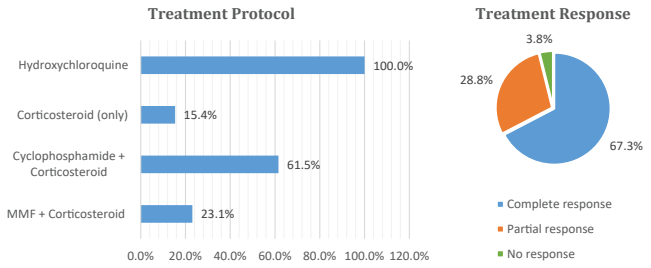

Figure 1: Distribution of study population according to treatment. (n = 52)

In biochemical profile analysis, 51.9% respondents had RBC > 5/HPF, 75.0% had WBC < 5/HPF, 75.0% had urinary PCR <3.5 g/g, 78.8% had serum albumin <35 g/g, 63.5% had serum creatinine ≤1.2 mg/dl and 38.5% had eGFR >90%.

Hydroxychloroquine was administered in 100% of the respondents, followed by Cyclophosphamide + Corticosteroid in 61.5% respondents (Figure 1). MMF + Corticosteroid was administered in 23.1% respondents and Corticosteroid (only) was administered in 15.4% respondents. Complete response was achieved by 67.3% respondents, followed by partial response for 28.8% and no response by 3.8% respondents.

Urine KIM-1 levels were assessed at base line and at 6 months (Figure 2). At baseline class IV showed highest level of mean urine KIM-1 levels at 672.7 ± 504.0 ng/mL, followed by class III with 307.7 ± 179.4 ng/mL mean urine KIM-1 level. At 6 months, class IV showed highest level of mean urine KIM-1 levels at 231.6 ± 191.4 ng/mL, followed by class III with 106.9 ± 34.7 ng/mL mean urine KIM-1 level. The difference in urine KIM-1 levels among histological groups of lupus nephritis both at baseline and at 6 months were statistically significant ($p < 0.05$), indicating a strong association between urine KIM-1 levels and lupus nephritis severity. Urine KIM-1 levels were compared between proliferative and non-proliferative groups of lupus nephritis patients, both at baseline and at 6 months (Figure 3). Mean urine KIM-1 level was significantly ($p < 0.05$) higher for proliferative group compared to non-proliferative group (588.5 ± 474.3 ng/mL and 191.77 ± 64.22 ng/mL, respectively) at baseline. At 6 months, mean urine KIM-1 level was also significantly ($p < 0.05$) higher for proliferative group compared to non-proliferative group (202.8 ± 176.2 ng/mL and 88.1 ± 14.77 ng/mL, respectively).

Urine KIM-1 levels were compared among patients with varying degrees of tubulointerstitial inflammation (Figure 4). Patients with moderate inflammation had highest mean urine KIM-1 level at 656.36 ± 543.54 ng/mL, followed by patients with mild inflammation having mean urine KIM-1 level to be 318.43 ± 201.36 ng/mL and patients with minimal inflammation having mean urine KIM-1 level to be 239.00 ± 85.81 ng/mL. There was statistically significant ($p < 0.05$) difference among the groups in term of mean urine KIM-1 level.

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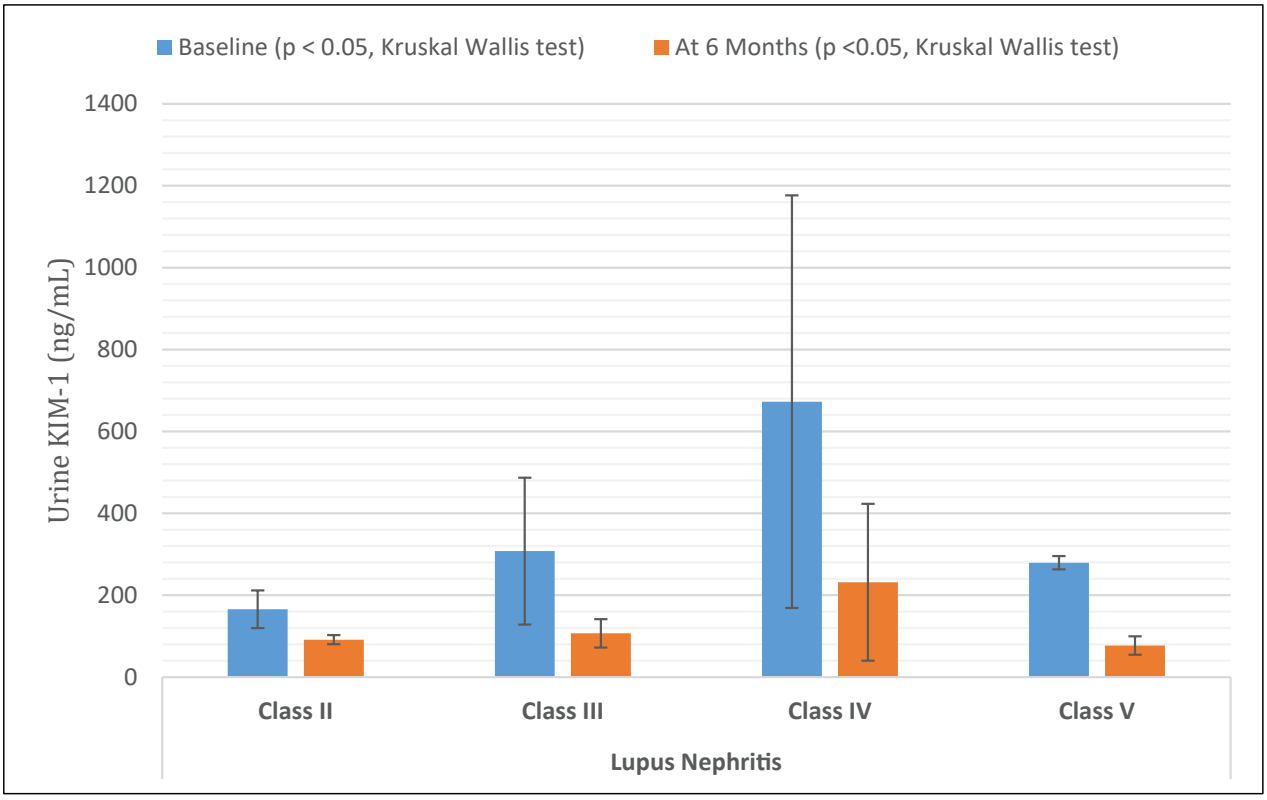

Figure 2: Urine KIM-1 levels at different stages of lupus nephritis.

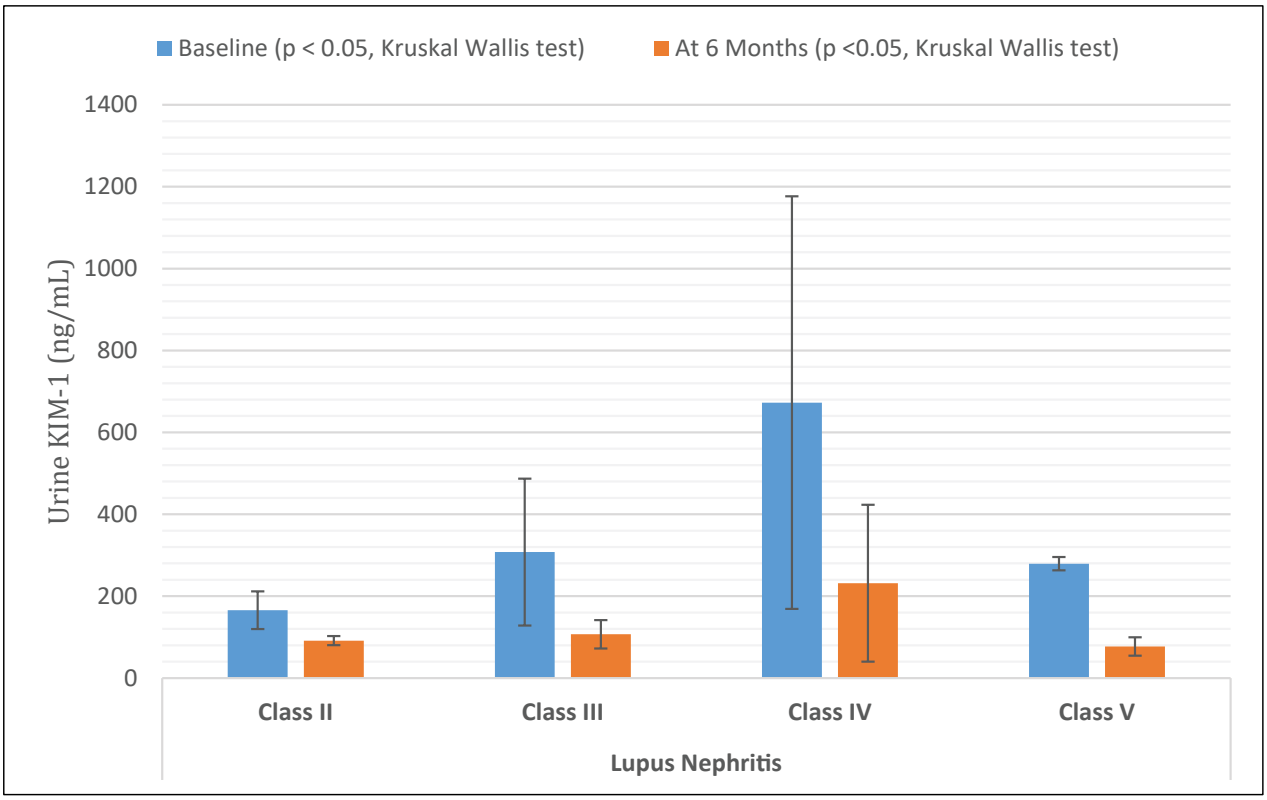

Figure 3: Comparison of urine KIM-1 levels between proliferative and non-proliferative groups.

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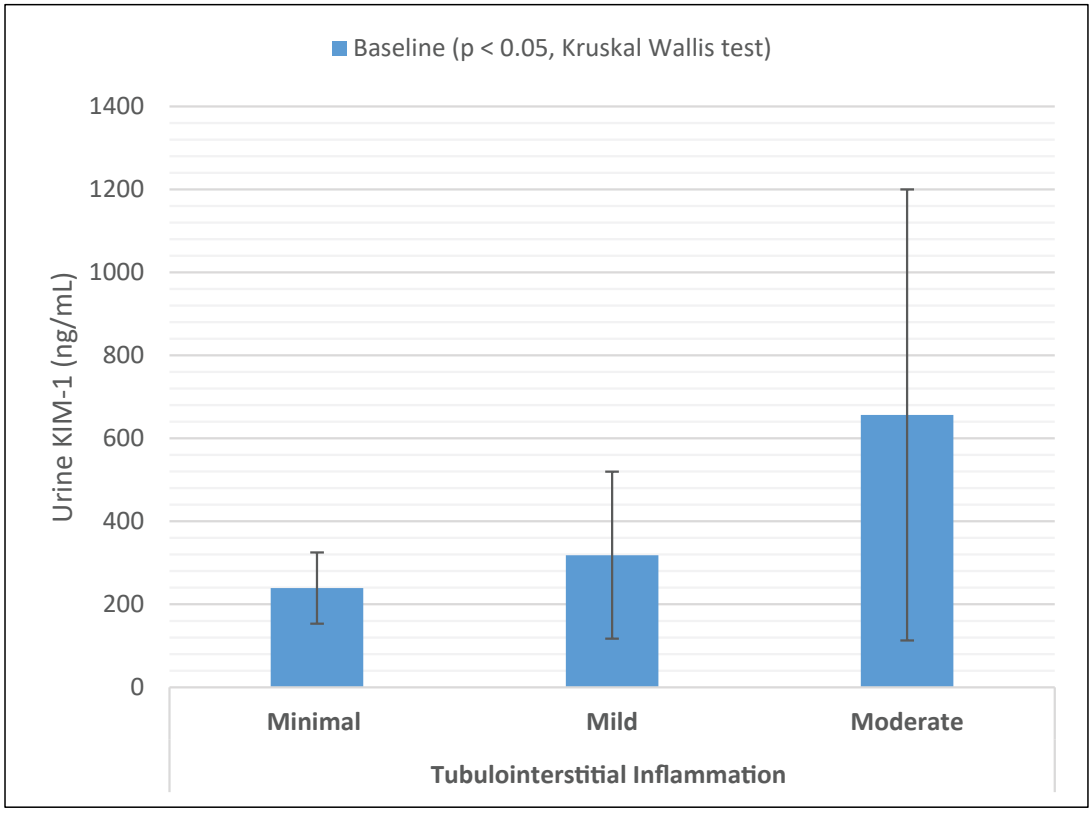

Figure 4: Urine KIM-1 levels at different severity groups for tubulointerstitial inflammation.

Biochemical parameters of respondents were compared based on treatment response at 6 months and retrospectively at baseline (Table II). Among respondents with complete response, mean urine KIM-1 level was lower (338.6 ± 261.7 ng/mL), compared to both partial response (731.4 ± 582.9 ng/mL) and non-response (1311.0 ± 147.1 ng/mL) group at baseline and this difference was statistically significant ($p < 0.05$). At 6 months, mean urine KIM-1 level was lower (107.2 ± 30.4 ng/mL), compared to both partial response (263.8 ± 172.3 ng/mL) and non-response (673.0 ± 297.0 ng/mL) group and this difference was statistically significant ($p < 0.05$). There were also statistically significant ($p < 0.05$) difference between response groups for mean serum creatinine and serum albumin level. Mean urinary PCR was significantly ($p < 0.05$) different at 6 months, but not at baseline ($p = 0.428$). Serum Hb% showed no statistically significant difference among response groups at baseline and at 6 months.

Table 2: Relation of biochemical profile with treatment response. (n = 52)

Characteristics		Treatment Response			P value
		Complete response (n=35)	Partial response (n=15)	Non-response (n=2)	
Baseline	Urine KIM-1 (ng/mL)	338.6 ± 261.7	731.4 ± 582.9	1311.0 ± 147.1	< 0.05
	Urinary PCR (mg/mmol)	3.12 ± 2.88	3.06 ± 1.38	5.10 ± 1.41	0.428
	Serum Hb (%)	13.35 ± 13.85	10.56 ± 1.56	9.40 ± 0.42	0.621
	S. Creatinine (mg/dL)	1.08 ± 0.49	1.14 ± 0.58	2.60 ± 0.28	< 0.05
	Serum Albumin (g/dL)	32.46 ± 5.61	26.47 ± 4.10	27.00 ± 4.24	< 0.05
At 6 Months	Urine KIM-1 (ng/mL)	107.2 ± 30.4	263.8 ± 172.3	673.0 ± 297.0	< 0.05
	Urinary PCR (mg/mmol)	0.25 ± 0.30	0.90 ± 0.74	3.75 ± 0.21	< 0.05
	Serum Hb (%)	11.39 ± 0.83	11.37 ± 1.48	9.60 ± 0.71	0.106
	S. Creatinine (mg/dL)	0.78 ± 0.28	1.12 ± 0.42	2.35 ± 0.35	< 0.05
	Serum Albumin (g/dL)	41.51 ± 1.65	38.53 ± 4.16	30.00 ± 1.41	< 0.05

Data presented as mean ± SD. ANOVA was done. P value < 0.05 was considered statistically significant.

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Mean urine KIM-1 level at baseline and at 6 months of treatment was assessed (Table III). Statistically significant ($p < 0.05$) decrease in mean urine KIM-1 level was observed, 489.3 ± 445.7 ng/mL and 174.1 ± 160.3 ng/mL respectively.

Table 3: Comparison of urine KIM-1 level at baseline and at 6 months of treatment in lupus nephritis. (n = 52)

Characteristics		Baseline	At 6 months	P value
Urine KIM-1	Mean±SD	489.3 ± 445.7	174.1 ± 160.3	< 0.05
(ng/mL)	Median	324.5	108	
	Range (min-max)	114 - 2303	51.2 - 883.0	

Wilcoxon test was done. P value < 0.05 was considered statistically significant.

Discussion

The study population had a mean age of 28.1 years, which is younger compared to other studies where the mean age at diagnosis was reported to be around 38.4 years [21]. This difference in age could be attributed to the demographic characteristics of the population studied. The female predominance (92.3%) in this study aligns with the well-documented higher prevalence of lupus nephritis in women [22] The mean BMI of 24.2 kg/m² in this study is consistent with findings from other research, which reported similar BMI ranges in lupus nephritis patients [23]. The prevalence of hypertension (46.2%) among respondents is comparable to Wong et al., 2024 showing significant association between lupus nephritis and hypertension [24]. Anemia was present in 30.8% of respondents, which is in line with the known hematologic manifestations of SLE [25]. Edema was observed in 59.6% of respondents, reflecting the common occurrence of nephrotic syndrome in lupus nephritis patients [26]. Proteinuria was present in a significant proportion of respondents, with 48.1% having proteinuria (++) and 34.6% having proteinuria (+++), which is consistent with the high prevalence of proteinuria reported in lupus nephritis [27]. For histological classification, 57.7% of respondents were classified as Class IV lupus nephritis, followed by 17.3% as Class III. This distribution is similar to other studies that have reported a higher prevalence of Class IV lupus nephritis, which is associated with more severe renal involvement [28].

In this study, hydroxychloroquine was administered to 100% of respondents, reflecting its widespread use in managing lupus nephritis. This aligns with the KDIGO 2024 Clinical Practice Guideline, which recommends hydroxychloroquine for all lupus nephritis patients to reduce the risk of kidney flares, end-stage kidney disease (ESKD), and death [29]. Cyclophosphamide combined with corticosteroids was used in 61.5% of respondents, consistent with standard treatment protocols for Class III and IV lupus nephritis [30]. Mycophenolate mofetil (MMF) combined with corticosteroids was administered to 23.1% of respondents, supported by evidence demonstrating its efficacy and favorable side effect profile compared to cyclophosphamide [31]. Corticosteroids alone were given to 15.4% of respondents, comparable to other studies. The response rates observed were 67.3% complete response, 28.8% partial response, and 3.8% no response. KDIGO guidelines note that treatment response rates can vary, but achieving a complete or partial response in a significant proportion of patients is a common goal [29]. The high rate of complete response in this study underscores the effectiveness of the treatment regimens used.

At baseline, Class IV lupus nephritis patients exhibited the highest mean urine KIM-1 levels (672.7 ± 504.0 ng/mL). After six months, the mean urine KIM-1 levels decreased but remained highest in Class IV patients (231.6 ± 191.4 ng/mL). At baseline, the mean urine KIM-1 level was significantly higher in the proliferative group (588.5 ± 474.3 ng/mL) compared to the non-proliferative group (191.77 ± 64.22 ng/mL). This trend persisted at six months, with the proliferative group showing higher mean urine KIM-1 levels (202.8 ± 176.2 ng/mL) than the non-proliferative group (88.1 ± 14.77 ng/mL). Liu et al., 2020 demonstrated that urine KIM-1 levels were significantly higher in active lupus nephritis patients compared to inactive patients with a positive correlation between urine KIM-1 levels and renal disease activity [32]. Nozaki et al., 2014 reported elevated urine KIM-1 levels in patients with active lupus nephritis [20]. El-Gendi et al. (2018) found that urine KIM-1 levels were significantly higher in patients with active nephritis compared to those with inactive nephritis and demonstrated a strong correlation between urine KIM-1 levels and markers of renal function [33]. These findings indicate a strong association between urine KIM-1 levels and the severity of lupus nephritis, with higher levels correlating with more severe disease, identifying urine KIM-1 as a reliable biomarker for detecting and monitoring lupus nephritis [20,34,35].

In present study, statistically significant differences ($p < 0.05$) in mean urine KIM-1 levels among patients with different tubulointerstitial inflammation group was observed. Seo et al., 2013 demonstrated that urine KIM-1 levels were correlating with the severity of tubulointerstitial injury [36]. Tanase et al., 2019 reported that KIM-1 is a reliable marker for identifying kidney injury and its associated inflammatory response [37]. These studies support the current findings, indicating that higher urine KIM-1 levels are indicative of more severe renal inflammation and damage.

In this study, urine KIM-1 levels were significantly ($p < 0.05$) lower in respondents with a complete response compared to those with partial or no response, both at baseline and after six months, indicating a strong association between lower urine KIM-1 levels and better treatment outcomes. These findings are consistent with other published research

highlighting the role of KIM-1 as a biomarker for renal injury and its potential to predict treatment response [37,38]. The study found significant differences in mean serum creatinine and serum albumin levels between response groups, which are critical for assessing renal function and kidney damage. Significant differences in mean urinary PCR at six months, but not at baseline, suggest changes in urinary PCR over time may be a more sensitive indicator of treatment response.

Conclusion

Among lupus nephritis patients, the use of hydroxychloroquine, cyclophosphamide, MMF, and corticosteroids aligns with current clinical practice guidelines and demonstrates the effectiveness of these therapies in achieving favorable treatment outcomes. The significant associations between urine KIM-1 levels and disease severity, as well as its correlation with renal function markers, highlight its utility in clinical practice. Future research should focus on further validating urine KIM-1 as a biomarker and exploring its potential in guiding treatment decisions and predicting patient outcomes in lupus nephritis.

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