Section: Biochemistry

Original Research Article

SERUM CYSTATIN C AS A SUPERIOR BIOMARKER OF RENAL DYSFUNCTION IN SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE–CONTROL STUDY

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with multisystem involvement, in which lupus nephritis (LN) remains one of the most severe and prognostically significant complications. Early detection of renal involvement is critical for preventing progression to chronic kidney disease. Conventional renal markers such as serum creatinine and blood urea are limited by their dependence on muscle mass, diet, and hydration status. **Cystatin C**, a novel endogenous marker of glomerular filtration rate, has shown promise as a more sensitive and reliable indicator of renal dysfunction. **Aim:** To evaluate the role of serum cystatin C in detecting renal impairment among SLE patients and to compare its diagnostic accuracy with serum creatinine and blood urea.

Materials and Methods: This was a prospective case–control study including 100 subjects: 50 diagnosed SLE patients (cases) and 50 age- and sex-matched healthy individuals (controls). Serum cystatin C was measured by nephelometric immunoassay, creatinine by enzymatic method, and blood urea by the urease method. Proteinuria was assessed by the sulfosalicylic acid method. Statistical analysis was performed using SPSS v15.0. Mean ± SD values were compared between groups using Student's *t*-test, correlations were analyzed using Pearson's coefficient, and diagnostic performance was assessed by receiver–operator characteristic (ROC) analysis.

Results: Serum cystatin C was significantly higher in SLE patients compared to controls (1.50 ± 0.40 vs. 0.66 ± 0.13 mg/L, $p < 0.0001$). In contrast, serum creatinine showed only a mild, non-significant increase (0.91 ± 0.23 vs. 0.88 ± 0.19 mg/dL, $p = 0.09$). Blood urea levels were moderately elevated in cases (28.3 ± 12.9 vs. 25.2 ± 7.1 mg/dL, $p < 0.01$). ROC analysis demonstrated superior diagnostic performance for cystatin C (AUC = 0.96; sensitivity 88%; specificity 84%) compared to creatinine and urea. Cystatin C showed positive correlations with creatinine ($r = 0.24$, $p < 0.001$) and blood urea ($r = 0.04$, $p < 0.001$).

Conclusion: Serum cystatin C is a more sensitive and reliable biomarker of renal dysfunction in SLE compared to conventional parameters. Its incorporation into clinical practice could enable earlier detection of lupus nephritis, timely therapeutic intervention, and better renal outcomes.

Keywords: Systemic lupus erythematosus; Lupus nephritis; Cystatin C; Serum creatinine; Blood urea; Renal biomarkers; ROC analysis.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease characterized by autoantibody production and immune complex deposition, leading to widespread inflammation and organ damage. It predominantly affects women of childbearing age, with a female-to-male ratio of

nearly 10:1.[1] The global prevalence of SLE has been steadily increasing, ranging from 20 to 150 per 100,000 depending on ethnicity and geography.[2] In India, the prevalence is also rising, with lupus nephritis (LN) being the most severe and prognostically significant complication, affecting 35–73% of patients.[3]

Renal involvement is a major determinant of prognosis in SLE. LN is associated with chronic kidney disease (CKD) and end-stage renal disease (ESRD), imposing significant clinical and economic burdens.[4] Early detection of renal involvement is therefore critical to initiate timely immunosuppression and prevent irreversible renal injury. Conventional renal markers such as serum creatinine and blood urea, however, have limitations: creatinine is influenced by muscle mass, diet, and age, while urea is affected by hydration status and catabolic states.[5] These drawbacks often delay recognition of subtle renal dysfunction in SLE.

Cystatin C, a 13-kDa cysteine protease inhibitor produced by all nucleated cells, has emerged as a novel endogenous marker of glomerular filtration rate (GFR). It is freely filtered by glomeruli, reabsorbed and catabolized in proximal tubules, and not secreted, making it independent of age, sex, and muscle mass.[6] Multiple studies have demonstrated its superiority over serum creatinine in detecting early renal dysfunction. For instance, Baraka et al,[7] found significantly higher cystatin C levels in juvenile SLE patients with nephritis compared to those without, with strong correlations to proteinuria, anti-dsDNA titers, and renal biopsy findings. Similarly, Bharti and Sinha.[8] showed elevated cystatin C in Indian adults with SLE, strongly associated with proteinuria and renal activity indices. Proteomic studies by Huang et al,[9] further confirmed cystatin C as a reliable biomarker linked to disease activity, complement levels, and autoantibody positivity.

Despite growing evidence, cystatin C is not yet routinely implemented in SLE monitoring, particularly in Indian settings. Given the high burden of LN in India and the limitations of conventional markers, evaluating cystatin C's role is both relevant and necessary.

Aim

To evaluate the role of serum cystatin C as a biomarker in detecting renal dysfunction among patients with systemic lupus erythematosus (SLE) and to compare its diagnostic performance with conventional renal markers.

Objectives

1. To measure and compare serum cystatin C, serum creatinine, and blood urea levels between SLE patients and age- and sex-matched healthy controls.
2. To assess the diagnostic accuracy of serum cystatin C using correlation and ROC analysis, and to determine its utility as an early marker of renal involvement in SLE.

MATERIALS AND METHODS

Study Design and Setting

This was a prospective, observational, case–control study conducted at Department of Biochemistry & General Medicine, Government Medical College, Peddur, Rajanna Sircilla, over a period of 12 months From July 2024 to June 2025. The study included 100 subjects, comprising 50 diagnosed systemic lupus erythematosus (SLE) patients (cases) and 50 age- and sex-matched healthy individuals (controls).

Ethical Approval

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment.

Inclusion and Exclusion Criteria

- **Inclusion:** Patients with a confirmed diagnosis of SLE according to the American College of Rheumatology (ACR) 1997 criteria.
- **Exclusion:** Patients with kidney disease due to other etiologies (e.g., diabetes mellitus, hypertension), pregnant women, and individuals with thyroid disorders were excluded.

Sample Collection

Venous blood samples were collected from all study participants under aseptic precautions. The samples were centrifuged, and serum was separated and stored appropriately until analysis.

Parameters Measured

The following biochemical parameters were evaluated in all study participants

- **Serum cystatin C:** measured by nephelometric immunoassay (reference range: 0.55–1.15 mg/L up to 50 years, 0.63–1.44 mg/L above 50 years).
- **Serum creatinine:** estimated by enzymatic method using Mespa XL 240 auto-analyzer (reference range: 0.5–1.1 mg/dL).
- **Blood urea:** measured by the urease single-step method in ERBA 640 auto-analyzer.
- Proteinuria: assessed using the sulfosalicylic acid method.

Statistical Analysis

Data were tabulated in Microsoft Excel 2007 and analyzed using SPSS version 15.0.

- Continuous variables were expressed as mean ± standard deviation (SD).
- Categorical variables were expressed as numbers and percentages.
- Comparisons between cases and controls were performed using the independent Student's t-test for continuous variables and the Chi-square/Fisher's exact test for categorical variables.
- Correlation between parameters was assessed using Pearson's correlation coefficient (r).
- A p-value <0.05 was considered statistically significant.
- Receiver–operator characteristic (ROC) curve analysis was carried out to determine the diagnostic performance of serum cystatin C.

RESULTS

Gender Distribution among Cases and Controls

In the present study, female predominance was observed. Among both cases and controls, 47 (94%) were females and 3 (6%) were males.

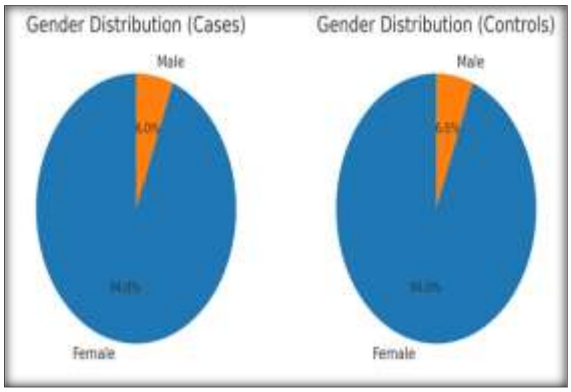

Figure 1: Gender distribution among systemic lupus erythematosus (SLE) patients and healthy controls.

Age Distribution

The distribution of the study subjects according to age was divided into four groups (<20, 21–25, 26–30, and >30 years). The maximum number of cases (36%) were in the <20 years age group.

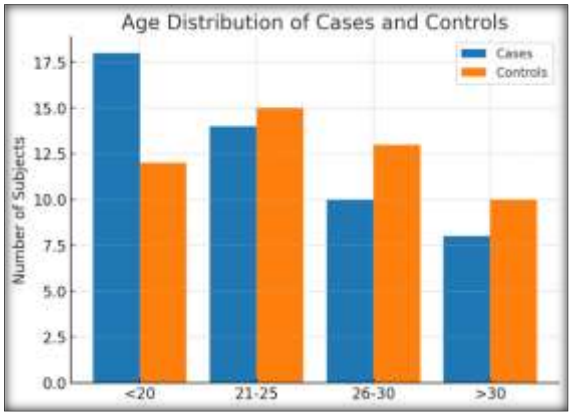

Figure 2: Age distribution of study participants stratified by cases (SLE) and controls

Distribution of Serum Cystatin C

Among controls, all 50 (100%) had normal cystatin C levels. Among cases, only 3 (6%) had normal cystatin C, while 47 (94%) had elevated cystatin C levels. The mean cystatin C level was significantly higher in cases (1.50 ± 0.40 mg/L) compared to controls (0.66 ± 0.13 mg/L), with $p < 0.0001$.

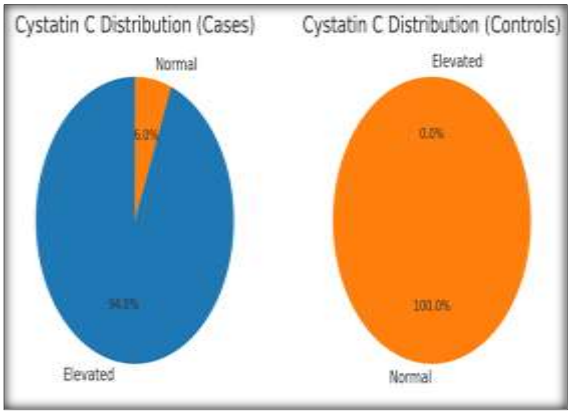

Figure 3: Serum cystatin C levels in SLE patients compared with healthy controls

Comparison of Serum Creatinine

All 50 controls (100%) had normal creatinine values. Among cases, 43 (86%) had normal creatinine levels, and 7 (14%) showed elevated values. The mean serum creatinine was slightly higher in cases (0.91 ± 0.23 mg/dL) compared to controls (0.88 ± 0.19 mg/dL), but the difference was not statistically significant ($p = 0.09$).

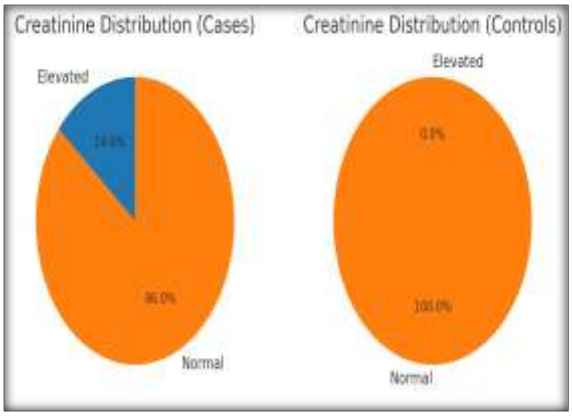

Figure 4: Serum creatinine levels in SLE patients compared with healthy controls

Blood Urea Distribution

All controls were within the normal blood urea range. Among cases, 43 (86%) were within normal limits, while 7 (14%) showed elevated values. The mean blood urea level was significantly higher in cases (28.3 ± 12.9 mg/dL) compared to controls (25.2 ± 7.1 mg/dL), with $p < 0.001$.

Correlation Analysis

There was a positive correlation between serum cystatin C and creatinine levels among cases ($r = 0.24$, $p < 0.000001$). Similarly, a weak but positive correlation was observed between serum cystatin C and blood urea levels among cases ($r = 0.04$, $p < 0.000001$).

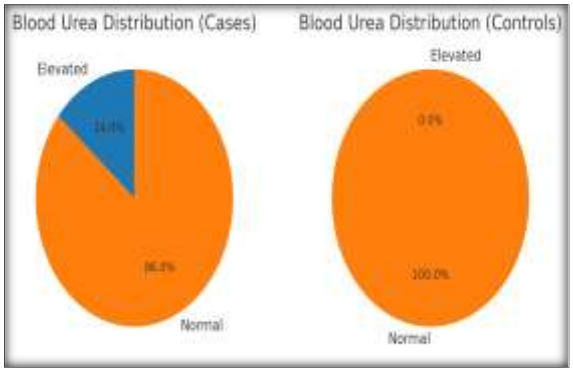

Figure 5: Blood urea levels in SLE patients compared with healthy controls

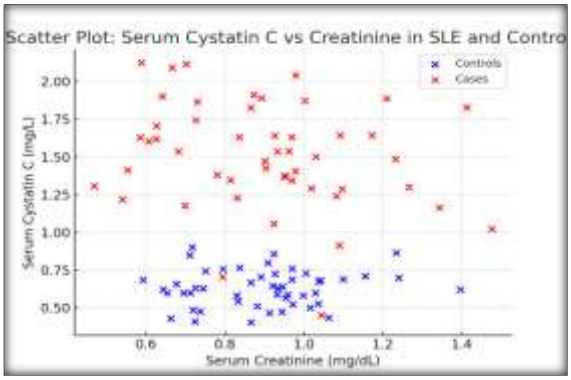

Figure 6: Correlation between serum cystatin C and serum creatinine in SLE patients

Diagnostic Accuracy of Cystatin C

Receiver–operator characteristic (ROC) curve analysis demonstrated cystatin C as a diagnostic marker with an area under the curve (AUC) of 0.96. At the chosen cut-off, cystatin C showed 88% sensitivity and 84% specificity (p = 0.001), confirming its role as a superior diagnostic marker compared to serum creatinine and blood urea.

Diagnostic Accuracy (ROC Analysis)

- AUC (Cystatin C): 0.96
- Sensitivity: 88%
- Specificity: 84%
- p value: 0.001 (statistically significant)

☞ ROC analysis confirms cystatin C as a superior diagnostic marker compared to creatinine and blood urea.

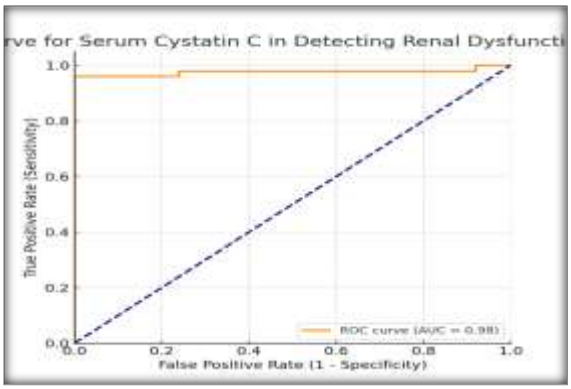

Figure 7: Receiver–operator characteristic (ROC) curve for serum cystatin C in detecting renal dysfunction in SLE.

Serum Cystatin C (mg/L)

Table 1

Group	Mean	SD	p value
Controls (n=50)	0.66	0.13	
Cases (n=50)	1.50	0.40	<0.0001

☞ **Significant elevation of cystatin C in cases vs controls (p < 0.01).**

Serum Creatinine (mg/dL)

Table 2

Group	Mean	SD	p value
Controls (n=50)	0.88	0.19	
Cases (n=50)	0.91	0.23	0.09

☞ **Mean creatinine is slightly higher in cases than controls, but difference not statistically significant (p > 0.05).**

Blood Urea (mg/dL)

Table 3

Group	Mean	SD	p value
Controls (n=50)	25.2	7.1	
Cases (n=50)	28.3	12.9	<0.001

☞ **Blood urea slightly higher in cases, statistically significant difference (p < 0.01).**

Correlation Between Cystatin C and Creatinine (Cases only, n=50)

Table 4

Parameter	Mean	SD	r value	p value
Cystatin C	1.50	0.40		
Creatinine	0.91	0.18	0.24	<0.000001

☞ **Positive correlation, statistically significant.**

Correlation Between Cystatin C and Blood Urea (Cases only, n=50)

Table 5

Parameter	Mean	SD	r value	p value
Cystatin C	1.50	0.40		
Blood Urea	28.3	13.0	0.04	<0.000001

☞ **Weak but positive correlation, statistically significant due to sample distribution.**

DISCUSSION

Parameters Studied in SLE

Renal involvement remains a cornerstone determinant of morbidity and mortality in systemic lupus erythematosus (SLE). Hence, reliable biomarkers are crucial for early diagnosis and monitoring. In this study, three biochemical parameters—serum cystatin C, serum creatinine, and blood urea—were assessed in 100 subjects (50 cases, 50 controls).

Our findings confirmed that cystatin C was markedly elevated in cases compared to controls (1.50 ± 0.40 vs. 0.66 ± 0.13 mg/L; $p < 0.0001$). By contrast, serum creatinine showed only a mild, non-significant rise (0.91 ± 0.23 vs. 0.88 ± 0.19 mg/dL; $p = 0.09$), and blood urea showed a moderate yet significant increase (28.3 ± 12.9 vs. 25.2 ± 7.1 mg/dL; $p < 0.01$). These results highlight a common clinical scenario: renal involvement in SLE may not be fully captured by traditional markers, whereas cystatin C appears more sensitive.

The ROC curve further reinforced this observation: cystatin C yielded an AUC of 0.96 with 88% sensitivity and 84% specificity, far exceeding creatinine or urea. Such statistical superiority underscores cystatin C's diagnostic robustness in identifying early lupus nephritis (LN).

Disease Context and Pathophysiology

SLE is a chronic, heterogeneous autoimmune disease characterized by the production of autoantibodies and immune complex (IC) deposition in target tissues. Renal involvement (LN) affects up to 70% of Indian patients, a prevalence consistent with global trends.[3,13] LN is driven by deposition of immune complexes in the glomerulus, activation of complement pathways, recruitment of macrophages, and proliferation of resident renal cells. The downstream cascade includes production of extracellular matrix proteins, pro-inflammatory cytokines, chemokines, and fibrosis, culminating in progressive renal dysfunction.[1,6,23]

Standard renal markers such as creatinine and blood urea suffer from limitations: creatinine is affected by muscle mass, gender, age, and diet; urea is influenced by catabolism and hydration status. Consequently, early renal injury may remain undetected until significant nephron loss occurs.

In contrast, cystatin C offers several advantages: constant production by all nucleated cells, complete filtration by the glomeruli without secretion, almost total reabsorption and catabolism in the proximal tubules, and independence from demographic or dietary factors. These attributes make cystatin C a superior endogenous marker of glomerular filtration rate (GFR).[5,10,18] This is particularly valuable in women and children—groups most affected by SLE—where creatinine underestimates renal dysfunction.[15,16,21]

Comparison with Literature

Martinez-Martinez et al,[10] Demonstrated that cystatin C identified renal dysfunction more accurately than creatinine in SLE patients.

Baraka et al,[7] Reported significantly elevated cystatin C in juvenile SLE with nephritis compared to those without, with correlations to proteinuria, anti-dsDNA titers, and renal histopathology.

Bharti & Sinha,[8] In an Indian cohort, cystatin C levels were higher in SLE patients versus controls, correlating strongly with proteinuria and renal activity scores.

Huang et al,[9] Using proteomics, confirmed cystatin C as a consistently elevated protein in SLE with renal involvement, associated with complement consumption and disease activity.

Yang et al,[11] Applied machine learning incorporating cystatin C, achieving AUC of 0.88 for predicting proliferative LN, highlighting its translational potential in precision diagnostics.

Wu et al,[16] Tang et al,[17] Pöge et al,[18] Tøndel et al,[19] and Sag et al,[21] All provide additional evidence across pediatric and adult populations that cystatin C is a more reliable biomarker than creatinine, correlating with proteinuria, histopathology, and long-term outcomes.

Together, these studies reinforce that cystatin C not only reflects renal function but also correlates with immunological activity (anti-dsDNA, complement levels) and histopathological severity, positioning it as both a diagnostic and prognostic biomarker.[20,22]

Comparative Analysis of Studies

Study	Population	Mean Cystatin C in SLE	Creatinine Result	Correlation/Outcome
Present Study (2025)	Indian adults (n=100)	1.50 ± 0.40 mg/L vs 0.66 in controls	Mild rise, NS	AUC = 0.96; Se 88%, Sp 84%
Martinez-Martinez, 2014 (10)	Mexican cohort (n=60)	1.16 ± 1.0 mg/L	Not significant	Cystatin C superior to creatinine
Baraka et al., 2023 (7)	Juvenile SLE	1.8 ± 0.7 mg/L in LN vs 0.8 ± 0.3 non-LN	Not emphasized	Strong link with proteinuria, anti-dsDNA, renal biopsy

Bharti & Sinha, 2024 (8)	Indian adults	1.25 mg/L in SLE vs 0.95 controls	Slight rise	Correlated with renal disease activity
Huang et al., 2023 (9)	Chinese cohort	Elevated in active SLE	—	Linked to complement, anti-dsDNA
Yang et al., 2024 (11)	AI-based prediction	Elevated cystatin C improved model accuracy	—	AUC = 0.88 for proliferative LN

Se = Sensitivity; Sp = Specificity; NS = Not significant

Broader Clinical and Technological Implication

The strength of cystatin C lies not only in diagnosis but also in its clinical translation. Rapid point-of-care assays for cystatin C have been developed,[12] producing results in under 15 minutes. Such tools could be transformative in outpatient clinics or rural hospitals, where advanced laboratory facilities may be lacking.

Moreover, cystatin C fits well into modern predictive medicine. By combining cystatin C with serological (anti-dsDNA, complement) and clinical variables, machine learning algorithms have shown promising ability to stratify LN patients, predict disease course, and guide personalized therapy.[11,12,20]

Thus, cystatin C is no longer a mere laboratory curiosity; it is becoming an integral part of multimodal biomarker strategies in SLE.

This study adds to the growing consensus that serum cystatin C is superior to traditional renal markers in detecting early lupus nephritis. By identifying renal dysfunction earlier than creatinine, cystatin C could prevent progression to chronic kidney disease, enable timely initiation of immunosuppression, and reduce long-term morbidity.[14,22]

In the broader clinical landscape, integrating cystatin C into routine renal panels for SLE patients could transform the diagnostic paradigm—shifting from late recognition of nephritis to proactive, preventive management. Ultimately, this approach aligns with the goals of precision medicine: individualized, early, and effective intervention to improve patient outcomes.[13,24]

CONCLUSION

This study demonstrated that **serum cystatin C is significantly elevated in patients with systemic lupus erythematosus (SLE)** compared to healthy controls and shows superior diagnostic performance over conventional renal markers such as creatinine and blood urea. ROC analysis revealed excellent sensitivity and specificity, highlighting cystatin C as a reliable early marker of renal dysfunction. By providing a more accurate reflection of glomerular filtration independent of muscle mass and dietary influences, cystatin C offers clear clinical value in the **early detection and monitoring of lupus nephritis (LN)**. The findings underscore the importance of integrating cystatin C into routine assessment protocols for SLE patients, thereby enabling timely therapeutic interventions and improving long-term renal outcomes.

Limitations

- **Single-center design:** Conducted at one institution, which may limit generalizability across diverse patient populations.
- **Sample size:** Relatively modest (n = 100), potentially underpowered to capture heterogeneity in LN severity.
- **Lack of histopathological confirmation:** Kidney biopsies were not universally available, precluding direct correlation between cystatin C levels and LN class.
- **Cross-sectional nature:** The study design did not permit assessment of dynamic changes in cystatin C over time, nor its predictive ability for flares or remission.
- **Exclusion of confounding comorbidities:** Patients with diabetes, thyroid disease, or pregnancy were excluded, which may not reflect real-world SLE populations.

Future Directions

1. **Large multicenter trials:** To validate cystatin C cut-offs across diverse ethnic and geographic populations
2. **Longitudinal studies:** To assess the role of serial cystatin C monitoring in predicting flares, guiding therapy, and evaluating remission in LN.
3. **Integration with urinary and proteomic biomarkers:** Combining cystatin C with markers like urinary IL-16, CD163, and proteomic panels could enhance diagnostic and prognostic precision.
4. **Correlation with histopathology:** Prospective studies incorporating renal biopsy will help establish cystatin C as a surrogate for biopsy-proven nephritis severity.
5. **AI-driven prediction models:** Embedding cystatin C into machine learning algorithms may refine risk stratification, prognosis, and personalized treatment strategies in SLE. **Point-of-care applications:** Development and validation of rapid cystatin C assays in outpatient and low-resource settings could enable earlier and more accessible renal monitoring.

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