ORIGINAL ARTICLE

Importance of Urinary Monocytic Chemoattractant Protein-1 as a Non Invasive Biomaker for lupus Nephritis Activity

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Abstract

Background: ***About 40-60% of people with systemic lupus erythematosus (SLE) will also have lupus nephritis (LN), a renal involvement symptom that can show in a variety of ways.***

Aim and objectives: ***For the purpose of assessing uMCP-1, or urine monocyte chemotactic protein-1, as a noninvasive indicator of lupus nephritis activity***

Subjects and methods: ***Of the 90 participants in this cross-sectional study, 30 were healthy controls of the same age and sex, and 60 were patients with SLE according to four or more American College of Rheumatology criteria. The participants' ages ranged from 18 to 55. From May 2023 through January 2024, patients were enrolled from the nephrology outpatient clinics and inpatient wards of Al-Azhar University Hospitals.***

Results: ***The active LN group had significantly greater urinary MCP-1 levels compared to the inactive LN group and controls ($p\sim0.001$). A strong positive connection was found between uMCP-1 and LN activity. Highly significant ($p<0.001$). In the active LN group, uMPC-1 was positively correlated with 24-hour urine protein, anti-dsDNA, renal SLEDAI, and biopsy activity index.***

Conclusion: ***There is a strong association between the clinical and laboratory parameters of the activity investigated and the urinary MCP-1 levels, which reveal a large increase in cases of LN activity. uMCP-1 could differentiate between renal disease that was inactive and active LN and/or recurrence. It reliably detected LN activity and relapse with high sensitivity and specificity, making it an excellent diagnostic tool.***

Keywords: Lupus nephritis activity; uMCP-1

1. Introduction

Patients with systemic lupus erythematosus (SLE), an immune-mediated disorder that can affect multiple systems, may exhibit a broad range of symptoms and signs as well as different laboratory findings. The severity of the disease and the organs affected determine the prognosis, which can be unpredictable.[1]

Renal involvement in systemic lupus erythematosus (SLE) is prevalent and can be life-threatening.[2] The definitive method for diagnosing and monitoring lupus nephritis (LN) continues to be kidney biopsy.[3]

Because kidney biopsies are intrusive and can cause a variety of problems,[4] there is a pressing need to find new noninvasive biomarkers that can accurately reflect the degree and activity of LN.[5]

One member of the CC subfamily of cytokines is monocyte chemoattractant protein-1 (MCP-1), which is also known as chemokine ligand 2 (CCL2).[6]

Research has demonstrated that the level of MCP-1 in the urine of LN patients is inversely proportional to the severity of their disease and the frequency of flare-ups, and that this level decreases when nephritis is treated.[7]

This study set out to assess uMCP-1, or urine monocyte chemotactic protein, as a noninvasive indicator of LN activity .

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2. Patients and methods

The participants in this cross-sectional study ranged in age from 18 to 55. Of them, 90 were assessed; 60 were determined to have systemic lupus erythematosus (SLE) according to four or more criteria set by the American College of Rheumatology. The remaining 30 were included as healthy controls, matched for age and sex. Patients were enrolled from May 2023 to January 2024 at Al-Azhar University Hospitals, specifically from the Nephrology outpatient clinics and inpatient wards.

The participants in the study were split into three categories: A total of thirty patients were divided into three groups: those with active LN (high serum creatinine, proteinuria greater than one gram per day or less than half a gram per day, hematuria, or active sediment in urine) and those with inactive LN. The third group consisted of thirty healthy controls of the same age and sex who did not exhibit any signs of renal or autoimmune disease.

Exclusion criteria:

People with LN who are either under the age of 18 or over the age of 55, and who have either chronic kidney disease (CKD), end-stage renal disease (ESRD), or have received a kidney transplant, patients who have had malignant diseases, and patients with liver cell failure, heart failure, respiratory failure, or infectious diseases in a month.

Methods:

We took a thorough medical history from all patients and controls. For SLE patients, we assessed variables such as patients' ages, sickness durations, urine symptoms, SLE signs (including joint aches, rash, cutaneous photosensitivity, and symptoms related to the central nervous system, such as seizures), hypertension symptoms, and treatment modalities.

All patients underwent a comprehensive clinical evaluation that included assessment of vital signs (temperature, pulse, peripheral pulsations, and blood pressure) as well as anthropometric measurements (weight, height, and body mass index), distribution of skin rashes, affectation of joints, examination of the chest, heart, abdomen, and central nervous system.

Serum creatinine, blood urea, C-reactive protein, uric acid, S. albumin, complete blood count, and liver function tests were part of the baseline evaluation. C3, C4, ANA IF, and anti ds DNA titer were among the serological indicators that were tested for activity.

Proteinuria was assessed using 24-hour urinary protein and urine analysis (new morning midstream urine) was performed. The QUANTIKINE Human CCL2/MCP-1 solid phase ELISA is used for the MCP-1 test in accordance with the manufacturer's instructions for the evaluation of urinary MCP-1 (uMCP-1).

The purpose of the SLEDAI was to evaluate the severity of SLE. In order to determine the relationship between clinical evaluation of LN and the urinary monocyte chemotactic protein-1, a noninvasive biomarker for LN, the renal component of SLEDAI (R-SLEDAI) was evaluated. All four parts of the SLEDAI questionnaire—urine red blood cells (RBCs), white blood cells (WBCs), proteinuria, and casts—make up R-SLEDAI.

Group 1's active LN patients all had percutaneous renal biopsies done. The kidney histopathology was categorized using the LN criteria set forth by the World Health Organization.

Sample collection and treatment:

Three milliliters of blood were collected via venipuncture, allowed to clot, and the serum was isolated through centrifugation at 2500×g for 10 minutes, ensuring that hemolyzed and lipemic sera were excluded. Three milliliters of freshly voided morning urine samples were procured and analyzed for quantitative evaluation of proteinuria and active urinary sediments (RBCs > 5/HPF, WBCs > 5/HPF, RBC casts, granular casts). An additional sample was centrifuged, and the supernatant was preserved for the measurement of MCP-1 levels. Specimens were sealed and stored at -20°C until the assay was conducted, with repeated freeze-thaw cycles being avoided. Immunoassays were performed using the MCP-1 ELISA Kit for urine MCP-1 (uMCP-1) assessment via ELISA with QUANTIKINE Human CCL2/MCP-1

Statistical analysis:

The data collection, tabulation, and statistical analysis were carried out using Windows software version 22.0 developed by SPSS Inc. of Chicago, Illinois, USA. Using the Shapiro-Whitney U test, we looked for evidence that the data followed a normal distribution. The qualitative data were shown using relative percentages and frequencies. To determine the difference between the qualitative variables, the chi-square test (x2) and Fisher's exact test were employed. Parametric data were presented as mean±SD (Standard deviation), whereas non-parametric data were presented as median and range.

When comparing normally distributed variables across more than two dependent groups, a one-way analysis of variance (ANOVA) was employed. For parametric variables, we utilized an independent T-test, and for non-parametric variables, we used a Mann-Whitney test to determine the difference between the three groups' quantitative variables. When looking for relationships between variables, we utilized

Spearman's correlation coefficient. We consider values close to 1 to indicate strong correlation and values near 0 to indicate weak correlation. The (+) sign indicates direct correlation, meaning that an increase in the frequency of the independent variable leads to an increase in the frequency of the dependent variable. On the other hand, the (-) sign indicates inverse correlation, meaning that an increase in the frequency of the independent variable decreases the frequency of the dependent variable.

Ethical Consideration:

The research took place in the inpatient and outpatient wards of the departments of nephrology and rheumatology at Al-Azhar University Hospitals. The patients were provided with a thorough description of the procedure and any potential risks in order to get their signed informed consent, which was then reviewed and approved by the ethical committee.

3. Results

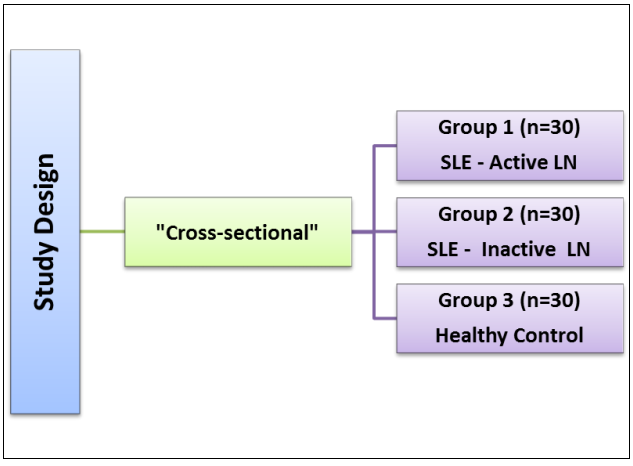

Figure 1. Visual depiction of the research plan.

Table 1. Distribution of demographic data between studied groups.

	ACTIVE LN GROUP N=30	INACTIVE LN GROUP N=30	CONTROL GROUP N=30	P-VALUE
AGE (YEARS) MEAN±SD	33.77±8.65	35.9±7.09	32.8±7.83	0.3
SEX				
MALE	7(23.3%)	9(30%)	7(23.3%)	0.79
FEMALE	23(76.7%)	21(70%)	23(76.7%)	
BMI MEAN±SD	26.8±4.36	25.62±3.49	26.27±3.64	0.49
DISEASE DURATION (MONTHS) MEAN±SD	37.63±21.20	60.83±35.45	0.00±0.00	<0.001

P-value>0.05: Not significant, p = 0.001 is very significant, and P = 0.05 is statistically significant., Standard deviation (SD) and body mass index (BMI)

When it came to age, sex, and body mass index, the groups under study did not differ significantly from one another, but when it came to illness duration, there were substantial variances, (table 1; figures 2 & 3).

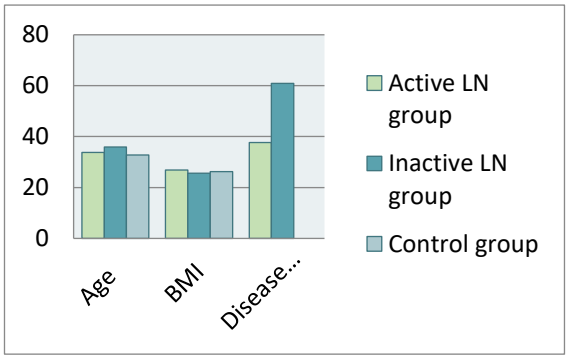

Figure 2. Distribution of age, BMI and disease duration between studied groups.

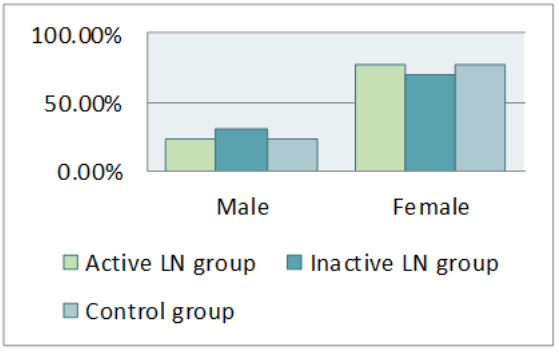

Figure 3. Sex distribution across the groups under study.

Table 2. Distribution of kidney function tests across the groups under study.

	ACTIVE LN GROUP N=30	INACTIVE LN GROUP N=30	CONTROL GROUP N=30	P-VALUE
ALBUMIN (G/DL) MEAN±SD	2.99±0.4	3.56±0.69	4.03±0.62	<0.001
CREATININE (MG/DL) MEAN±SD	1.82±0.63	1.32±0.32	0.99±0.46	<0.001
UREA (MG/DL) MEAN±SD	58.36±30.54	39.53±17.08	22.03±4.7	<0.001
URIC ACID MEAN±SD	7.72±2.39	6.44±2.36	4.82±1.1	<0.001
PROTEINURIA (MG/24H) MEDIAN (RANGE)	2350(808-6340)	461.5(53.12-2657)	30(3-33)	<0.001

P-value>0.05: Not significant, pł0.001 is extremely significant, and P-value<0.05 is statistically significant., SD stands for standard deviation.

The groups under study differed statistically significantly in terms of albumin, creatinine, urea, uric acid, and proteinuria, (table 2; figures 4 & 5).

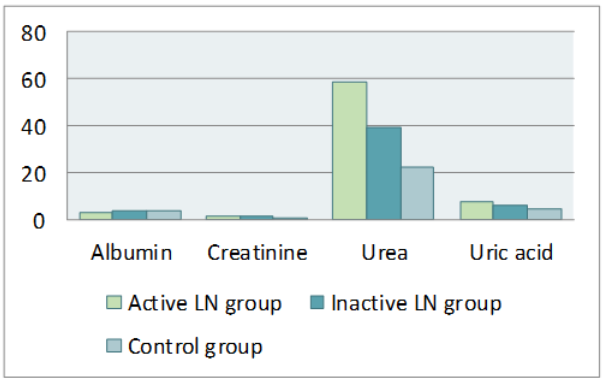

Figure 4. Distribution of kidney function in studied group.

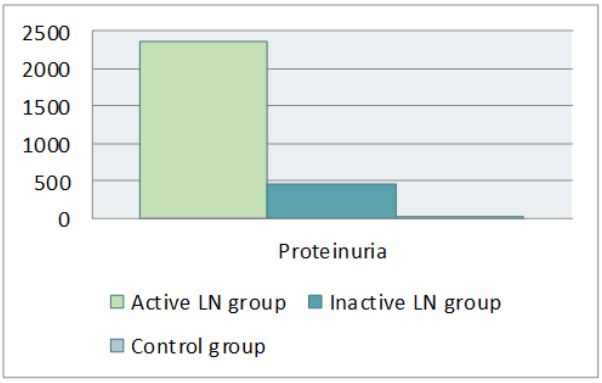

Figure 5. Distribution of proteinuria in studied group.

Table 3. Distribution of inflammatory markers between the studied groups.

	ACTIVE LN GROUP N=30	INACTIVE LN GROUP N=30	CONTROL GROUP N=30	P-VALUE
ESR (MM/HR) MEAN±SD	97.66 ±23.07	37.56±27.13	16.23±6.66	<0.001
CRP (MG/DL) MEDIAN (RANGE)	8.5(1.4-1.73)	6.29(1.22-31)	2.57(0.59-10.31)	0.001
ANTI-DSDNA (IU/ML) MEAN±SD	92.5±29.63	14.2±13.43	2.92±1.54	<0.001
C3 (MG/DL) MEAN±SD	50.52±12.09	110.01±23.41	120.04±40.85	<0.001
C4 (MG/DL) MEAN±SD	8.97±3.85	31.26±6.19	34.01±9.29	<0.001
		ANA IF		
< 1/20	0(0%)	0(0%)	30(100%)	<0.001
1/40	0(0%)	14(46.7%)	0(0%)	
1/80	0(0%)	15(50%)	0(0%)	
1/160	15(50%)	1(3.3%)	0(0%)	
1/320	13(43.3%)	0(0%)	0(0%)	
1/640	2(6.7%)	0(0%)	0(0%)	

P-value>0.05: Not significant, p^0.001 is very significant, and P<0.05 is statistically significant., ANA stands for antinuclear antibody, CRP for c-reactive protein, ESR for erythrocyte sedimentation rate, and SD for standard deviation.

The groups under study differed statistically significantly in terms of ESR, CRP, Anti-dsDNA, C3, C4, and ANA IF, (table 3; figures 6 & 7).

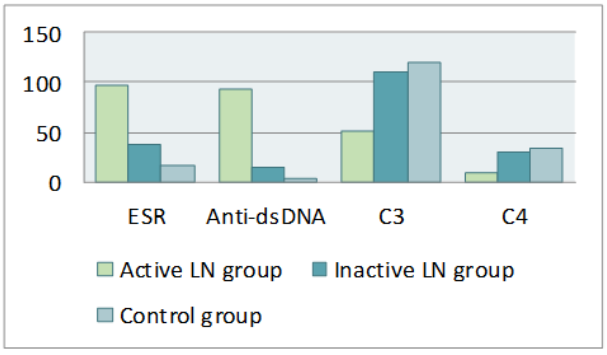

Figure 6. Distribution of inflammatory markers in studied group.

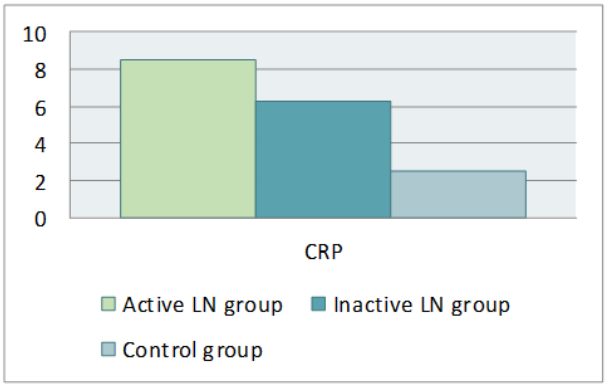

Figure 7. Distribution of CRP in studied group.

Table 4. Distribution of urinary MCP-1 between the studied groups.

	ACTIVE LN GROUP N=30	INACTIVE LN GROUP N=30	CONTROL GROUP N=30	P-VALUE
MCP-1 PG/MG MEAN±SD	1238.3±361.46	221.03±84.79	20.67±6.92	<0.001

P-value>0.05: Not significant, p = 0.001 is very significant, and P = 0.05 is statistically significant.Standard deviation, or SD.

In terms of uMCP-1, there was a statistically significant rise in the Active LN group of people, followed by the Inactive LN group and the Control group, (table 4; figure 8).

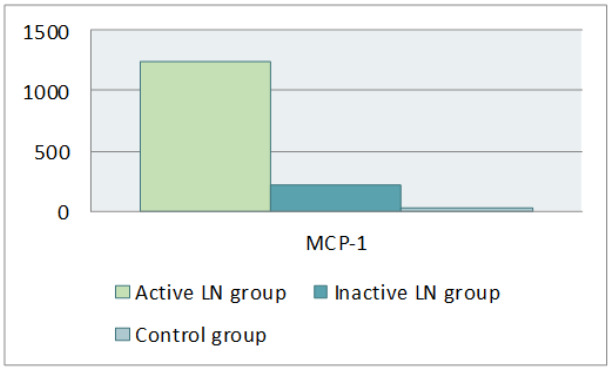

Figure 8. Distribution of Urinary MCP-1 between the studied groups.

Table 5. Correlation between activity of LN and uMCP-1.

	ACTIVITY OF LN	
	R	p
MCP-1	0.867	<0.001

According to this table, there was significant positive correlation between activity of LN and uMCP-1, (table 5; figure 9).

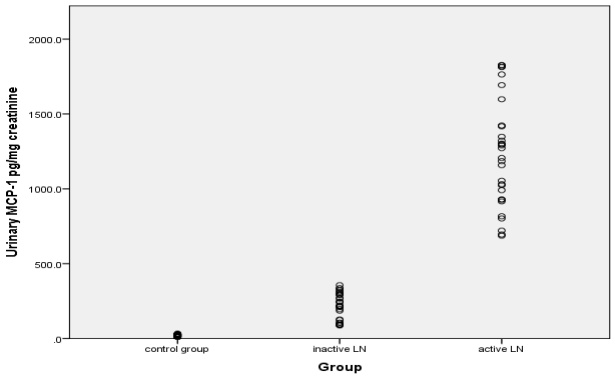

Figure 9. Correlation between activity of LN and uMCP-1.

4. Discussion

Lupus nephritis (LN) is a very serious clinical manifestation in patients with systemic lupus erythematosus (SLE), which is known to cause a high rate of infection and mortality.[8]

Although renal biopsy is the most accurate way to diagnose and categorize kidney histological abnormalities, it is too invasive to be used for ongoing patient monitoring. [3]

Numerous research efforts have focused on creating new, noninvasive biomarkers for LN, particularly in urine, which reflects the underlying biological processes of kidney injury and repair in illness.[9]

A β-chemokine called MCP-1 is in charge of bringing in monocytes and T-lymphocytes in both the acute and chronic stages of inflammation. [10]

It follows that these findings are consistent with Zedan et al. [11]

When looking at the comorbidities of hypertension, diabetes mellitus, chronic kidney disease, and cardiovascular illnesses, the present study found a statistically significant distinction among the groups.

Serum albumin, Creatinine, urea, uric acid, and proteinuria were all significantly different between the groups in this study. When comparing groups II and III, patients in group I exhibited lower serum albumin levels and higher Creatinine, urea, and uric acid levels.

Although both groups had proteinuria, it was more concentrated in the first (median proteinuria in group I was 2350 (808-6340) mg/24 hr, compared to 461.5 (53.12-2657) mg/24 hr in group II individuals with inactive LN). This corroborated what had been found in Abozaid et al.[12]

In this study, the levels of immunological markers such as ANA, anti-ds DNA titer, and ESR were considerably greater in the first group as opposed to the second and both groups were higher than the control group. This difference was shown to be statistically significant. In comparison to groups II and III, group I also absorbed more C3 and C4.

To back up this effort Elsaid et al.,[13] and Zedan et al.,[11] their research revealed a statistically significant variation in ESR, C3, C4, and anti-dsDNA among the tested populations.

All three groups had their uMCP-1 levels evaluated in this investigation. In comparison to the inactive LN group (221.03±84.79 pg/mg) and the control group (20.67±6.92 pg/mg), the levels in the active LN group were higher (1238.3±361.46 pg/mg).

According to this research El Shehaby et al.,[14] who found that active LN had substantially greater u MCP-1 levels than both the inactive LN and control groups.

Additional backing was provided by Taha et al.,[15] and Ding et al.,[16], 16 people who came up with identical findings.

Following in the footsteps of Davies et al.,[17] who discovered elevated uMCP-1 levels in SLE patients relative to healthy controls. The predictive power of uMCP-1 for LN activity is higher than that of other assessed markers.

A substantial positive association was found between the activity of LN and uMCP-1 (r=0.867, p <0.001), and the disease activity was assessed in group I using SLEDAI.

El Shehaby et al.,[14] and Abujam et al.,[18] came to the same conclusion. Additional backing was provided by El-Shinnawy et al.,[19] for whom the level of MCP-1 was observed to be significantly correlated with R-SLEDAI (p=0.002).

A strong positive correlation (r = 0.923, p <0.001) was seen in the present study between 24-hour urine protein and urinary MCP-1 in the active LN group.

Concurring with this research, Kim et al.,[20] and Alzawawy et al.,[21] discovered a positive correlation between proteinuria and MCP-1 excretion in urine. This corroborated what had been found in Živković et al.,[22] The researchers discovered a positive correlation between proteinuria and urinary MCP-1, but not with serum MCP-1 (r=0.839; P<0.001)

In this investigation, individuals with active lymph nodes (LN) were shown to have significantly higher levels of anti-dsDNA and urine MCP-1 (r = 0.954, p<0.001). With regard to

Kiani et al.,[23] and Al Zawawy et al.,[22] noticed identical outcomes.

The present investigation found a noteworthy inverse relationship between MCP-1, C3 (r=-0.575, p <0.001), and C4 (r=-0.719, p<0.001) in individuals suffering from active nephritis.

Concurring with this research El-Shehaby et al.,[14] and Taha et al.,[15] for whom the outcomes were same. Over the years that followed, this correlation strengthened and additional evidence emerged. Tawfik et al.,[24] and Zedan et al.,[11] reached the same conclusions.

While the chronicity index and urine MCP-1 did not correlate in the present investigation, the biopsy activity index and urinary MCP-1 did correlate positively.

All of these findings line up with Rovin et al.,[25], and Torabinejad et al., [26]

Levels of MCP-1 were significantly correlated with histological classes of active LN, with levels rising in proliferative classes IV and III, respectively, in the present investigation.

Perpendicular to it Mohammed et al.,[27] and Ghobrial et al.,[28] correlated well with histological characteristics and showed that MCP-1 concentrations were substantially greater in diffuse proliferative LN.

According to the current study, Urinary MCP-1 could be a good way to tell active LN from non-active LN by calculating an area under the curve and then setting a cutoff point. The test's specificity was 83.6% and sensitivity was 95% when the cutoff point was ≥0.42 mg/dl, distinguishing between the two groups. A ROC area of 0.93 was calculated.

4. Conclusion

There is a strong association between the clinical and laboratory parameters of the activity investigated and the urinary MCP-1 levels, which reveal a large increase in cases of LN activity. uMCP-1 could differentiate between renal disease that was inactive and active LN and/or recurrence. It reliably detected LN activity and relapse with high sensitivity and specificity, making it an excellent diagnostic tool.

In this study, UMCP-1 showed a strong correlation with pathological class, activity index, and 24-hour urine protein. Unfortunately, uMCP-1 levels cannot be utilized to monitor therapy response since they are still not a reliable indicator of renal activity when used alone.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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