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Infection Patterns in Systemic Lupus Erythematosus and Their Association With Anti-dsDNA Antibodies, C-reactive Protein (CRP), and Erythrocyte Sedimentation Rate (ESR): A Cross-Sectional Study

Savitha Abraham [1], Gayathri Prathap [2], Amritha Srikantan [3], Adarsh Srikantan [4], Srikantan S. Pillai V [2]

1. Internal Medicine, Government Medical College, Thiruvananthapuram, IND 2. Internal Medicine, Sree Uthradom Thirunal Academy of Medical Sciences, Thiruvananthapuram, IND 3. Radiology, Sree Uthradom Thirunal Academy of Medical Sciences, Thiruvananthapuram, IND 4. Critical Care Medicine, Bharati Vidyapeeth (Deemed to be University), Pune, IND

Corresponding author: Srikantan S. Pillai V, srikantansreedharan@gmail.com

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Abstract

Introduction: Infections are a major cause of morbidity and mortality in patients with systemic lupus erythematosus (SLE), particularly in the setting of immunosuppressive therapy. Distinguishing infection from disease flare can be challenging because of overlapping clinical features. This study evaluated the pattern of infections in SLE and their association with anti-double-stranded DNA (dsDNA), C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR).

Methods: A hospital-based cross-sectional study was conducted from June 2024 to June 2025 at a tertiary care center in Kerala, India. A total of 130 patients with SLE aged ⩾13 years were clinically evaluated for infections. The associations between infection and dsDNA, CRP, and ESR were analyzed using the chi-square test.

Results: The study population was predominantly female (99%), with 83% residing in rural areas. Respiratory and urinary tract infections were the most frequently identified infections. Elevated ESR was observed in 92% of patients but showed no significant association with infection. Similarly, dsDNA levels were not significantly associated with infection. In contrast, CRP demonstrated a significant association with infection, suggesting its potential role in differentiating infection from SLE disease activity. Escherichia coli and Acinetobacter were among the notable organisms isolated.

Conclusion: Infections represent a substantial clinical burden in SLE. CRP may be a useful marker for identifying infection and distinguishing it from disease flare, whereas ESR and dsDNA appear less reliable for this purpose.

Categories: Rheumatology, Internal Medicine, Infectious Disease
Keywords: anti-dsdna, c-reactive protein, erythrocyte sedimentation rate, infection, systemic lupus erythematosus

Introduction

Systemic lupus erythematosus (SLE) is a chronic, heterogeneous, multisystem autoimmune disease characterized by immune dysregulation, autoantibody production, and immune-complex-mediated tissue injury [1]. Its clinical spectrum ranges from relatively mild mucocutaneous and constitutional manifestations to potentially life-threatening involvement of the kidneys, nervous system, hematological system, and other vital organs. The disease predominantly affects women, with hormonal influences, X-chromosome-related mechanisms, genetic susceptibility, and environmental triggers collectively contributing to its marked sex disparity [2].

The pathogenesis of SLE involves a complex interaction between genetic predisposition, hormonal factors, environmental exposures, and abnormalities in both innate and adaptive immunity [3]. Autoantibodies, particularly antinuclear antibodies (ANA) and anti-double-stranded DNA (anti-dsDNA) antibodies, play a central role in disease pathogenesis and are important components of contemporary classification and diagnostic assessment. The 2019 European Alliance of Associations for Rheumatology (EULAR)/American College of Rheumatology (ACR) classification criteria have further refined the identification and classification of SLE by incorporating weighted clinical and immunological domains [4].

Despite substantial advances in diagnosis and treatment, SLE remains a major clinical challenge because of its unpredictable course, diverse organ involvement, and susceptibility to complications [5]. Among these, infection represents a particularly important and potentially preventable cause of morbidity and mortality

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[6]. Patients with SLE are vulnerable to infections because of disease-related immune dysfunction as well as the cumulative effects of corticosteroids and other immunosuppressive therapies [7]. Infection and disease activity may present with overlapping clinical features, making early differentiation challenging and potentially delaying appropriate treatment.

The burden of infection in SLE is influenced by disease activity, organ involvement, treatment intensity, and host-related factors [8]. The relationship between infection and commonly used inflammatory and serological markers remains complex. Anti-dsDNA antibodies are associated with disease activity in many patients, while inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) may provide additional, albeit imperfect, information. Interpreting these markers in the setting of infection can be clinically challenging, as changes may reflect active lupus, superimposed infection, or both.

Several studies have evaluated laboratory markers for this purpose. Kim et al. found CRP to be a more sensitive and specific marker of bacterial infection in SLE than procalcitonin or S100A8/A9 [9]. Littlejohn et al. and Abdel-Magied et al. reported that the ESR/CRP ratio, procalcitonin, and the neutrophil-to-lymphocyte ratio help distinguish infection from flare in febrile patients with SLE [10,11], and Amarnani et al. examined features discriminating flare from infection in a multicenter hospital system [8]. Most of these studies used quantitative or high-sensitivity CRP and markers such as procalcitonin, which are not uniformly available in resource-limited public hospitals, and few were conducted in South Asia. Indian studies have described the incidence, spectrum, and risk factors of infection in SLE [12,13] but have not examined how routinely available markers behave in relation to infection.

Data from Kerala are particularly relevant. The regional infectious disease profile includes endemic tropical infections such as leptospirosis, and investigations available in government tertiary care centers are often limited to low-cost tests; at our center, CRP is reported qualitatively. Describing the infection spectrum and the behaviour of ESR, qualitative CRP, and anti-dsDNA in this setting may therefore inform practice in comparable resource-limited settings.

The primary objective of this study was to determine the proportion and pattern of infections among hospitalized patients with SLE at a tertiary care center in Kerala. The secondary objective was to evaluate the association of infection status with anti-dsDNA, CRP, and ESR. The study assessed associations between infection and these markers; it was not designed to evaluate their accuracy in distinguishing infection from disease flare.

Materials And Methods

This hospital-based cross-sectional study was conducted from June 2024 to June 2025 in the Department of Internal Medicine, Government Medical College, Thiruvananthapuram, Kerala, after approval from the Institutional Review Committee and the Institutional Human Ethics Committee (HEC No. 08/02/2024/MCT), and is reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [14].

Consecutive patients aged ⩾13 years admitted with SLE fulfilling the 2019 EULAR/ACR classification criteria [4] were enrolled, regardless of the reason for admission, after written informed consent; no patient younger than 18 years was enrolled. Patients with primary immunodeficiency, uncontrolled diabetes mellitus (HbA1c >8%, study-defined), chronic kidney disease on dialysis, HIV infection, malignancy, or current chemotherapy or radiotherapy were excluded. The sample size of 130 was calculated for the prevalence estimate (urinary tract infection (UTI) prevalence 31.48%, 95% confidence, 8% absolute precision) and was not powered for the association analyses.

A structured proforma recorded demographics, comorbidities, previous hospitalization and disease flare, presenting symptoms, the infection diagnosis, laboratory results, and treatment requirements (intravenous antibiotics, oxygen supplementation, mechanical ventilation, and surgical intervention). Anaemia was defined as haemoglobin <12 g/dL in women and <13 g/dL in men (World Health Organization criteria adopted in Indian national guidelines) [15], and thrombocytopenia as a platelet count <150,000/µL. No patient had received antibiotics before admission, as ascertained by the routine admission questionnaire, and blood samples were drawn at admission, before antimicrobial therapy.

CRP was measured by slide latex agglutination (CRP Latex, Agappe Diagnostics Ltd., Ernakulam, India; analytical sensitivity ~6 mg/L) and recorded qualitatively (elevated, >6 mg/L), although the kit permits semiquantitative estimation. Anti-dsDNA IgG was measured by fluorescence enzyme immunoassay (FEIA) (EliA dsDNA, Phadia AB, Thermo Fisher Scientific, Uppsala, Sweden); values >15 IU/mL were positive, and equivocal values (10-15 IU/mL) were classified as negative. ESR was measured by an automated analyser and considered elevated above age/2 mm/h in men and (age + 10)/2 mm/h in women. Cultures were sent when clinically indicated, at the treating physician's discretion and according to the suspected site of infection, and processed by standard methods [16].

Infection required compatible clinical features with microbiological confirmation and/or response to

antimicrobial therapy; when cultures were negative or unavailable, the diagnosis rested on clinical findings and treatment response. Site-specific criteria were as follows: upper respiratory tract infection, sore throat, rhinorrhoea, or cough with fever without lower respiratory signs; lower respiratory tract infection, cough, fever, or dyspnoea with focal chest signs and/or new radiographic infiltrates; UTI, urinary symptoms with pyuria, with or without significant bacteriuria; skin and soft-tissue infections (cellulitis, furuncle, abscess, infected ulcer), characteristic clinical findings, with pus culture where available; superficial fungal infections (tinea corporis, pityriasis versicolor), clinical appearance confirmed by potassium hydroxide (KOH) mount; and other infections (leptospirosis, tubercular lymphadenitis, spontaneous bacterial peritonitis, acute gastroenteritis), standard clinical and laboratory criteria. Infection status was determined by the attending senior physician, using clinical features, vital signs, and the total leukocyte count, and was recorded on the proforma before CRP results were available. Vital signs and leukocyte counts were recorded categorically and were not analysed, and disease activity was not assessed with a standardized index.

Data were analysed using IBM SPSS Statistics version 27 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as frequencies and percentages, with 95% confidence intervals (CI) for infection proportions. Associations of infection status with laboratory markers and clinical characteristics were tested using Pearson's chi-square test or, when expected cell counts were below five, two-sided Fisher's exact test. For CRP, sensitivity, specificity, positive and negative predictive values (PPV, NPV), and the odds ratio with 95% CI (Woolf method) were calculated, and a sensitivity analysis excluded patients with chronic or superficial conditions (dental caries, tinea corporis, pityriasis versicolor). All analyses were univariable, there were no missing data, and $p < 0.05$ was considered statistically significant.

Results

Patient demographics

Among the 130 patients with SLE, the majority were aged 25-50 years (93, 71.5%), followed by those aged <25 years (27, 20.8%) and >50 years (10, 7.7%). There was a marked female predominance, with 129 (99.2%) females and one (0.8%) male, as presented in Table *1*.

Characteristic	Category	n	%
Age (years)	<25	27	20.8
	25-50	93	71.5
	>50	10	7.7
Sex	Female	129	99.2
	Male	1	0.8
Total		130	100.0

TABLE 1: Demographic Characteristics of Patients With Systemic Lupus Erythematosus (N = 130)

Distribution of the study population by age group and sex.

Pattern and proportion of infections

Among the 130 patients with SLE, 54 (41.5%) had an infection at admission, while 76 (58.5%) had no infection. The most frequent infections were upper respiratory tract infection (URTI), UTI, and lower respiratory tract infection (LRTI), each affecting 10 patients (7.7%). These were followed by tinea corporis (7, 5.4%) and infected ulcers (4, 3.1%). Dental caries and pityriasis versicolor occurred in two patients each (1.5%). Gluteal abscess, bronchitis, leptospirosis, tubercular lymphadenitis, spontaneous bacterial peritonitis, cellulitis, pyrexia of unknown origin, furuncle, and acute gastroenteritis were each reported in one patient (0.8%), as presented in Table *2*.

Diagnosis	n (%) (N = 130)	95% CI
No infection	76 (58.5)	49-67
Upper respiratory tract infection	10 (7.7)	4-14
Urinary tract infection	10 (7.7)	4-14
Lower respiratory tract infection	10 (7.7)	4-14
Tinea corporis	7 (5.4)	2.4-11
Infected ulcer	4 (3.1)	0.99-8.2
Dental caries	2 (1.5)	0.27-6
Pityriasis versicolor	2 (1.5)	0.27-6
Gluteal abscess	1 (0.8)	0.04-4.8
Bronchitis	1 (0.8)	0.04-4.8
Leptospirosis	1 (0.8)	0.04-4.8
Tubercular lymphadenitis	1 (0.8)	0.04-4.8
Spontaneous bacterial peritonitis	1 (0.8)	0.04-4.8
Cellulitis	1 (0.8)	0.04-4.8
Pyrexia of unknown origin	1 (0.8)	0.04-4.8
Furuncle	1 (0.8)	0.04-4.8
Acute gastroenteritis	1 (0.8)	0.04-4.8

TABLE 2: Pattern and Proportion of Infections Among Patients With Systemic Lupus Erythematosus (N = 130)

Infections identified at admission, by diagnosis; each patient with infection had a single infection diagnosis.

Microbiological culture results

Microbiological investigations were performed in a subset of patients. Blood cultures were positive in one of 20 samples (5%), yielding Acinetobacter spp. Urine cultures were positive in one of 10 samples (10%), with Escherichia coli isolated. Among 10 sputum cultures, two (20%) showed growth, with Klebsiella spp. and Acinetobacter spp. isolated in one sample each. All 11 sputum samples tested for acid-fast bacilli were negative. Pus cultures showed growth in three of six samples (50%), with Staphylococcus aureus isolated from one sample and Klebsiella spp. from two. The single ascitic fluid culture showed no growth, as presented in Table *3*.

Specimen / Investigation	No. of samples tested	Samples with growth, n (%)	Organisms isolated	No growth / Negative, n (%)
Blood culture	20	1 (5)	Acinetobacter spp. (1)	19 (95)
Urine culture & sensitivity	10	1 (10)	Escherichia coli (1)	9 (90)
Sputum culture & sensitivity	10	2 (20)	Klebsiella spp. (1); Acinetobacter spp. (1)	8 (80)
Sputum AFB	11	0 (0)	None	11 (100)
Pus culture & sensitivity	6	3 (50)	Staphylococcus aureus (1); Klebsiella spp. (2)	3 (50)
Ascitic fluid culture	1	0 (0)	None	1 (100)

TABLE 3: Microbiological Culture Results Among Patients With Systemic Lupus Erythematosus

Results of microbiological investigations, which were performed when clinically indicated, by specimen type. AFB: acid-fast bacilli.

Distribution of ESR, CRP, and anti-dsDNA

ESR was elevated in 119 (91.5%) patients and normal in 11 (8.5%). CRP was elevated in 51 (39.2%) patients and normal in 79 (60.8%). Anti-dsDNA measured by FEIA was elevated in 50 (38.5%) patients and normal in 80 (61.5%), as presented in Table *4*.

Laboratory parameter	Category	n (%)
ESR	Normal	11 (8.5)
	Elevated	119 (91.5)
CRP (latex agglutination)	Normal	79 (60.8)
	Elevated	51 (39.2)
Anti-dsDNA (FEIA)	Normal	80 (61.5)
	Elevated	50 (38.5)

TABLE 4: Distribution of ESR, CRP, and Anti-dsDNA Levels Among Patients With Systemic Lupus Erythematosus (N = 130)

Proportion of patients with normal and elevated erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and anti-dsDNA levels at admission.

Association of infection status with ESR, CRP, and anti-dsDNA

ESR was elevated in 89.5% of patients without infection and 94.4% of those with infection, with no significant association (Fisher's exact test, $p = 0.360$). Elevated CRP was observed in 28.9% of patients without infection compared with 53.7% of those with infection, a statistically significant association ($\chi^2 = 8.12$, $p = 0.004$). Anti-dsDNA was elevated in 35.5% of patients without infection and 42.6% of those with infection, with no significant association ($\chi^2 = 0.67$, $p = 0.414$). Against infection status, elevated CRP had a sensitivity of 53.7% (29/54), specificity of 71.1% (54/76), PPV of 56.9% (29/51), and NPV of 68.4% (54/79). Patients with elevated CRP had higher odds of infection (odds ratio 2.85, 95% CI 1.37-5.90). In a sensitivity analysis excluding the 11 patients with dental caries, tinea corporis, or pityriasis versicolor, elevated CRP was present in 25/43 (58.1%) patients with acute infection compared with 22/76 (28.9%) without infection ($\chi^2 = 9.79$, $p = 0.002$; odds ratio 3.41, 95% CI 1.56-7.46). Four of the 11 excluded patients (36.4%) had elevated CRP. The status of each marker in patients with and without infection is as presented in Table *5*.

Laboratory parameter	Category	Without infection (N = 76), n (%)	With infection (N = 54), n (%)	Test statistic	p-value
ESR	Normal	8 (10.5)	3 (5.6)	Fisher's exact test	0.360
	Elevated	68 (89.5)	51 (94.4)	-	-
CRP (latex agglutination)	Normal	54 (71.1)	25 (46.3)	$\chi^2 = 8.12$	0.004
	Elevated	22 (28.9)	29 (53.7)	-	-
Anti-dsDNA (FEIA)	Normal	49 (64.5)	31 (57.4)	$\chi^2 = 0.67$	0.414
	Elevated	27 (35.5)	23 (42.6)	-	-

TABLE 5: : Association of Infection Status With ESR, CRP, and Anti-dsDNA Levels (N = 130)

Comparison of erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and anti-dsDNA status between patients with and without infection. FEIA: fluorescence enzyme immunoassay.

Clinical characteristics and treatment requirements

Among the 54 patients with infection, 37 (68.5%) had fever, 21 (38.9%) required intravenous antibiotics, six (11.1%) required oxygen supplementation, three (5.6%) required mechanical ventilation, and one (1.9%) required surgical intervention; no patient without infection had fever or required these interventions. Anaemia was more frequent among patients with infection (57.4% vs. 35.5%; $\chi^2 = 6.12$, $p = 0.013$), whereas thrombocytopenia, diabetes mellitus, previous hospitalization, and a history of disease flare did not differ significantly between groups, as presented in Table *6*.

Variable	Without infection (N = 76), n (%)	With infection (N = 54), n (%)	Test	p-value
Diabetes mellitus	7 (9.2)	4 (7.4)	Fisher's exact	1.000
Previous hospitalization	34 (44.7)	26 (48.1)	$\chi^2 = 0.15$	0.701
History of disease flare	31 (40.8)	19 (35.2)	$\chi^2 = 0.42$	0.517
Anaemia	27 (35.5)	31 (57.4)	$\chi^2 = 6.12$	0.013
Thrombocytopenia (<150,000/μL)	25 (32.9)	20 (37.0)	$\chi^2 = 0.24$	0.625
Intravenous antibiotics	0 (0)	21 (38.9)	$\chi^2 = 35.25$	<0.001
Oxygen supplementation	0 (0)	6 (11.1)	Fisher's exact	0.004
Mechanical ventilation	0 (0)	3 (5.6)	Fisher's exact	0.069
Surgical intervention	0 (0)	1 (1.9)	Fisher's exact	0.415

TABLE 6: Clinical Characteristics and Treatment Requirements According to Infection Status (N = 130)

Comorbidities, past history, haematological findings, and treatment requirements in patients with and without infection.

Discussion

In this cross-sectional study of 130 hospitalized patients with SLE, 41.5% had an infection at admission, most often URTI, UTI, and LRTI, which were equally frequent; elevated CRP was associated with infection, whereas ESR and anti-dsDNA were not. Because infection may mimic or precipitate disease activity [8,17], these findings are relevant to the evaluation of unwell patients with SLE.

The female predominance and age distribution are consistent with the known epidemiology of SLE [2]. Infections spanned respiratory, urinary, cutaneous, gastrointestinal, and other bacterial infections [18]. Among Indian studies, Chatterjee et al. found bacterial infections to be predominant, with tuberculosis the most common [12], and Jha et al. reported UTI as the most common infection, with Escherichia coli the most frequent isolate [13], findings broadly consistent with the present study.

Only 7 of 58 cultures were positive. Cultures were sent only when clinically indicated, and many infections in this cohort, such as upper respiratory tract infections (frequently viral), KOH-confirmed superficial fungal infections, and dental caries, do not call for bacterial culture; sterile cultures therefore do not exclude infection, and some misclassification is possible.

ESR was elevated in 91.5% of patients irrespective of infection status, likely reflecting underlying lupus activity, which limits its ability to discriminate infection [10]. Neither ESR nor anti-dsDNA was significantly associated with infection, but this does not establish that they lack clinical value: the study was not powered to detect modest differences, and both remain relevant to assessing disease activity [8]. Yu et al. found haemoglobin, CD4 lymphocyte count, IgM, and anti-Sm antibody to be independently associated with multisystem involvement in hospitalized SLE patients with infection [19].

The CRP association persisted after excluding chronic and superficial conditions (odds ratio 3.41), and because infection status was recorded before CRP results were available and samples preceded antimicrobial therapy, it is unlikely to reflect incorporation bias or antibiotic effect. Discrimination was modest, however (sensitivity 53.7%, specificity 71.1%), so a normal CRP does not exclude infection. These findings agree with Kim et al., who identified CRP as a sensitive and specific marker of bacterial infection in SLE [9], and with studies showing that the ESR/CRP ratio, procalcitonin, and the neutrophil-to-lymphocyte ratio help separate infection from flare [10,11].

Disease activity and treatment may have influenced these results. Lupus activity raises ESR and anti-dsDNA but is often accompanied by only modest CRP elevation [10,11], which may explain why ESR was elevated almost universally while CRP tracked infection more closely. Corticosteroids and other immunosuppressants increase infection risk and can blunt fever and inflammatory responses [7]. Because neither disease activity nor treatment was recorded, the CRP association should be regarded as unadjusted.

Infection carried a substantial clinical burden: 38.9% of patients with infection required intravenous antibiotics and 11.1% required oxygen supplementation, and anaemia was more frequent among them, an observation that warrants further study. CRP may provide supportive information when infection is suspected, but it should be interpreted with clinical, microbiological, and imaging findings rather than alone [16,20].

Strengths of the study include consecutive enrolment, a structured proforma with data recorded at admission, recording of infection status before CRP results were available, and blood sampling before antimicrobial therapy.

This study has several limitations. It was a single-center study of hospitalized patients with a relatively small sample size that was not powered for the association analyses; the findings may not apply to outpatients or to patients on dialysis or with uncontrolled diabetes, who were excluded. Disease activity was not measured, so the study cannot address whether these markers distinguish infection from flare. Most infections were diagnosed clinically, and the infection category combined acute and chronic superficial infections, although the CRP association persisted when the latter were excluded. CRP was recorded qualitatively and measured once. Treatment data were not collected, as they were beyond the scope of this study, so the influence of treatment, a major determinant of infection risk in SLE, could not be assessed, and all analyses were univariable; vital signs and leukocyte counts were not analysed. Larger multicenter studies are needed to validate these findings.

Conclusions

In this cross-sectional study of 130 hospitalized patients with SLE, 41.5% had an infection at admission, most commonly upper and lower respiratory tract and UTIs. ESR was elevated in most patients regardless of infection status, and neither ESR nor anti-dsDNA was associated with infection. Elevated CRP was significantly more frequent among patients with infection, and the association persisted after excluding chronic and superficial conditions, but with a sensitivity of 54% and specificity of 71% it was only a modest discriminator.

CRP may therefore provide supportive information when infection is suspected in a patient with SLE, but it should not be used alone to diagnose or exclude infection. Because disease activity was not assessed, this study cannot determine whether CRP distinguishes infection from lupus flare. Prospective multicenter studies using quantitative CRP and standardized disease activity indices are needed to address this question.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Srikantan S. Pillai V, Gayathri Prathap, Amritha Srikantan, Savitha Abraham, Adarsh

Srikantan

Acquisition, analysis, or interpretation of data: Srikantan S. Pillai V, Gayathri Prathap, Amritha Srikantan, Savitha Abraham, Adarsh Srikantan

Drafting of the manuscript: Srikantan S. Pillai V, Gayathri Prathap, Amritha Srikantan, Savitha Abraham, Adarsh Srikantan

Critical review of the manuscript for important intellectual content: Srikantan S. Pillai V, Gayathri Prathap, Amritha Srikantan, Savitha Abraham, Adarsh Srikantan

Supervision: Srikantan S. Pillai V

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Human Ethics Committee, Medical College, Thiruvananthapuram issued approval 08/02/2024/MCT. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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