EADV 2026: Advances in Diagnosis and Assessment of Lupus Erythematosus

Author:	*Dimitrina Guleva[1] 1. Department of Dermatology, Military Medical Academy, Sofia, Bulgaria *Correspondence to dimiguleva@gmail.com

Conflicts of interest:	The author has declared no conflicts of interest.
Keywords:	Biomarkers, emerging therapies, lupus erythematosus (LE), new scoring systems.
Citation:	EMJ Dermatol. 2026;14[1]:23-26. https://doi.org/10.33590/emjdermatol/T20T7QBI

LUPUS ERYTHEMATOSUS (LE) is an autoimmune connective tissue disease with a wide range of skin (cutaneous lupus erythematosus [CLE]) and/or systemic (systemic lupus erythematosus [SLE]) manifestations. Cutaneous involvement is found in up to 80% of patients with SLE,[1] and CLE is observed as an initial manifestation of SLE in 0–42% of cases.[2] LE skin changes are divided into LE-specific (acute CLE, subacute CLE, chronic CLE) and LE-nonspecific (vascular and non-vascular signs) subtypes.[3] Some of these are more likely to lead to scarring (chronic CLE), but others have a higher risk of progression to SLE (acute CLE, subacute CLE).[4]

NOVEL SCORING SYSTEMS

The standard clinical tool is the Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI), assessing both activity (CLASI-A; 0–70) and damage (CLASI-D; 0–56) in the most affected lesion per area. A key outcome measure in CLE trials is CLASI-A-50, where decreasing ≥50% of the activity lesions is crucial.[5]

The Cutaneous Lupus Activity Investigator's Global Assessment—Revised (CLA-IGA-R) is a new global assessment that evaluates disease activity across all lesions. It rates the erythema (0–4), other morphologic characteristics (OMC; 0–4), and follicular activity (0–1). CLA-IGA-R is mostly used as an endpoint in trials;[6] however, the lack of a body surface area (BSA) score in this newly proposed tool is emerging as a disadvantage.

During the European Academy of Dermatology and Venereology (EADV) Congress 2026, François Chasset introduced a score that detects patients with chronic CLE at risk of systematisation based on sociodemographic, clinical, and biological features, including American College of Rheumatology (ACR)/European Alliance of Associations for Rheumatology (EULAR) classification.[7] He presented their novel, validated, simple three-item score system to measure the risk of progression to severe SLE, including age <25 years (1 point), phototypes V–VI (1 point), and antinuclear antibody (ANA) titres ≥1:320 (5 points).[8] Simple recommendations are built depending on results, with 0–1 points being reassuring and 6–7 points warranting close monitoring. This tool could be very helpful for identifying low-risk patients.

NEW BIOMARKERS

Traditional biomarkers such as ANA, anti-double-stranded DNA, anti-Smith antibodies, complement, and proteinuria continue to be essential. Limitations of late organ involvement, sensitivity and specificity, and limited prediction of lupus flares have led to a search for new

CC BY-NC 4.0 Licence • Copyright © 2026 EMJ

prognostic biomarkers. Novel emerging biomarkers are becoming more organ- and pathway-specific, which may enable more successful treatment options.

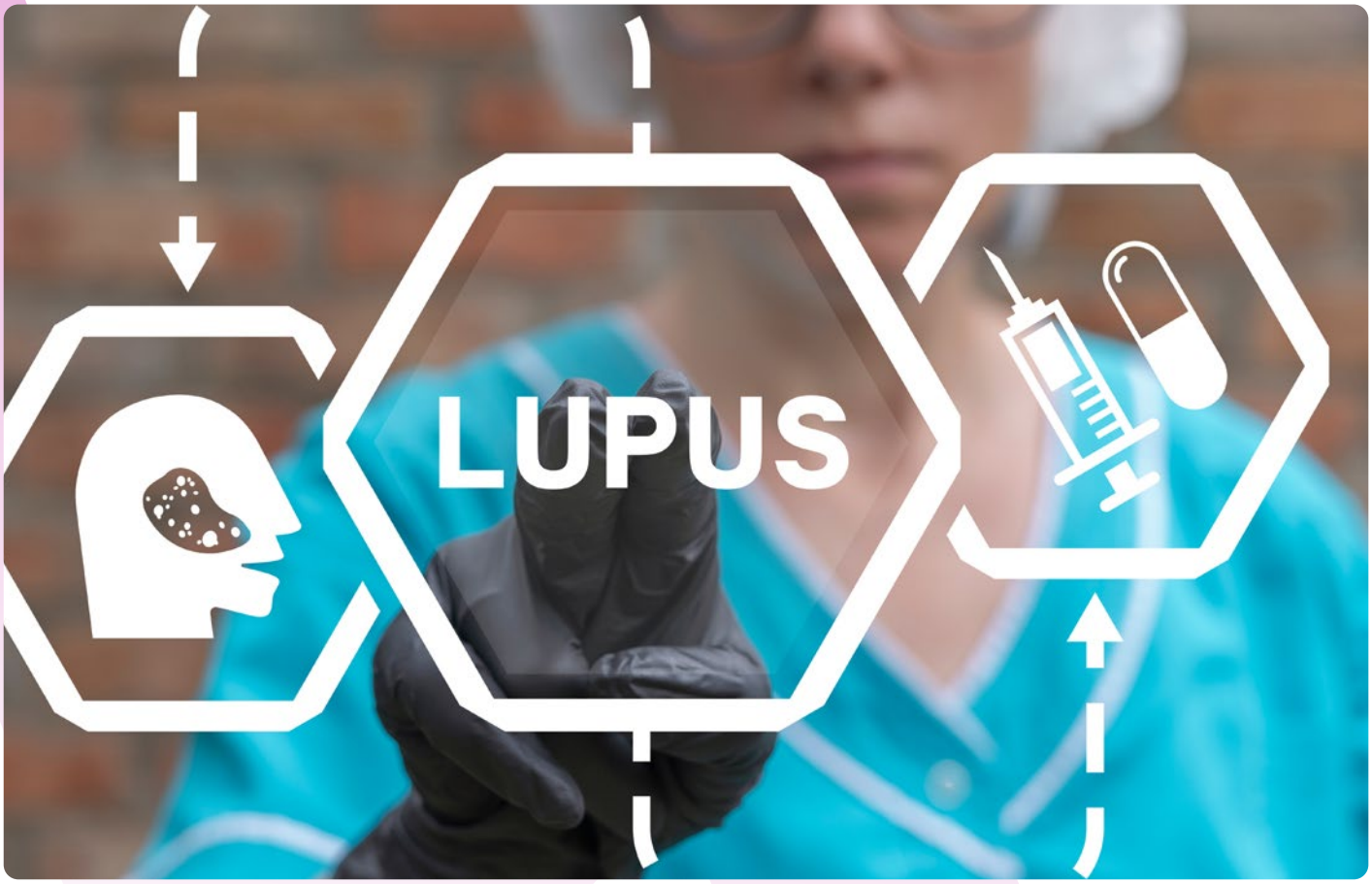
Type I interferon (IFN) signature is well studied in SLE as a potential treatment response biomarker accessible to clinical practice.[9] A monocentric study of patients with CLE confirmed the main role of the Type I IFN pathway, showing a strong correlation with disease activity and flare risk.[10]

New literature data suggest that the Type I IFN signature may promote tissue-resident memory cell-related features in CLE, representing their existence in lesional lupus skin and their role in local immune memory for possible recurrences.[11]

'Liquid' biopsy, which helps measure disease activity and potential flares in patients with lupus nephritis via soluble urinary biomarkers, was a hot topic. One example is urinary soluble CD163, characterised by a strong association with histological activity, treatment response, new active disease detection, and flare prediction.[12] It is indicative of active disease, but not of chronic disease, and it could easily recognise those patients who need systemic immunosuppressant therapy.

Most important is the skin as a tissue biomarker. Generalised discoid lesions, LE-nonspecific lesions (vascular; periungual telangiectasia), and serological abnormalities (ANA, anti-double-stranded DNA, low C3/C4, anti-Smith, and pancytopenia) represent prognostic factors. However, the IFN-I gene signature locally in the skin is the main and most promising biomarker, whereas MxA, GBP1, and CXCP 10 biomarkers have a supportive function in monitoring disease activity.[13]

Furthermore, skin biomarkers have a role in predicting antimalarial response in CLE. Patients with a high IFN-I signature are more likely to be associated with good hydroxychloroquine response, while the occurrence of increased myeloid dendritic cells and TNF-α expression predicts poor response to hydroxychloroquine.[14]



Future directions are multi-omics (integration of transcriptomics and proteomics), which are new candidate biomarkers for subacute cutaneous lupus erythematosus.[15]

NEW TREATMENT MODALITIES

Conventional therapies have been used for years. The algorithm proposed by Chasset et al.[16] includes potential CLE mimickers among patients with CLE and SLE, general prevention, and the most suitable treatment steps.

Novel emerging modalities are under discussion, and most of them are in ongoing Phase II trials for CLE: Type I IFN pathway (anifrolumab), pDC-directed therapies, TLR7/8 (enpatoran), JAK/STAT inhibitors, and TYK2 inhibitors.[17]

The most promising and advanced clinical development is anifrolumab, which is currently approved only for SLE, but is used off-label in refractory and difficult-to-treat CLE lesions.[18]

CONCLUSION

Novel diagnostic and treatment strategies are coming, but we should be alert in all CLE lesions refractory to therapy despite negative immunological parameters for underlying overlap syndromes.



CC BY-NC 4.0 Licence • Copyright © 2026 EMJ

References

1. Cervera R et al. Systemic lupus erythematosus: clinical and immunologic patterns of disease expression in a cohort of 1000 patients. The European working party on systemic lupus erythematosus. Medicine (Baltimore). 1993;72(2):113-24.
2. Curtiss P et al. A systematic review of the progression of cutaneous lupus to systemic lupus erythematosus. Front Immunol. 2022;13:866319.
3. Gilliam JN, Sontheimer RD. Distinctive cutaneous subsets in the spectrum of lupus erythematosus. J Am Acad Dermatol. 1981;4(4):471-5.
4. Biazar C et al. Cutaneous lupus erythematosus: first multicenter database analysis of 1002 patients from the European Society of Cutaneous Lupus Erythematosus (EUSCLE). Autoimmun Rev. 2013;12(3):444-54.
5. Albrecht J et al. The CLASI (Cutaneous Lupus Erythematosus Disease Area and Severity Index): an outcome instrument for cutaneous lupus erythematosus. J Invest Dermatol. 2005;125(5):889-94.
6. Elman SA et al. Correlation between CLASI and CLA-IGA-R among patients with CLE in a multi-center cross-sectional study in the US. Poster P0305. EADV Congress, 30 September-3 October, 2026.
7. Fredeau L et al. Risk factors of progression from discoid lupus to severe systemic lupus erythematosus: a registry-based cohort study of 164 patients. J Am Acad Dermatol. 2023;88(3):551-9.
8. Masseran C et al. Comparison of patients with isolated cutaneous lupus erythematosus versus systemic lupus erythematosus with cutaneous lupus erythematosus as the sole clinical feature: a monocentric study of 149 patients. J Am Acad Dermatol. 2024;90(6):1210-17.
9. Vital EM et al. Anifrolumab efficacy and safety by type I interferon gene signature and clinical subgroups in patients with SLE: post hoc analysis of pooled data from two phase III trials. Ann Rheum Dis. 2022;81(7):951-61.
10. Murat de Montai Q et al. Interferon-α biological activity is associated with disease activity and risk of flare in cutaneous lupus erythematosus: a monocentric study of 184 patients. J Am Acad Dermatol. 2025;92(5):1039-48.
11. Gu HJ et al. Expression pattern of tissue-resident memory T cells in CLE. Lupus. 2021;30(9):1427-37.
12. Renaudineau Y et al. Urinary soluble CD163 is useful as "liquid biopsy" marker in lupus nephritis at both diagnosis and follow-up to predict impending flares. J Transl Autoimmun. 2024;9:100244.
13. Lambers WM et al. Myxovirus resistance protein a is a useful additional histological marker in suspected cutaneous lupus erythematosus. Acta Derm Venereol. 2020;100:adv00234.
14. Zeidi M et al. Increased myeloid dendritic cells and TNF-α expression predicts poor response to hydroxychloroquine in cutaneous lupus erythematosus. J Invest Dermatol. 2019;139(2):324-32.
15. Tao Y et al. Verification of biological markers of subacute cutaneous lupus erythematosus via TMT labelling proteomics combined with transcriptome data. Ann Med. 2025;57(1):2500696.
16. Chasset F, Teboul A. Update on cutaneous lupus erythematosus pathogenesis, diagnosis and management. J Eur Acad Dermatol Venereol. 2026;40(5):782-800.
17. Falla Rivera PE et al. Emerging targeted therapies in cutaneous lupus erythematosus: a scoping review of evidence from 2014 to 2026. Poster P1690. EADV Congress, 30 September-3 October, 2026.
18. Blum FR et al. Anifrolumab for treatment of refractory cutaneous lupus erythematosus. Clin Exp Dermatol. 2022;47(11):1998-2001.