

SLE-DAS: ready for routine use?

It is with great interest that we read your recently published article, 'Derivation and validation of the SLE disease activity score (SLE-DAS)'.¹ Systemic lupus erythematosus (SLE) is a multisystem disease with protean clinical manifestations. The disease runs a variable course, with no reliable co-relation between various organ involvement and biochemical markers. There have been numerous attempts to document diseases activity in an objective manner, with only limited success.² In this article, the authors' effort to translate the disease activity in SLE into a more responsive score by quantifying arthritis, proteinuria, leucopenia and thrombocytopenia seems promising. However, we would like to raise a few points of concern.

1. The median physician global assessment (PGA) and systemic lupus erythematosus disease activity index 2000 (SLEDAI-2K) scores in both the derivation and validation cohorts suggest that they were of low disease activity group; hence, the validity of the score in a more active disease group may not be the same.^{3,4}
2. Is proteinuria alone sufficient to assess renal disease activity?
 - a. Renal manifestations, such as the presence of urinary RBCs, WBCs and cellular casts, are not factored into the score, even though their presence has got a significant prognostic role and is a part of SLEDAI-2K.
 - b. Proteinuria may not always indicate disease activity as it can also be a result of irreversible scarring in the kidney. Hence, we believe proteinuria in the presence of active sediments is of higher specificity for disease activity.
3. Ongoing requirement of glucocorticoids is another important factor that could have been given weightage, as it indirectly reflects the physicians' perspective of activity, and may also alter with magnitude of organ involvement.
4. Although rare, severe manifestations, such as lupus pancreatitis, need consideration as they reflect severe disease.

We tried to address the concern of performance of SLE disease activity score (DAS) in a cohort of lupus nephritis with high disease activity. In the same analysis, we tried to look into the effect of non-inclusion of urinary sediment in SLE-DAS. Patient characteristics and disease activity measures are presented in [table 1](#) and [table 2](#) respectively.

We retrospectively calculated SLE-DAS for 41 patients of lupus nephritis at baseline, 3 months and 6 months.

To assess the validity of SLE-DAS in our population, we compared the global scores of SLE-DAS with PGA and SLEDAI using Spearman's correlation. At baseline, SLE-DAS score showed moderate positive correlation with SLEDAI-2K ($r=0.70$, $p<0.001$) and a weak positive correlation with PGA ($r=0.32$, $p<0.05$). At 6 months follow-up, SLE-DAS score showed strong positive correlation with SLEDAI-2K ($r=0.92$, $p<0.001$) and a weak positive correlation with PGA ($r=0.35$, $p<0.05$). This may imply that SLE-DAS has got a better correlation with SLEDAI-2K when the disease activity is low and its performance among patients of high disease activity may not be robust.

Renal component of SLE-DAS was calculated and the effect size was determined for renal component of SLEDAI-2K and

Table 2 Disease activity scores of patients included (N=41)


Disease activity score	Baseline	At 3 months	At 6 months
PGA, median (IQR)	2.3 (2.1–2.6)	1.2 (0.8–1.9)	1.0 (0.0–1.0)
SLEDAI-2K, median (IQR)	23.0 (18.0–28.5)	6.0 (3.0–8.0)	2.0 (2.0–6.0)
SLE-DAS, median (IQR)	28.14 (21.81–38.98)	7.37 (1.32–9.84)	2.08 (0.74–8.39)
SLE-DAS renal, median (IQR)	10.15 (7.73–12.07)	6.53 (0–8.43)	0 (0–6.70)
SLEDAI-2K renal, median (IQR)	12.0 (8.0–12.0)	4.0 (0.0–4.0)	0.0 (0.0–4.0)

SLE-DAS, systemic lupus erythematosus disease activity score.

SLE-DAS using standardised test [$r=Z/\sqrt{n_1+n_2}$]. Effect sizes were interpreted using Cohen's criteria as large (≥ 0.5), medium (≥ 0.3) or small (≥ 0.1).⁵ Among our patients, renal SLE-DAS significantly reduced from median (IQR) 10.15 (7.73–12.07) at baseline to 0 (0–6.70) at 6 months. The effect size for the renal component of both SLE-DAS and SLEDAI-2K were large at -0.572 and -0.602 , respectively ($p=0.18$), showing good sensitivity for change.

We had a single patient with persistent proteinuria with no active urinary sediments at the end of the induction phase for proliferative lupus nephritis. The patient had normal complements, low anti-dsDNA titer and her baseline renal biopsy showed increased chronicity score. Her renal component of SLE-DAS score was 13.19 at baseline and 10.17 due to contribution from proteinuria at 6 months whereas her renal component of SLEDAI-2K had fallen from 12 to 4. Though the observation was in a single patient, the fact remains that proteinuria alone is not specific for active disease. Using SLE-DAS in such a scenario would portray a falsely high disease activity and a word of caution is necessary.

Though SLE-DAS is a brave and innovative attempt in quantifying SLE disease activity, there may be no added advantage over the existing SLEDAI-2K score in its current state. Its performance in high disease activity and in those without proteinuria but with active sediments in the urine will have to be reconfirmed.

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Table 1 Patient characteristics (N=41)

Gender, female, n (%)	39 (95.12)
Age (years), mean (SD)	30 (8.4)
Disease duration (months), median (IQR)	15 (3.5–32.0)

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REFERENCES

- 1 Jesus D, Matos A, Henriques C, *et al.* Derivation and validation of the SLE disease activity score (SLE-DAS): a new SLE continuous measure with high sensitivity for changes in disease activity. *Ann Rheum Dis* 2019;**78**:365–71.
- 2 Mikdashi J, Nived O. Measuring disease activity in adults with systemic lupus erythematosus: the challenges of administrative burden and responsiveness to patient concerns in clinical research. *Arthritis Res Ther* 2015;**17**.
- 3 Petri M, Genovese M, Engle E, *et al.* Definition, incidence, and clinical description of flare in systemic lupus erythematosus. A prospective cohort study. *Arthritis Rheum* 1991;**34**:937–44.
- 4 Gladman DD, Ibañez D, Urowitz MB. Systemic lupus erythematosus disease activity index 2000. *J Rheumatol* 2002;**29**:288–91.
- 5 Cohen J, Analysis SP. Statistical power analysis. *Curr Dir Psychol Sci* 1992;**1**:98–101.