RHEUMATOLOGY

Letter to the Editor (matters arising from published papers)

doi:10.1093/rheumatology/kex314

Comment on: Auto-antibodies to double-stranded DNA as biomarker in SLE: comparison of different assays during quiescent and active disease: reply

Sir, We appreciated reading the letter of Piga *et al.* [1] in response to our recent report concerning the detection of autoantibodies directed to anti-dsDNA in SLE [2]. In this letter, they discuss their own results of serial measurements of anti-dsDNA using RIA by the Farr assay [3]. Comparable findings were obtained regarding the number of patients who had an increased anti-dsDNA >20% level in the 6 months prior to flare occurrence. Moreover, they demonstrated that intensification in immunosuppressive treatment could be effective in preventing flares in patients who experienced an increase in anti-dsDNA level ⩾50% without any symptoms (n = 64), as none of the patients who were treated (n = 15) had a flare, but 32.6% (16/49) of the non-treated patients did experienced a flare.

So both our studies confirm the fact that monitoring anti-dsDNA levels is important in the follow-up of SLE patients. Their main concern is that by using enzyme-labelled anti-isotype assay (EliA), which has a high specificity but a lower sensitivity in quiescent SLE, early changes in anti-dsDNA levels are missed. However, when comparing EliA and the Farr assay, as we did in our study, the same percentages of patients with an increase of anti-dsDNA before an exacerbation were found. Also when we apply this at ⩾50% threshold instead of a ⩾20% increase, only two patients would have been missed using EliA, but also by using Farr. So, even though EliA is less sensitive in quiescent disease, an increase in anti-dsDNA levels probably will be detected by using this technique. As Piga *et al.* stated, a relative increase is much more important than absolute values, as steadily increased anti-dsDNA do not predict a flare. Of course, prospective studies using EliA should be performed to confirm these findings.

Funding: No specific funding was received from any bodies in the public, commercial or not-for-profit sectors to carry out the work described in this manuscript.

Disclosure statement: The authors have declared no conflicts of interest.

Karina de Leeuw[1], Laura Bungener[2], Caroline Roozendaal[2], Hendrika Bootsma[1] and Coen A. Stegeman[3]

[1]*Department of Rheumatology and Clinical Immunology,* [2]*Department of Laboratory Medicine and* [3]*Department of Nephrology, University Medical Center Groningen, Groningen, the Netherlands*

Accepted 10 July 2017

Correspondence to: Karina de Leeuw, Department of Rheumatology and Clinical Immunology, University Medical Center Groningen, Hanzeplein 1, 9713 GZ, Groningen, The Netherlands. E-mail: k.de.leeuw@umcg.nl

References

1 Piga M, Floris A, Mathieu A, Cauli A. Comment on: Auto-antibodies to double-stranded DNA as biomarker in systemic lupus erythematosus: comparison of different assays during quiescent and active disease. Rheumatology 2017; doi: 10.1093/rheumatology/kex313.

2 de Leeuw K, Bungener L, Roozendaal C, Bootsma H, Stegeman CA. Auto-antibodies to double-stranded DNA as biomarker in systemic lupus erythematosus: comparison of different assays during quiescent and active disease. Rheumatology 2017;56:698–703.

3 Floris A, Piga M, Cauli A, Mathieu A. Predictors of flares in systemic lupus erythematosus: Preventive therapeutic intervention based on serial anti-dsDNA antibodies assessment. Analysis of a monocentric cohort and literature review. Autoimmun Rev 2016;15:656–63.

LETTER