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LETTER TO THE EDITOR

Autoantibody phenotyping of antinuclear antibody-negative systemic lupus erythematosus patients

Adrian Lee

[1]*Department of Clinical Immunology & Allergy, Westmead Hospital, Westmead, Australia*
[2]*Department of Immunopathology, Icpmr & Westmead Hospital, Westmead, Australia*
[3]*Centre for Immunology & Allergy Research, Westmead Institute for Medical Research, The University of Sydney, Westmead, Australia*

Dear Editor,

I have read Li et al.'s[1] interesting article on their antinuclear antibody (ANA)-negative cohort of systemic lupus erythematosus (SLE) patients. I have a few comments to make on their study.

Firstly, it was interesting to see the profound thrombocytopenia in the ANA-negative SLE cohort. This cohort may, indeed, be related (or equivalent) to the recently-identified ANA-positive immune thrombocytopenia (ITP) subset which has a higher chance of association with or progression to SLE and other connective tissue diseases over the ANA-negative ITP.[2] In this study, ITP patients were deemed as ANA-positive, if they had a HEp-2 titer of >1:100.[2] Therefore, it would be worthwhile to see what proportion of Li et al.'s[1] study's ANA-negative patients actually had a positive ANA titer at 1:100 assuming that they also screened all patients at this titer. There is no doubt that the generous definition of ANA-negative at a cut-off of 1:320 would have introduced some selection bias.

Additionally, it would have been desirable to see the specific ANA profiles of these patients. The ANA indirect immunofluorescence (IIF) is a screening assay and the presence of specific ANAs-particularly those associated with SLE-in the presence of a negative ANA IIF makes this diagnostically helpful. Modern immunoassays detecting specific ANAs are usually quite sensitive analytically. For instance, about 6% of ANA-negative SLE patients have anti-Sm detected[3]-an immunologic criterion of the SLE International Collaborating Clinics (SLICC) criteria. Anti-Ro60 and anti-Ro52 autoantibodies have also been associated with ITP and SLE/ITP,[2] and about 10% of patients with a low-level anti-Ro60 IgG may be negative on ANA IIF (screened 1:80) even with the sensitive HEp-2000 IIF substrate (ImmunoConcepts) with hyper-expressed Ro60 antigen.[4] Thus, the detection of specific autoantibodies may assist with diagnosis and potentially subtyping of SLE.[5]

In conclusion, additional details and immunophenotyping of the ANA-negative cohort may prove useful in understanding these patients clinically.

Correspondence: Adrian Lee, MD.
E-mail: adrian.lee1@health.nsw.gov.au

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