'Neuropsychiatric lupus or not? Cerebral hypoperfusion by perfusion-weighted MRI in normal-appearing white matter in primary neuropsychiatric lupus erythematosus' by Papadaki *et al*

I read with interest the article by Papadaki *et al*¹ relating to the role of cerebral hypoperfusion in neuropsychiatric lupus. While I applaud the quality of work and the intricate interpretation of enhanced MR imaging, the authors came to the wrong conclusions.

First of all, the definitions (of which I was a co-author) for evaluating a diverse group of patients as having central nervous system systemic lupus erythematosus (SLE) are outdated and not evidence based.² The best objective measure for a central nervous system inflammatory process is a lumbar puncture. Pleocytosis, increased protein levels, increased IgG synthesis rates, oligoclonal bands or antineuronal antibodies are the only objective metrics available to make this diagnosis outside of obvious neuroimaging abnormalities or a brain biopsy. None of the patients in the paper was reported to have a spinal tap.

Second, the publication was not the first to evaluate the role of hypoperfusion in neuroimaging in neuropsychiatric lupus as claimed. The authors correctly note that vasospasm in the watershed regions plays a vasomotor role in other disorders (see refs, 39–41) but apparently were unaware of previous work in this area germane to lupus.

Our group clearly demonstrated that the majority of lupus patients with neuropsychiatric lupus had single photon emission computerized tomography (SPECT) imaging abnormalities consistent with hypoperfusion in the watershed regions.³ In other words, most patients had vasomotor instability on an autonomic basis ('Raynaud's of the brain') in, for example, the frontal-parietal interface where the vasculature is very small, numerous and prone to spasm. While some of these patients have an inflammatory process, the majority develop 'lupus fog' as a consequence of intermittent hypoperfusion. This should not be considered to be neuropsychiatric lupus and is managed

with cognitive behavioural therapy, anxiety reduction measures, biofeedback and approaches that target the dysautonomia of lupus.⁴

Daniel J Wallace

Correspondence to Dr Daniel J Wallace, Division of Rheumatology, Cedars-Sinai Medical Center, Los Angeles, CA 90048, USA; danielwallac@gmail.com

Handling editor Josef S Smolen

Funding This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Provenance and peer review Not commissioned; internally peer reviewed.

© Article author(s) (or their employer(s) unless otherwise stated in the text of the article) 2019. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

To cite Wallace DJ. *Ann Rheum Dis* 2019;**78**:e5.

Received 2 January 2018

Accepted 2 January 2018

Published Online First 17 January 2018

► <http://dx.doi.org/10.1136/annrheumdis-2018-212949>

Ann Rheum Dis 2019;**78**:e5. doi:10.1136/annrheumdis-2018-212937

REFERENCES

- 1 Papadaki E, Fanouriakis A, Kavroulakis E, *et al*. Neuropsychiatric lupus or not? Cerebral hypoperfusion by perfusion-weighted MRI in normal-appearing white matter in primary neuropsychiatric lupus erythematosus. *Ann Rheum Dis* 2018;**77**:441–8.
- 2 Anon. The American College of Rheumatology nomenclature and case definitions for neuropsychiatric lupus syndromes. *Arthritis Rheum* 1999;**42**:599–608.
- 3 Driver CB, Wallace DJ, Lee JC, *et al*. Clinical validation of the watershed sign as a marker for neuropsychiatric systemic lupus erythematosus. *Arthritis Rheum* 2008;**59**:332–7.
- 4 Stojanovich L. Autonomic dysfunction in autoimmune rheumatic disease. *Autoimmun Rev* 2009;**8**:569–72.