'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for evaluating a diverse group of patients as having central nervous system systemic lupus erythematosus (SLE) are outdated and not evidence based.[2] The best objective measure for a central nervous system inflammatory process is a lumbar puncture.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]

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