Neuropsychiatric Manifestations of Systemic Lupus Erythematosus

To the Editor:

Neuropsychiatric manifestations of systemic lupus erythematosus (NPSLE) are responsible for high rates of morbidity and mortality. Zirkzee, *et al*[1] emphasize the importance of ascertaining the different NPSLE phenotypes, which could be helpful in making potential therapeutic decisions. However, it is evident that there were patients who had both profiles described by the authors, that is, inflammatory and ischemic. Because of such overlapping of severe NPSLE manifestations, we consider that physicians must keep in mind starting therapy for both patterns while waiting to define the pathogenesis of the neurological expression. It is evident that high disease activity represents one of the most important risk factors for developing NPSLE, as obtained using the Systemic Lupus Erythematosus Disease Activity Index, and it includes NPSLE manifestations as the main factor. We consider that the study by Zirkzee, *et al* could have incorporated other biomarkers suggested by some algorithms, and lumbar puncture has been suggested as a diagnostic approach[2] that could help in excluding other involved factors besides SLE activity, such as infections. Recently, it has been reported that positron emission tomography scanning is more sensitive than magnetic resonance imaging (MRI)[3]; although Zirkzee, *et al* reported 100% sensitivity of MRI, this sensitivity is lower when there is no focal neurological deficit related to NPSLE, as described in some cases of small-vessel central nervous system angiitis, as well as other manifestations[4]. Moreover, conventional MRI may present limitations in interpretation because lesions may be acute and/or chronic[5], and we recognize that MRI studies should include special techniques (e.g., diffusion and metabolites).

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REFERENCES

1. Zirkzee EJ, Steup-Beekman GM, van der Mast RC, Bollen EL, van der Wee NJ, Baptist E, et al. Prospective study of clinical phenotypes in neuropsychiatric systemic lupus erythematosus; multidisciplinary approach to diagnosis and therapy. J Rheumatol 2012;39:2118-26.
2. Bertsias GK, Boumpas DT. Pathogenesis, diagnosis and management of neuropsychiatric SLE manifestations. Nat Rev Rheumatol 2010;6:358-67.
3. Lee SW, Park MC, Lee SK, Park YB. The efficacy of brain 18F-fluorodeoxyglucose positron emission tomography in neuropsychiatric lupus patients with normal brain magnetic resonance imaging findings. Lupus 2012 Aug 31. [E-pub ahead of print]
4. Cellucci T, Tyrrell PN, Sheikh S, Benseler SM. Childhood primary angiitis of the central nervous system: Identifying disease trajectories and early risk factors for persistently higher disease activity. Arthritis Rheum 2012;64:1665-72.
5. Peterson PL, Axford JS, Isenberg D. Imaging in CNS lupus. Best Pract Res Clin Rheumatol 2005;19:727-39.

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