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IMAGES IN NEUROLOGY

Brain atrophy after cortical hyperintensities in systemic lupus erythematosus

Atrofia cerebral após hiperintensidades corticais no lúpus eritematoso sistêmico

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A 29-year-old woman with systemic lupus erythematosus (SLE) developed seizures, renal failure and coma. Neurological examination was unremarkable; eletroencephalogram and spinal fluid analysis were normal, anti-DNA antibodies were positive. Brain MRI disclosed cortical hyperintensities (Figure). She received metylprednisolone and cyclophosphamide with no improvement, but recovered consciousness after plasmapheresis. She evolved with psychosis, cognitive complaints and follow-up MRI disclosed brain atrophy. Positive anti-DNA antibody, plasmapheresis response and selective grey matter involvement suggest that cortical hyperintensities were secondary to an immune response against neuronal components rather than postseizures changes or vasculitis[1]. Neurodegeneration may ensue after cortical hyperintensities in SLE.

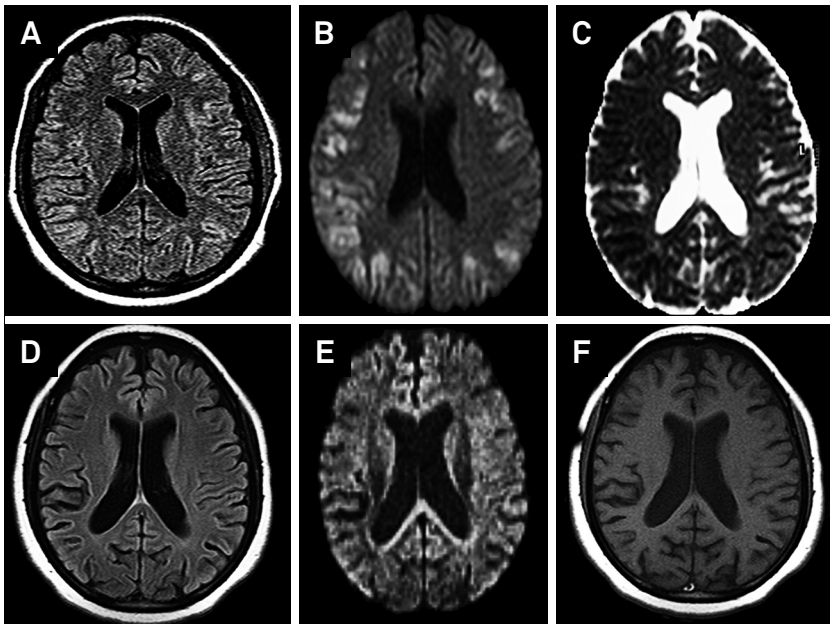

Figure. Initial MRI: A, B and C. Axial FLAIR images showing cortical hyperintensities (A) with increased signal on DWI (B) and decreased signal on ADC map (C), suggestive of cytotoxic edema. Follow up MRI six months later: D, E, F. Axial FLAIR (D) and DWI (E) images showing improvement of signal abnormalities. A non-contrast axial T1 image (E) showing brain atrophy.

Reference

1. Luyendijk J, Steens SC, Ouwendijk WJ, Steup-Beekman GM, Bollen EL, Grond J et al. Neuropsychiatric systemic lupus erythematosus: lessons learned from magnetic resonance imaging. Arthritis Rheum. 2011;63(3):722-32. doi:10.1002/art.30157

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