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; electroencephalogram and spinal fluid analysis were normal, anti-DNA antibodies were positive. Brain MRI disclosed cortical hyperintensities (Figure). She received methylprednisolone 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. Neurodegeneration may ensue after cortical hyperintensities in SLE.

Reference