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A rare case of encephalitis in systemic lupus erythematosus

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Systemic lupus erythematosus (SLE) is an autoimmune disease associated with production of autoantibodies against the antigens of cellular nuclei or cytoplasm and chronic inflammatory cascade activation [1]. This disease affects multiple systems, including the kidneys, skin, serous membranes and the nervous system [2-4]. Meningoencephalitis is an extremely rare complication, affecting about 1% of SLE patients [5].

A 38-year-old woman was admitted to the Neurology Department following her first-ever generalized tonic-clonic seizure, which had occurred one month earlier. She had been receiving treatment for SLE and its complications for the previous 12 years, including anemia, antiphospholipid syndrome, and renal insufficiency. Her current therapy includes immunosuppression with cyclosporine and prednisone. Neurological examination revealed no abnormalities. EEG revealed bilateral epileptiform changes in the frontotemporal areas along with irregular background activity. Magnetic resonance imaging (MRI) showed diffuse lesions surrounded by edema without contrast enhancement, hyperintense in T2-weight and

hypointense in T1-weighted images mainly in the medial part of the right temporal lobe. Similar, smaller changes were seen in the right insula and bilaterally around the third ventricle (Figure 1A-C). MR angiography revealed features of vasculitis in the vessels within the affected area.

A slight pleocytosis of 14 cells with neutrophils prevalence but normal protein and glucose levels were found in the cerebrospinal fluid (CSF). Assessment of CSF antibodies excluded Lyme disease, tick-borne and herpesvirus encephalitis. Leukopenia and a decreased platelets level without changes in the levels of the inflammatory markers were noticed in serum. Serum testing was positive for anti-dsDNA (140 U WHO/ml) and cANCA (34.5 U) antibodies. No serum antibodies against NMDA, AMPA-1, AMPA-2, LGI-1 or CAPR2 receptors were noticed, neither were the neuro-oncogenic antibodies. Oligoclonal IgG bands type IV were present both in the CSF and the serum, indicating the lesions of the blood-brain barrier.

The differential diagnosis included particularly conditions commonly seen in immunocompromised patients, like autoimmune encephalitis, opportunistic viral or bacterial neuroinfection, as well as neoplasms and paraneoplastic syndromes. The past herpesvirus infection and the location of the lesions in the temporal lobe suggested viral encephalitis, however, the antibodies level in blood and CSF was assessed as clinically irrelevant. Moreover, viral encephalitis cases usually have a fulminant course, whereas this patient did not manifest such symptoms. Neoplasms were excluded by MRI. Paraneoplastic syndrome was not supported by the presence of any relevant antibodies. The encephalitis caused by infiltration of the lupus antibodies via the damaged blood-brain barrier seemed to be the most probable preliminary diagnosis.

The treatment included an increased dose of steroids, immunoglobulin infusion and an antiepileptic drug (levetiracetam). The control MRI after 2 weeks revealed a regression of the inflammatory lesions (Figure 1D-F). The positive response to this therapy additionally

supported the preliminary diagnosis. The patient was transferred to the Immunology Department for further examinations and treatment modification.

Autoimmune encephalitis is an extremely rare complication of SLE that could have a non-characteristic, oligosymptomatic course with disproportions between the clinical and radiological image. Therefore, a broad-spectrum diagnostic including the atypical inflammatory diseases is required in SLE patients.

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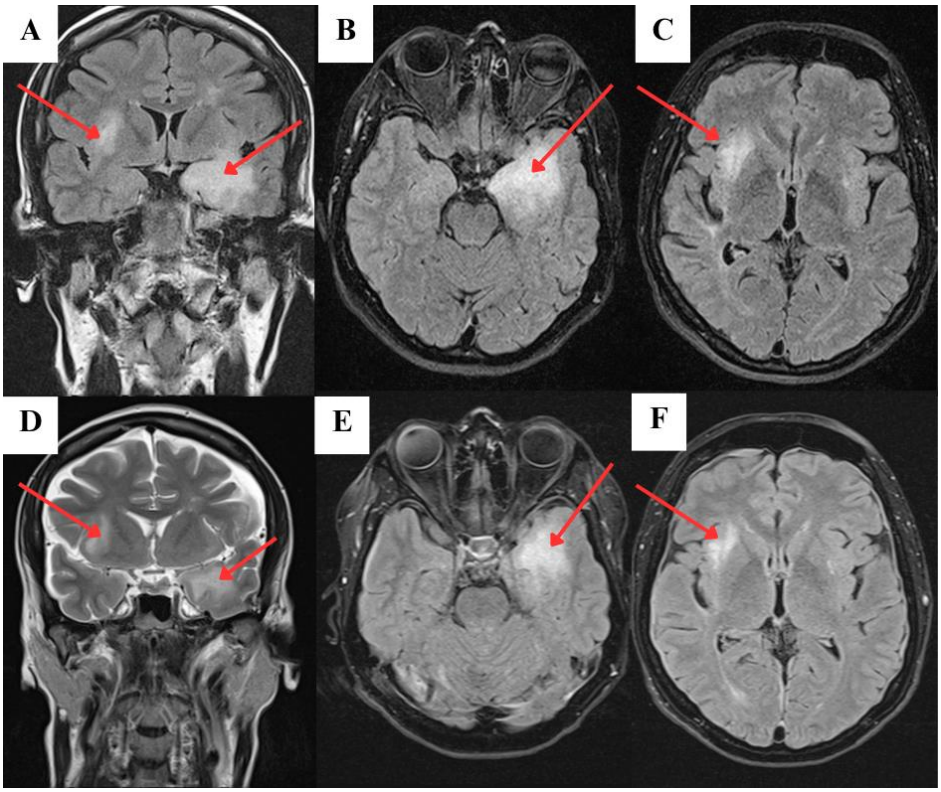

Figure 1 Head magnetic resonance imaging (MRI) scans; **A–C** – MRI upon the admission showing diffuse lesions surrounded by edema without contrast enhancement, hyperintense in T2-weighted and hypointense in T1-weighted images, localized primarily in the medial part of the right temporal lobe; **D–F** – follow-up MRI after 2 weeks of treatment showing regression of the inflammatory lesions

Short title: A rare case of encephalitis in systemic lupus erythematosus