

Case report

Multiphasic disseminated encephalomyelitis, systemic lupus erythematosus and antiphospholipid syndrome

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Abstract

We report a case of multiphasic disseminated encephalomyelitis (MDEM) associated with systemic lupus erythematosus (SLE) and antiphospholipid syndrome. The initial presentation was suggestive of multiple sclerosis. Further clinical attacks, MRI imaging, and CSF findings led to a diagnosis of disseminated encephalomyelitis (DEM). Multiple episodes of neurological dysfunction, which differed in clinical presentation, further categorized the diagnosis as multiphasic DEM. The co-occurrence SLE and antiphospholipid syndrome is unusual and provided an additional diagnostic challenge.

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1. Introduction

Disseminated encephalomyelitis is an uncommon demyelinating disease that usually presents with a monophasic course; however a multiphasic (MDEM) occurrence may also develop. Typically described in children it can also occur in adults [1,2]. The co-occurrence of DEM with autoimmune diseases is rarely described [3]. We report a case of MDEM associated with systemic lupus erythematosus and antiphospholipid syndrome.

2. Case report

A 34-year-old Hispanic woman, married with three children, had a past history of Migraine and joint pains with persistent positive antinuclear antibodies. She had developed neurological symptoms approximately 4 years earlier,

when she developed transient sensory symptoms suggestive of Lhermitte's phenomenon. Ten days after her last delivery she experienced blurred vision, back pain and bladder retention. At that time her brain MRI showed small areas of increased signal intensity within the deep white matter of the right posterior parietal lobe and left frontal lobe visualized on T2 and FLAIR sequences. MRA was normal. The spinal fluid showed three WBC, glucose 44 mg/dl, total protein 90 mg/dl, normal IgG index, absent oligoclonal bands and myelin basic protein 140.13 ng/dl (0.07–4.10). She received a course of IV methylprednisolone pulses (1 g each) and improved within a few days. A month later developed sensory thoracic paresthesias from the T8 level down to her feet. At that time, brain MRI showed subtle mild foci of high signal on the FLAIR images within the white matter, the two previous lesions had disappeared. Cervical MRI was negative for intrinsic lesions.

Follow up brain MRI a few weeks later revealed small numerous high signal intensity lesions with prominent involvement of the corpus callosum, periventricular and cerebellar peduncles in T2 and FLAIR sequences; there was no contrast enhancement in T1. The cervical MRI showed one white matter lesion at C2–C3 with contrast enhancement. She was diagnosed with multiple sclerosis and was started on interferon beta 1a 30 mcg IM weekly and

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symptomatic medications for fatigue and headache. For a period of 6 months she had mild intermittent exacerbation-like symptoms described as increasing numbness, fatigue and headaches; these were always related to her menstrual period.

She received oral or IV steroids at least for three times. She never returned to her clinical baseline.

After this 6 months period the patient developed nausea, vomiting, headaches, mild dysarthria, left hemiparesis

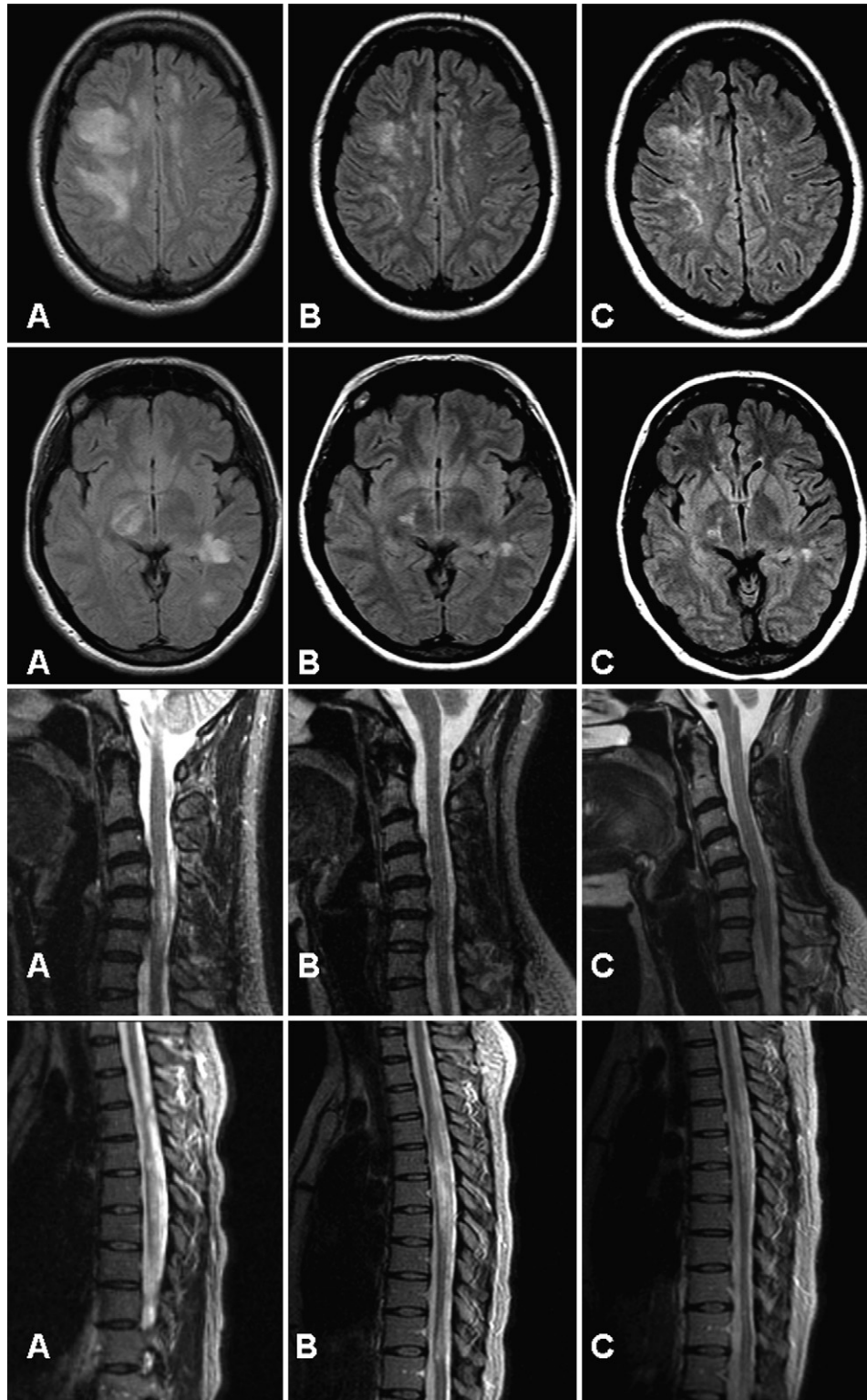


Fig. 1. Brain MRI FLAIR images. Cervical MRI STIR image, and thoracic MRI STIR image at presentation (A), at 2 months follow-up (B), and at 3 months follow-up (C).

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