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LETTER TO THE EDITOR

Posterior reversible encephalopathy syndrome in a patient with systemic lupus erythematosus after cessation of oral prednisone

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Dear Editor,

Posterior reversible encephalopathy syndrome (PRES) is a neurological disorder that is first described by Hinchey and colleagues in 1996 [1]. It is associated with various clinical features such as hypertension, seizures, headache, visual changes and conscious disturbance [2]. In previous reports, PRES have been reported in patients with SLE. The use of immunosuppressive drugs was an established cause of PRES in SLE [3]. Here we describe a case of PRES due to cessation of oral prednisone. To the best of our knowledge, this is the first report of PRES due to cessation of oral prednisone.

A 34-year-old man presented to our department with headache and blurred vision for 1 day. He had a history of SLE and lupus nephritis for 6 years. Three months before admission, the patient presented to the department of rheumatology with a flare of SLE including malar rash, arthritis, and pancytopenia. The patient was free of ophthalmologic and neurologic symptoms then. He was hospitalized and received intravenous pulse therapy of methylprednisone at 250 mg/day given intravenously for 3 days. The patient was discharged symptom free and was taking a maintenance dose of oral prednisone at 60 mg/day. A week before admission, the patient had suddenly stopped taking oral prednisone. He gradually developed bilateral headache and blurred vision 6 days after cessation of oral prednisone. His headaches were described as constant, bilateral temporoparietal and were associated with nausea and vomiting. The patient was only able to distinguish between light and dark. The visual acuity was severely impaired and he was unable to count fingers before his eyes. On examination, the pupils were round, equal and accommodate to light. Optical fundus examination was unremarkable. The anti-nuclear antibodies and anti-dsDNA antibodies were positive. Laboratory examinations revealed urea nitrogen 13.7 mmol/l, serum creatinine 197 μmol/l, uric acid 885 μmol/l, C3 of 0.46 g/l (reference range 0.79–1.52 g/l), C4 of 0.16 (reference range 0.16–0.38 g/l). The admission head MRI was performed and fluid attenuated inversion recovery (FLAIR) sequence showed bilateral hyperintensities in the frontal, parietal, temporal, occipital lobes and the cerebellum, which is consistent with vasogenic edema (Fig. 1a). The patient continued oral prednisone at a dose of 50 mg on a daily basis after admission. The patient was free of symptoms 1 week after restoration of the oral prednisone treatment. Follow-up brain MRI was performed 6 days after admission and the vasogenic edema on FLAIR images was obviously resolved (Fig. 1b). The patient was discharged with complete clinical resolution.

Posterior reversible encephalopathy syndrome is a clinical and radiological entity that may present with a constellation of symptoms including headache, seizures, visual disturbances and altered mental status. Although the precise mechanism remains incompletely understood, blood–brain-barrier leakage and endothelial dysfunction seem to play key role in the development of PRES. Radiological examination is important to rule out alternative causes and aid

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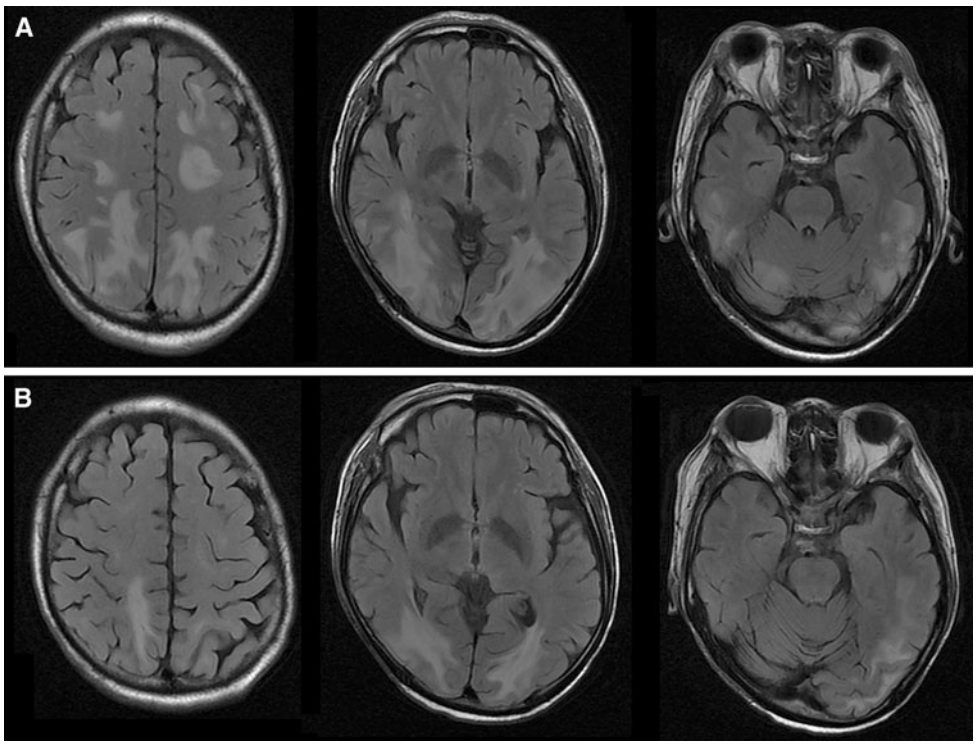

Fig. 1 Admission MRI FLAIR images (**a**) showing diffuse hyperintense signal in the frontal, parietal, temporal, occipital lobes and the cerebellum. Follow-up MRI (**b**) performed 6 days after admission and re-commencement of prednisolone showed partial regression of hyperintense signals on FLARI images

diagnosis. MRI may reveal abnormal findings in the occipital and parietal regions consistent with vasogenic edema. The lesions were usually bilaterally symmetric and could be best appreciated with FLAIR sequence on MRI scan [4]. In our case, the T2 FLAIR hyperintense lesions were more extensive and involve the frontal, parietal, temporal, occipital lobes, and the cerebellum. Although subcortical and deep white matters of the posterior cerebral regions are commonly affected in patients with PRES, atypical radiographic patterns may occur in frontal lobe, temporal lobe and, cerebellum.

To date, several PRES cases have been reported in SLE patients [3–5]. The clinical and neuroradiological features were not specific. The similarities of the clinical manifestations between PRES and neuropsychiatric SLE makes the diagnosis challenging. Since hypertension, immunosuppressive agents and cytotoxic drugs use are quite common in the setting of SLE, the peculiar role of SLE in the pathogenesis of PRES is not clear. In previous reports, the use of cytotoxic drugs or intravenous methylprednisolone has been associated with PRES in most patients with SLE [5]. In our case, cessation of oral prednisone seems to be the insulting factor of PRES. Our patient quickly recovered after restoration of oral prednisone. Physicians should be aware of neurological and ophthalmological symptoms in patients with SLE. As our case highlights, early identification and appropriate management of insulting factor is important for reversal of the clinical symptoms.

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Conflict of interest The author reports no conflict of interest.

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