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CASE REPORT

Reversible MRI lesions after seizures

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After generalized or partial seizures, transient lesions may appear on magnetic resonance (MR) images. The mechanisms of MR changes might be a defect in cerebral autoregulation and blood–brain permeability. We report a patient with partial and secondary generalized tonic–clonic seizures. After her first seizure which was generalized tonic–clonic in nature, we detected multiple high signal intensities over the frontal cortical area on proton density images which were enhanced with gadolinium on TI-weighted images. The first and repeated EEGs showed no abnormalities or epileptic discharges. We started carbamezapine (600 mg/d) and excluded systemic diseases like vasculitis, infections, aetiological factors causing cerebrovascular diseases. In the follow-up, she was seizure free under antiepileptic therapy and no other nerological deficit. Repeated MR scans after 24 months from her first seizure revealed no pathologic signal intensities. Although the pathophysiology is unknown, recognition of reversible lesions helps diagnostic and therapeutic approaches to abnormal MR findings after seizures.

Key words: magnetic resonance imaging; epilepsy; irreversible lesions; partial seizure.

INTRODUCTION

Neuroimaging techniques, magnetic reasonance imaging (MRI) and computerized tomography (CT) are the most important and specific imaging techniques for evaluation of patients with seizures. MRI can detect the pathological findings in 20–70% of patients with partial seizures[1]. After generalized or partial seizures, transient lesions may appear on magnetic resonance imaging (MRI)[1–4]. The pathophysiology of these lesions are not clearly understood but may be explained by vasogenic or cytotoxic oedema as a consequence of regional ischaemia or other cellular metabolic disturbances[1]. We present a patient with complex partial and secondary generalized seizures and multiple reversible cortical and subcortical abnormalities appeared on different parts of the same hemisphere.

CASE REPORT

A 30-year-old, left handed woman was admitted to our hospital with a generalized tonic–clonic seizure following paraesthesia on the right side of the face. After regaining consciousness 2 hours later, she nad no neurological deficit. Cranial CT imaging immediately after this first episode showed a small low-density area on the left frontal precentral corticosubcortical area. The following day, a cranial MRI scan was carried out showing multiple high signal intensities in the left frontal area on proton density images which were enhanced with gadolinium on T1-weighted imates (Fig. 1 A–C). There was no abnormality on her electroencephalography (EEG). Thereafter she was started on carbamezepine (600 mg/day). She was normotensive in the clinical follow-up. Routine haematological values were normal (haematocrit, white-cell count, platelet count, prothrombin time, and partial-thromboblastin time); values for sodium, potassium, chloride, calcium, phosphorus, glucose, uric acid, total protein, albumin, globulin, bilirubin, aspartate aminotransferase, lactate dehydrogenase, creatine kinase, alkaline phophatase, urae nitrogen and creartinine were normal. There was no abnormality in her electrocardiogram (ECG) and plain chest film. Serum immunelectrophoresis gave normal patterns; tests for antinuclear antibody (ANA), antiDNA, anticardiolipin antibody

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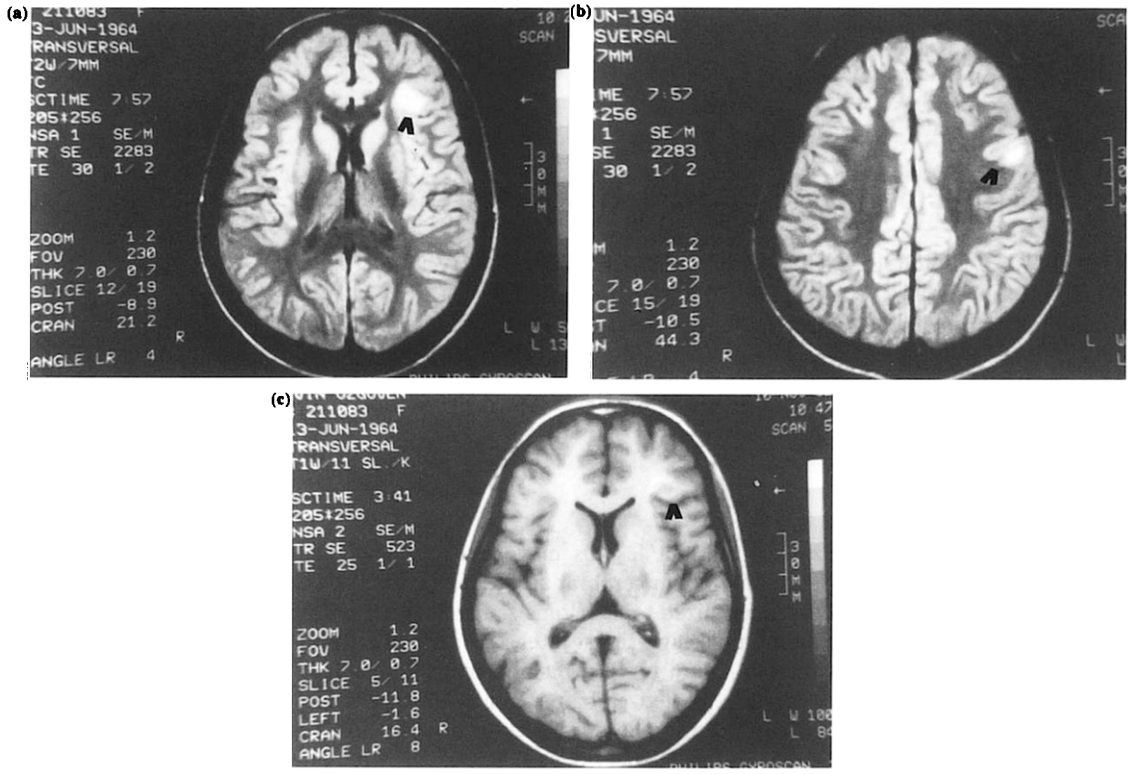

Fig. 1: There were multiple high signal intensities appeared over the left frontal precentral corticosubcortical and paracentral cortical areas on proton density (0.5T, TR 2283, TE 30) images (a, b) which also became enhanced with gadolinium on T1-weighted (0.5T, TR 523, TE 25) images (c).

(ACA), rheumatoid factor (RF), human immundeficiency (HIV) antibodies were negative; anti smooth muscle antibody, protein C, protein S, C3, C4, fibrinogen and fibrin degradation products levels were measured to be normal. Carcinoembryonic antigen (CEA), alpha featoprotein (AFP) and human choriogonodotrophin (β-HCG) were normal. A tuberculin skin test was negative. Lumbar puncture yielded colourless fluid with normal protein, glucose and contained no cells. Bacterial, fungal and tuberculosis cultures were negative and no oligoclonal band was detected. Microscopical examination showed no acid-fast bacilli or other micro-organisms. Evoked potential measurements were normal. Transthoracal and transesophagial electrocardiographic images showed no pathology other than grade II mitral valve prolapsus. Twenty-four hours Holter monitorization did not detect any cardiac arrythmia. On cerebral digital subctraction angiography, no specific findings were detected. Computerized brain mapping and sleep deprivation and sleep EEGs were all normal. A pulp biopsy was made to exclude Raynaud phenomenon and normal findings were observed.

Eight months later, she had three consecutive seizures that started with an abrupt speech arrest, deviation of the eyes, loss of contact and secondarily generalized tonic–clonic seizures in one day and an episode of complex partial seizure presented with loss of contact, repetitive lipsmaking and chewing and secondarily generalized tonic–clonic seizure 12 months later due to non-compliance of drug use. On her repeated EEGs, there was no abnormality. Repeated MRI scans done after these seizure and 24 months from her first seizure as a control scan revealed no pathologic signal intensities (Fig. 2 a, b). She was followed for 40 months and she was free of seizure with carbamezapine 600 mg/day and also she had no symptoms and signs of any other systemic or cerebral disease.

DISCUSSION

The reversible MRI lesions were recently described after a single partial seizure and lesions point to the areas of maximal ictal activity[2–4]. In experimental studies, regional hyperfusion and

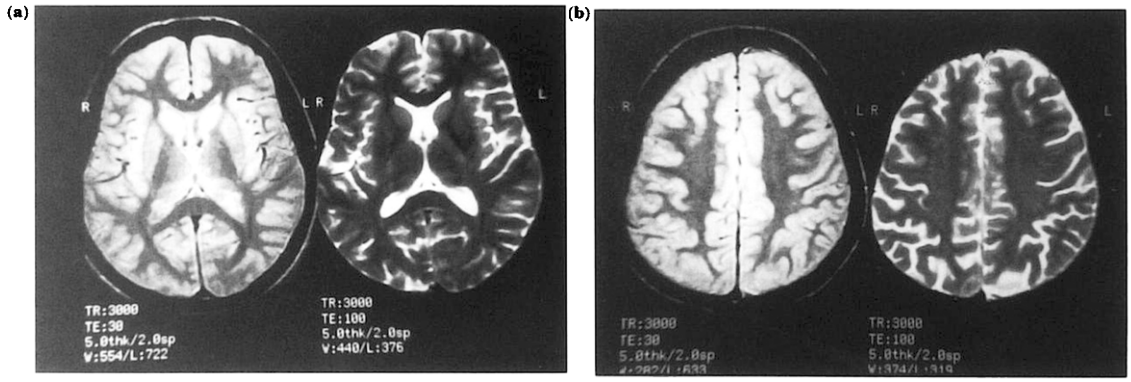

Fig. 2: Repeated MRI scans done after a seizure and 24 months from her first seizure as a control scan revealed no pathologic signal intensities on both proton density (1.5T, TR 3000, TE 30) and T2-weighted (1.5T, TR 3000, TE 100) images at the level of the foramen Monro (a) and centrum semiovale (b).

increased vascular permeability consistent with cerebral vasogenic oedema accompanied with partial seizures and there may be also contrast enhancement as a result of transitory regional hyperperfusion[2]. And also there might be cytotoxic cerebral oedema due to regional ischaemia or other cellular metabolic disturbances[2]. The CT and MRI lesions consistent with cerebral oedema appeared especially after partial status epilepticus[1,2]. So the lesions and the severity of the seizures are correlated, i.e. the frequency and duration of the seizures[2–4]. In India, it was suggested that postictal cerebral oedema might be more common in patients with occult cerebral cysticerosis[5,6]. And also in the syndrome of mitochondrial myopathy, encephalopathy, lactic asidosis and strokelike episodes (MELAS) there might be transient MRI abnormalities after seizures[7]. The timing of development of MRI changes is not well established. In the studies, lesions appeared less than 5 days after onset of partial seizure and could resolve in 30–90 days[2,3]. If the lesions resolved incompletely, tumor like underlying lesions should be excluded.[2,3] Rao and colleagues emphasized venous thrombosis in a patient with diappearing brain lesions[8]. In our patient, the lesions were located on the frontal lobe just after the secondary generalized tonic–clonic seizure. In the following year the patient had two more complex partial and secondary generalized seizures but we did not detect any abnormality on MRI after seizures. Also, there was no abnormality in repeated blood and serological tests. In the follow-up there is no correlation between ictal activity and MRI lesions and we cannot explain the transient MRI lesions only by cerebral oedema occurred after partial seizure. These detected lesions could affect the treatment protocol, i.e. antiepileptic drugs might be started after the first seizure in patients with MRI lesions. In the studies, there are no pitfalls on the prognosis of these patients.

After single partial seizures focal reversible MRI changes that might indicate a focus could be observed and the patient should be treated even if it is the first seizure. The patient should also be followed with periodic MRI scans to exclude any underlying lesion.

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