Case report

Curable focal epilepsy

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Case report

This gentleman presented, aged 36, with numbness on the left side of his face, which subsided over 10 days. Four months later, he developed numbness with pins and needles affecting his left arm followed shortly affecting his right arm and then both legs within a month. Magnetic resonance imaging (MRI) brain showed two white matter lesions involving the corpus callosum with no other abnormalities. MRI whole spine revealed high signal abnormalities at C2, C3, T7 and T8. He was subsequently diagnosed with multiple sclerosis (MS). He responded well to steroids and was commenced on interferon beta-1A. He remained well for the next 2 years with no further relapses with an expanded disability status scale (EDSS) score of 0. He then complained of a sensory disturbance in both thighs and knees with some urgency with a documented EDSS of 1.5. A few months later he stated to complain of a metallic taste in the mouth followed by loss of awareness but without losing consciousness. He had two to three of these stereotypical attacks per day over the course of 2 months. Imaging revealed an enhancing lesion in the right insular cortex (Figure 1), which was considered a relapse and the anatomical substrate of his ‘seizures’. After 3 days of iv methylprednisolone he made a good recovery.

Discussion

In a recent population study of 5041 patients with MS, 102 (2%) patients had a history of seizures, which is similar to other studies. Sixty-seven (1.3%) patients with epileptic seizures could not be explained by any other cause than MS and it was the initial manifestation in seven of these patients.1,2

Gasparini et al. recently described a 42-year-old man with a 13-year history of MS and minimal disability (EDSS 1) complaining of paroxysmal attacks lasting seconds to minutes characterized by stiffness and jerking of the limbs which evolved on two occasions to generalized convulsion seizures. Electroencephalogram abnormalities were observed at the vertex region and MRI brain scan revealed the presence of a small non-enhancing intra-cortical lesion in the right paracentral lobule that had been absent 3 months earlier. Interestingly, this patient, as with our case, did not respond to anti-convulsants but to methylprednisolone.3 There are only a few other cases that have reported a causal relationship between cortical lesions and seizures where these cases, responded to steroids rather than anti-epileptic drugs.4–7 Recently, natalizumab was demonstrated to reduce the frequency of complex partial seizures and stop the evolution into generalized tonic clonic seizures in a 24-year-old patient that was resistant to anti-epileptic drugs and glatiramer acetate.8 It is well recognized that lesions in MS brains not only occur in periventricular and deep white matter but also in cortical lesions. However, this very common pathological finding together with the relatively uncommon development of epilepsy among patients with MS suggests other factors are involved. The causal pathophysiological relationship between MS and epilepsy is not clear. Possible theories include an ‘oedema effect’ of demyelinating lesions where the acute
presentation responds to steroids rather than standard anti-epileptic medication. The other hypothesis is that the plaque may itself act as an ‘irritative focus’. It has been demonstrated that abnormal sodium channel activity has been observed in axons that have undergone specific insults such as demyelination resulting in neuronal hyperexcitability. This would explain the 3-fold increase in prevalence of epilepsy among patients with MS compared to the background risk as well as explain how some cases respond to standard anti-epileptic medication. It is possible that there is a variable contribution of both hypotheses to the seizure tendency. Patients with MS and epilepsy could be classified into (i) those with seizures associated with MS relapse who have rarely recurrent fits and (ii) patients with progressive cognitive deterioration in whom seizures were recurrent and possibly complicated with status epilepticus. The former may be more responsive to steroids whereas the latter may respond to anti-epileptic medication or with disease modifying drugs in treatment resistant cases as described above.

In summary, this case describes a clinico-radiological correlate of focal epilepsy secondary to an active plaque of demyelination which responded to steroids. This has been rarely described in the literature and emphasizes the need to consider focal epilepsy as the sole manifestation of an acute relapse which may be resistant to anti-epileptic medication.

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References