CASE REPORT

Jacksonian seizure as the relapse symptom of multiple sclerosis

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Epilepsy is more common in patients with multiple sclerosis (MS) than in the general population, occurring in 2-3% of patients. Convulsions may be either tonic–clonic in nature or partial complex. In these individuals, seizures most likely result from lesions present in the cerebral cortex and subcortical white matter. A Jacksonian seizure is a type of simple partial seizure characterized by abnormal movements that begin in one group of muscles and progress to adjacent groups of muscles. We describe a case of Jacksonian seizure as the relapse symptom of MS. Focal motor seizures of this patient have been observed before and presumably marking the clinical onset or during acute bouts of MS. In this case, Jacksonian seizures appear to be the sign of a flare of MS, while the majority of seizures had been reported occur unrelated to MS relapses.

Key words: Jacksonian, multiple sclerosis, seizure

INTRODUCTION

Multiple sclerosis (MS) is considered a T cell-mediated autoimmune disease of the central nervous system (CNS) with a complex genetic background.[1] A Jacksonian seizure was first described by the English neurologist Dr. John Hughlings Jackson in 1863 and is a type of simple partial seizure characterized by abnormal movements that begin in one group of muscles and progress to adjacent groups of muscles (motor seizure). These movements reflect the march of seizure activity arising from the prerolandic gyrus area of the brain through the brain›s motor cortex. The causes of seizures may include a lesion of the frontal lobe of the brain, lack of oxygen resulting in tissue damage, or tissue damage resulting from brain tumor or stroke.[2]

Seizures can occur in MS patients and the risk of epilepsy seems to be three times higher in patients with MS than in the general population.[3] Seizures can be the presenting symptom of MS but have been observed in relapsing-remitting as well as in secondary or primary progressive MS. Here, we describe a case of MS exacerbation with recurrent of focal motor seizure. For better clarity, we split the description of seizures and epilepsy from the nonepileptic MS course.

CASE REPORT

A 46-year-old woman complained of the sudden appearance of muscle twitching or contractions of the fingers of right hand, spreading to arm and face on the same side. Seizure was happened three to four times per day as reported by herself. This is sometimes felt as a tingling sensation of right lower and then upper extremity. The seizures usually lasted 30-60 s. There was no loss of awareness, alertness, or consciousness during seizures.

Multiple sclerosis onset and course

In October 2000, a previously healthy 34-year-old woman complained of the acute onset of paresthesia, numbness, and tingling of lower and upper extremities. She had unsteadiness gait with fatigability. These symptoms continued at least 2-3 months and then followed by a focal motor or sensory seizures of hands occasionally.

On neurologic examination right-side finger to nose dysmetria and hyper reflexia was detected. Routine laboratory tests were normal except mild anemia. Brain MRI was showed low signal nonenhancement lesions on T1-weighted and some high signal changes of white matter on T2-weighted. The possible MS was suggested and the patient followed.

During 2001-2008 the patient did not complain of any symptom referable to MS and EDSS (expanded disability status scale) was zero. The patient received no treatment for MS during this period.

Multiple sclerosis course during 2009-2012

The second MS relapse occurred in May 2009 and was characterized by blurred vision and diplopia. Neurological examination disclosed a mild right internuclear ophthalmoparesis and MRI revealed the absence of new T2-w or Gd-enhancing lesions [Table 1].

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A course of methyl-prednisolone 1 g/day for 5 days was administered and symptoms subsided in 4 weeks. The patient was not received beta interferons because of she had past history of seizures.

The third MS relapse occurred 3 years later (September 2012) and was characterized by focal motor seizures of right hand, reduction of vision of both eyes, and diplopia.

In neurologic examination, the limitation of ocular motility with multidirectional nystagmus and mild-to-moderate optic atrophy of both eyes together with dysmetria and tandem gait ataxia is reported. In addition, memory, attention, and problem solving were mildly reduced, and EDSS score increased to 2 [Table 1]. MRI activities emerged, showing new T2-W lesions in brain and Gd- enhancing T1-W brain lesions [Figures 1 and 2]. Brain MRI with T2-W and FLAIR conditions show multiple high-signal-intensity plaques in the periventricular, semi-oval, cortico-medullary junction regions [Figures 3 and 4].

A new course of methyl-prednisolone 1 g/day for 5 days was administered and symptoms subsided in 4 weeks.

Epilepsy onset and course

In October 2000, 6 months after the appearance of the sensory and visual disturbance syndrome, the patient presented a focal epileptic seizure. Interictal EEG was normal. No specific treatment for seizures was initiated. Recurrent simple partial seizures especially focal motor type was occasionally happened between 2000 and 2012; however, because of lack of abnormal EEG patterns patient did not receive any antiepileptic drug.

In September 2013, the third and the last attack of MS relapse presented with recurrent focal motor seizures (Jacksonian type). The stepwise focal motor seizures confirmed the Jacksonian type.

DISCUSSION

Some paroxysmal symptoms of MS (for example, spasticity, numbness, tingling, and dysarthria) can mimic (or be) simple partial seizures. For this reason, it can be difficult to diagnose MS in people experiencing seizures. It can also be difficult to recognize seizures in those already known to have the disease.[4]

Epilepsy affects between 0.5% and 1% of world population.[5-7] Among other factors, MS represents a risk factor for epilepsy.[8] The pathophysiology of seizures in MS remains to be elucidated although cortical and subcortical lesions may reasonably explain their increased frequency in MS.[4,9] Seizures can occur in MS patients as the presenting symptom or as a relapse, being either related or unrelated to other, nonepileptic clinical

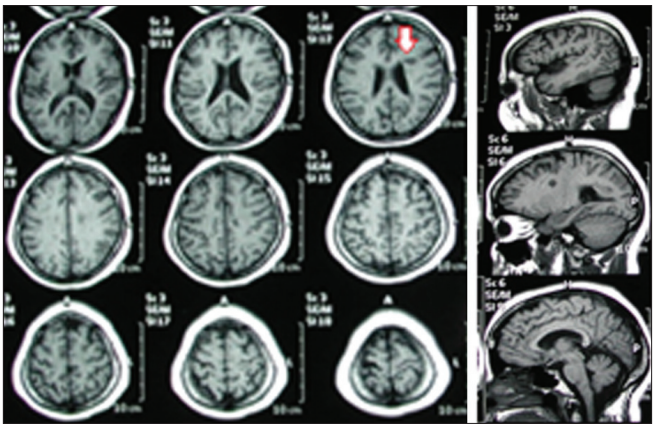

Figure 1: Axial brain magnetic resonance imagine view of multiple scelerosis patient before contract (a) and after contrast (b)

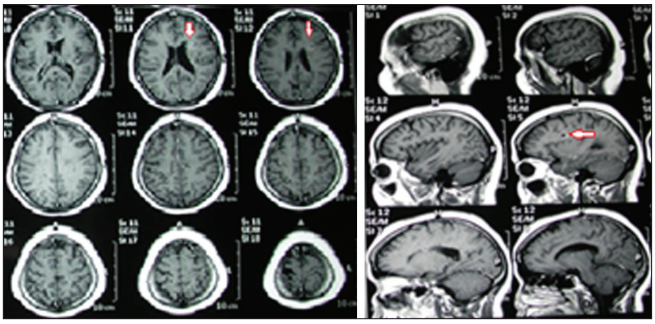

Figure 2: Sagital brain magnetic resonance imagine view of multiple scelerosis patient before contract (a) and after contrast (b)

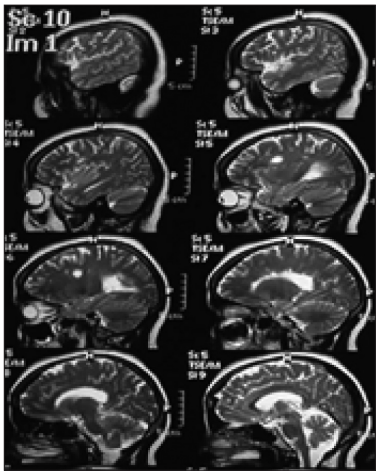

Figure 3: Brain magnetic resonance imagine (T2-Wieghted image) of multiple scelerosis patient

relapses.[4] Data from the literature review show that in the MS patients 1.95% experienced seizures at any time during life. Patients experiencing seizures before MS diagnosis were classified into three categories: (a) 25 (7.3% of patients with MS and seizures) with seizure as the initial presentation of MS; (b) 27 (7.9%) with seizures appearing with other signs and symptoms of MS; and (c) 68 (20%) with seizures occurring years or an unknown period of time before MS onset. Seizure occurring as a symptom of MS relapse was found in 29 patients.[9,10] The prevalence of seizures among MS patients

Table 1: Clinical follow-up of the MS case from MS onset (2000) up to November 2012

Year	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012
Non-epileptic MS relapses	1	0	0	0	0	0	0	0	0	1	0	0	1
Brain MRI activity (new T2 or Gd+lesions or both)	-	-	-	-	-	-	-	-	-	+	-	-	+
EDSS score	0	0	0	0	0	0	0	0	0	1	1	1	2
MS therapy	-	-	-	-	-	-	-	-	-	+	-	-	+
Partial seizures	4	3	4	3	2	3	2	3	3	6	4	2	3
AEDs (daily dose in mg)	-	-	-	-	-	-	-	-	-	400 CBZ	-	-	600 CBZ

MS=Multiple sclerosis; MRI=Magnetic resonance imagination; EDSS=Expanded disability status scale; AED=Administrated daily drug; CBZ=Carbamazepine

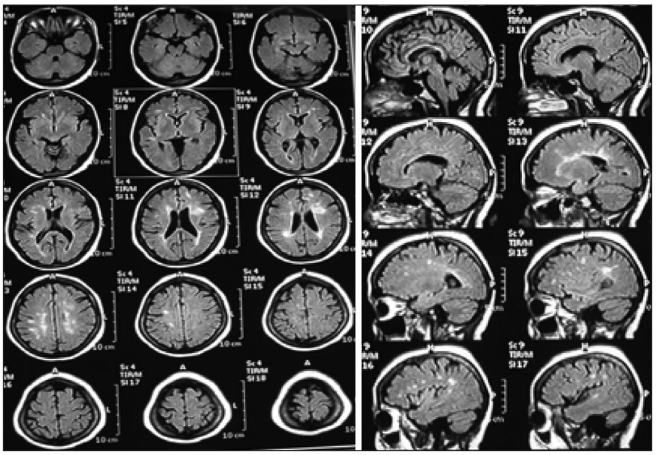

Figure 4: Brain magnetic resonance imagine; axial view (a) and sagital veiw (b) of multiple scelerosis patient two months after treatment

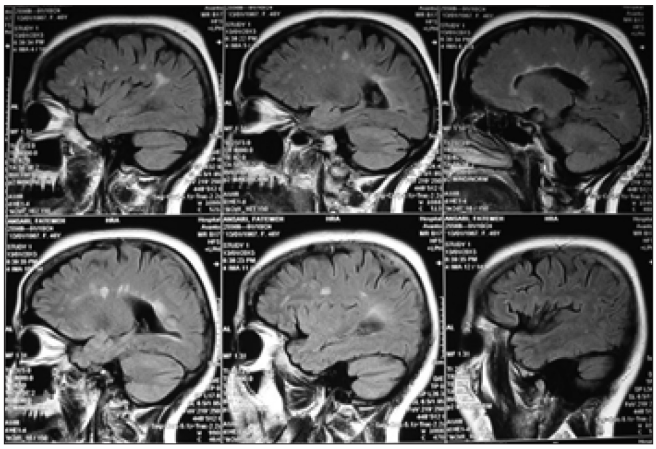

Figure 6: Brain MRI (FLAIR condition) with multiple demyelinating plaques two months later

was higher than that in the general population, indicating a relationship between seizures and MS. Seizures occurred before MS diagnosis in a small percentage of patients.[3]

In our case, seizures occurred in both conditions as indicated on Table 1. Epileptic and non-epileptic symptoms were concomitant at disease onset during 2000 and 2012. In contrast, focal seizures appeared to be clinically and temporally isolated between 2001 and 2012 [Table 1]. Experimental and clinical studies have shown that inflammation mechanisms are activated in epilepsy.[11,12] Proinflammatory cytokines such as IL-1β, TNF-a, and IL-6

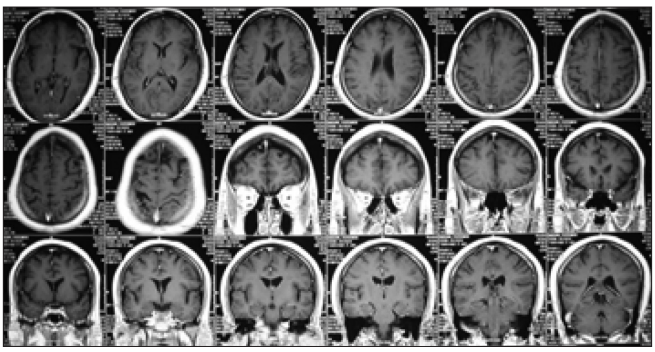

Figure 5: Brain MRI with contrast two months later & without active leison

have been shown to be overexpressed in experimental models of seizures, prominently by glia,[13,14] suggesting that glia activation may contribute to vascular inflammation in epilepsy and MS.[15] Brain MRI was done 2 months later and showed many nonenhansible demyelinating plaques [Figures 5 and 6]. The vast majority of people with MS can control or eliminate their seizures with antiseizure medication, and many people do not have to take any medications at all. Our case the partial seizures well controlled with carbamazepine (600 mg/day) However, a small percentage of people with MS may have seizures that do not respond to medication at all.

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