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Recurrent Seizures and Prolonged Post-Ictal Aphasia in a Patient with Multiple Sclerosis

Key Words

Multiple sclerosis
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Abstract

Objective: To evaluate the duration and underlying pathology of post-ictal aphasia in a patient with multiple sclerosis. **Clinical Presentation:** A 36-year-old female patient with chronic multiple sclerosis developed recurrent seizures followed by prolonged stereotyped post-ictal aphasia that lasted for 4–6 days. There was no response to a therapeutic trial of steroids. **Investigations:** A brain MRI did not reveal new demyelinating plaques. EEG showed persistent abnormalities from the left temporal lobe, which had no temporal relationship to the aphasic episodes. **Conclusion:** We believe that these aphasic episodes represent a post-ictal phenomenon of unusually long duration that has not been reported before.

Introduction

Patients with multiple sclerosis (MS) infrequently develop epilepsy. Epilepsy, however, is more prevalent in patients with MS than in the general population [1]. Paroxysmal speech disorders are commonly observed in complex partial seizures [2], but their association with seizures in MS patients is rare and has not been well studied [3]. We report a case of a 36-year-old female with MS who had several recurrent seizures followed by post-ictal speech disturbances of long duration.

Case Report

A 36-year-old right-handed female had a clinically definite chronic remitting and relapsing MS since the age of 21 (as proven by clinical behaviour, MRI, positive oligoclonal bands in the cerebrospinal fluid and evoked potential studies). After 15 years of a remitting and relapsing course, she became chair-bound with gross cerebellar ataxia, bilateral pyramidal weakness of both lower limbs and a spastic bladder. Since July 1995, she has experienced attacks of generalized tonic-clonic seizures. A period of motor arrest and unresponsiveness preceded each tonic-clonic seizure. Intravenous diazepam controlled the convulsions which lasted less than 2 min. In the post-ictal stage she

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seemed to be confused, muttering some religious verses to herself and was neither able to follow simple orders nor to express herself fluently. As her aphasia evolved she also showed difficulty in initiating conversation, echolalia, and an inability to name objects. Repetition was intact. This lasted for periods ranging from 4 to 6 days after which she returned to her normal state. She suffered from 6 attacks with a 2- to 4-month interval between each attack. Following the first attack she was treated with carbamazepine in a 600-mg dose daily. Following the second episode, methylprednisolone pulse (1 g i.v. daily for 5 days) was added to carbamazepine, and it had no effect on the duration of the attacks. Drug levels were always within therapeutic range. EEGs were performed 2 days following the onset of the first attack, within 2 h of the fourth one, and when she was back to normal. All of them showed slow irregular background activity with no reaction in eye opening and closing. However, the background activity was more organized with a fair response in eye opening and closing in the last EEG. A persistent focus of frequent intermittent slow activity in the delta range was seen in the left temporal leads with occasional sharp waves in the same areas (fig. 1a, b). MRI of the brain showed multiple high signal lesions mainly in the periventricular white matter, internal capsule, pons, and cerebellum. None of these lesions were enhanced with gadolinium (fig. 2). No new subcortical demyelinating plaques were detected. There was also a moderate focal atrophy of the left temporal horn and hippocampus. It was essentially similar to a study done 1 year earlier. Otherwise, the routine laboratory tests and cerebrospinal fluid studies at this time were within the normal range apart from the presence of oligoclonal bands.

Discussion

A causal relationship between MS and epilepsy has been proposed by many authors [4–6]. Cortical and subcortical involvement found on post-mortem studies were thought to contribute to the pathophysiology of convulsions in patients with MS. As many as 25% of such patients had plaques adjacent to the cerebral cortex [5]. Compared to a 0.5% prevalence in the general population, the prevalence of seizures in MS patients varied from 1.7 to 8% [1, 7]. Aphasia, on the other hand, is

more uncommon in MS with a frequency of 0.7–1.0%, in spite of frequent involvement of the language pathways in the disease process [8].

In relation to epilepsy, aphasia can be either a manifestation of an ictal as in partial complex seizures or a post-ictal phenomenon (todd phenomenon) [3, 9–11]. As an ictal phenomenon (during which the patient is alert but dysphasic) there is usually a correlation between the clinical seizure and simultaneous EEG changes, denoting corresponding electrical ictal activity that would cease once the clinical fit was over [9, 10]. In our patient, however, such a pattern was not seen. We have observed a stereotyped sequence of a period of motor arrest, stare and tonic-clonic convulsions, followed by prolonged post-ictal aphasia. The initial language disturbance could have been simply a reflection of a post-ictal confusional state. The subsequent events noted, however, were similar to those seen in transcortical motor aphasia. We could not demonstrate a clear temporal relationship between the aphasia and any ictal EEG changes. Instead there was a persistent focus of slow wave activity which was irrespective to the presence or absence of any seizures or speech disturbances. More invasive studies such as sphenoidal EEG led to exclude the possibility of a continuous ictal phenomenon not revealed by surface EEG. Nevertheless the failure of i.v. valium and other anti-epileptic treatments given during the attacks to reverse the aphasia is a strong indicator against these attacks being of ictal nature. Rolak et al. [10] demonstrated post-ictal aphasia in 5 patients among 44 epileptics with transient post-epileptic neurological deficit, the duration of which varied between 30 min and 36 h. Most of the patients in this study had an underlying structural lesion associated with the deficit. Similarly, our patient had a temporal lobe abnormality that could account for the EEG

a

b

Fig. 1. EEG, illustrating intermittent focal activity from the left hemisphere, consists of shaft high amplitude delta waves (a) and intermittent sharp transients consisting of spikes from the same side (b).

a

Fig. 2. MRI, axial T₂-weighted image shows periventricular high signal white matter lesions (**a**), and coronal T₁-weighted image shows atrophic changes with dilated 3rd and lateral ventricles and in particular the left temporal horn (**b**).

b

changes and the development of seizures, but the duration of aphasia was much longer than that described in the literature. Prolonged aphasia could alternatively be a reflection of a relapse of MS. In such instances, a large and anatomically correlating plaque is frequently demonstrated [3, 8, 12, 13]. In our case no new or relevant demyelinating lesions could be documented on MRI to support the possibility of a relapse. Furthermore, the stereotyped nature of these episodes and the failure to respond to steroid pulse therapy do not favour the occurrence of demyelinating relapses.

In conclusion, we feel that the prolonged aphasia in our patient represents a seizure-related phenomenon of a post-ictal rather than an ictal nature. We are not aware of any similar report of a post-ictal state that would last as long as 4–6 days.

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References

<p>1 Ghezzi A, Montanini R, Basso PF, Zaffaroni M, Massimo E, Cazzullo CL: Epilepsy in multiple sclerosis. <i>Eur Neurol</i> 1990;30:218–223.</p> <p>2 Gabr M, Lüders H, Dinner D, Morris H, Wyllie E: Speech manifestations in lateralization of temporal lobe seizures. <i>Ann Neurol</i> 1989;25:82–87.</p> <p>3 Spat J, Goldenberg G, Mamoli B: Simple dysphasic seizures as the sole manifestation of relapse in multiple sclerosis. <i>Epilepsia</i> 1994;35:1342–1345.</p> <p>4 Kinnunen E, Wikstrom J: Prevalence and prognosis of epilepsy in patients with multiple sclerosis. <i>Epilepsia</i> 1986;27:729–733.</p>	<p>5 Brownell B, Hughes JT: The distribution of plaques in the cerebrum in multiple sclerosis. <i>J Neurol Neurosurg Psychiatry</i> 1962;25:315–320.</p> <p>6 Waxman SG: <i>Clinicopathological Correlations in Multiple Sclerosis and Related Disorders</i>. Adv Neurol, vol 31. New York, Raven Press, 1981.</p> <p>7 Trouillas P, Courjon J: Epilepsy with multiple sclerosis. <i>Epilepsia</i> 1972;13:325–333.</p> <p>8 Achiron A, Ziv I, Djaldetti R, Goldberg H, Kurtizky A, Melamed E: Aphasia in multiple sclerosis: Clinical and radiologic correlations. <i>Neurology</i> 92;42:2195–2197.</p> <p>9 Suzuki I, Shimizu H, Ishijima B, Tani K, Sugishita M, Adachi N: Aphasic seizure caused by focal epilepsy in the left fusiform gyrus. <i>Neurology</i> 1992;42:2207–2210.</p>	<p>10 Rolak LA, Rutecki P, Ashizawa T, Harati Y: Clinical features of Todd's post epileptic paralysis. <i>J Neurol Neurosurg Psychiatry</i> 1992;55:63–64.</p> <p>11 Wells CR, Labar DR, Solomon GE: Aphasia as the sole manifestation of simple partial status epilepticus. <i>Epilepsia</i> 1992;33:84–87.</p> <p>12 Friedman J, Brem H, Mayeux R: Global aphasia in multiple sclerosis. <i>Ann Neurol</i> 1983;13:222–223.</p> <p>13 Thompson AJ, Kermode AG, Moseley IF, MacManus DG, McDonald WI: Seizures due to multiple sclerosis: Seven patients with MRI correlations. <i>J Neurol Neurosurg Psychiatry</i> 1993;56:1317–1320.</p>
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