## SHORT REPORT

# Late Onset Status Epilepticus as a Sign of Multiple Sclerosis

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Received: October 17, 2003

Key Words: Multiple sclerosis, epilepsy, status epilepticus

Multiple sclerosis (MS) is a chronic disabling neurologic disease with a wide spectrum of symptoms and clinical signs in which epileptic seizures may be the first observable symptom. Epilepsy is a common condition with a frequency of 0.5-1% in a normal population. The exact prevalence of epileptic seizures in patients with MS is still a matter of controversy. The reported prevalence rates range between 1.7 and 7.5% (1-6). Similar frequency rates in a general population have also been reported in a recent study (7). Meanwhile, when compared with that expected in the general population, a 3-fold increase in risk for developing epilepsy has been estimated (8).

Any seizure type can be seen in MS, although reports on prevailing seizure type are inconsistent (1,5,6,9-12). Episodes of status epilepticus (SE) have also been reported in MS patients. Simple partial motor, generalized tonic-clonic, generalized nonconvulsive and complex partial SE cases have been reported (3,5,8,13,14). One other controversy is that of seizures as the sole manifestation of MS relapse and onset of MS symptoms with SE is fairly rare (6,9).

We report a case of late adult onset SE, which is the first symptom of underlying multiple sclerosis.

### **Case Report**

A 44-year-old woman was admitted to the hospital following seizures. According to an informant, she had no

known disease at the time and no history of intoxication or trauma. The patient had experienced 5 episodes of generalized tonic-clonic seizure and had not regained consciousness between seizures. In the emergency room she continued to have generalized tonic-clonic seizures. No findings suggesting a focal onset of seizures were observed. At first examination she was unconscious, had dilated and unresponsive pupils and indifferent plantar responses. She was administered a loading dose of phenytoin intravenously in the emergency room, but continued to have seizures that were resolved after intravenous diazepam administration. Laboratory workup including routine blood chemistry and urine investigations and computerized cranial scanning were unremarkable. An electroencephalogram that was obtained 1 day after her seizures revealed no pathologic findings, and subsequent EEGs were also normal.

The patient, after regaining consciousness, stated that she had a diplopia attack that resolved spontaneously 2 or 3 years ago, and occasionally had paresthesia and clumsiness in her arms; but she had not paid attention to these symptoms. On neurologic examination she had no pathological finding except for brisk tendon reflexes and extensor plantar response on the right. The patient was evaluated extensively for causes of seizures, but no other reason was found. Serological tests for *Borrellia burgderfori*, HIV and autoimmune antibodies yielded negative results. The CSF was clear and colorless under normal pressure with normal cell count and protein content, but IgG and the IgG index were above normal. CSF immune-fixation electrophoresis revealed positive oligoclonal IgG banding. Bacteriological and virological examinations revealed no abnormalities. Cranial MRI scanning demonstrated multiple demyelinating lesions in the periventricular white matter, cerebellum, subcortical and deep white matter (Figure). Visual evoked potentials (EP) were delayed.

With the diagnosis of relapsing-remitting MS, the patient was treated with mega-dose (1000 mg) methylprednisolone for 5 days. She was commenced on carbamazepine and remained seizure-free on follow-up examinations.

#### Discussion

SE is a dangerous condition with a high mortality rate, and it requires prompt treatment. Its etiology is similar at all ages. Eighty-two percent of cases of tardive SE are of symptomatic origin, with cerebrovascular disease ranking first (15). Epileptic seizures in MS have been reported for a long time. It is well established using MRI that clinically silent lesions are common in MS and it is not surprising that a seizure may be the only manifestation of a new lesion. Accurate diagnosis of MS is an important point when relating the seizures to this disease. In our case the relapsing-remitting MS diagnosis was confirmed with history, MRI, EP and CSF findings. Although seizures were not the first symptom of the disease, they were the reason the patient sought medical assistance.

The mutual physiopathologic association between MS and epilepsy is controversial. The problem is whether epileptic seizures are the symptoms of MS or just coincidental. Demyelinating lesions themselves have been proposed to act as an irritative focus, as demonstrated by patients with large, unchanging subcortical plaques who experience prolonged seizure activity (1). Edema, which causes a size change over a 4-6-week period in new lesions, may play a part in seizure production in critically located lesions (9,16,17). Other factors, some of which are still not clearly understood such as the fiber, electrolytic changes, size of the plaque, reactive gliosis and the enzyme Na<sup>+</sup>-K<sup>+</sup> ATPase, seem also to play a part in the production mechanism (2).

With contemporary immunologic, electrophysiologic and neuroradiological diagnostic procedures the diagnosis of MS made much more certain and in this way the differentiation of some symptoms as well as epileptic seizures in patients becomes more reliable.

While the onset of MS symptoms may antedate the diagnosis of MS by years, as happened in our patient, MS should be remembered in the etiology of tardive SE before reaching a final diagnosis of idiopathic SE.



Figure 1. Axial T2-weighted MR scanning showing multiple periventricular and large biparietal hyper-intense white matter lesions.

One other issue that merits comment is the approach to this patient in the emergency room. The many regimens that have been proposed for the treatment of SE attest diazepam as the drug to be given first. Immediately thereafter, a loading dose of phenytoin is administered if seizures continue. This patient was not given diazepam until seen by a neurologist. We suggest that approaches to emergency medical conditions like SE must be emphasized in medical emergency education programs.

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