MULTIPLE SCLEROSIS AND LYME DISEASE

In 1994, I saw two cases of classic severe far advanced MS that followed documented Lyme disease that they had suffered many years before. I treated both of these with ceftriaxone with indifferent results.

In 2001, I saw in close succession, two patients who had exposure to tick bites that had resulted in a classic Lyme disease dermatologic reaction.

The first patient had been sick for a year with fatigue, fever, paresthesia, and unsteadiness on her feet. An MRI of her brain had revealed findings consistent with MS. Her search of “the web” convinced her that she had Lyme disease because she had been exposed to ticks and had a bull’s-eye rash just before becoming ill. Serologic tests were equivocal. I agreed with her diagnosis and arranged for her to have a 21-day course of ceftriaxone 2 grams intravenously. She became afebrile, her joint pain disappeared and she became essentially asymptomatic. Within several months she was able to resume her fulltime employment from which she as on disability leave.

MRIs done yearly since then have shown no progression of the abnormalities that were considered consistent with MS. She remains on suppressive doxycycline therapy.

On July 4, 2001, the second patient had a tick bite followed by a bull's eye rash. The rash persisted for a month and her mild arthritis flared to the point that she was becoming unable to work. She concluded that she might have Lyme disease and I agreed. She was given a 21-day course of ceftriaxone 2 grams intravenously with gradual improvement and she returned to work.

She was maintained on oral doxycycline, azithromycin, and cefuroxime. After the initial clinical response she developed paresthesias and balance difficulty, and in July 2003, became so ataxic that she had difficulty in standing. She was hyperreflexic. Her MRI showed findings compatible with MS. At that time, I decided to treat her with glatiramer acetate and another course of intravenous ceftriaxone.

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She stated five days after the start of glatiramer acetate that her

balance greatly improved. She is now back to work fulltime but she is still somewhat ataxic and
has joint pains more typical of Lyme disease then of MS. She is being maintained on oral doxycycline and azithromycin. A delay in getting the intravenous ceftriaxone started, allowed
her to observe a rapid clinical response of her ataxia that possibly was because of the glatiramer
acetate. The tantalizing aspect of this part of her history is that it raises the speculation that in
cases of Lyme disease with autoimmune features, glatiramer acetate might be worthwhile as part
of the initial preventive therapy.

ARTICLES THAT SUPPORT THE HYPOTHESIS

There are articles in the medical literature that are consistent with the hypothesis that Lyme
disease can cause a clinical picture almost identical to MS. Pachner has called Lyme disease the
great imitator.[3] Chmielewska-Badora has questioned whether there is a connection between
Lyme disease and MS.[10] Lakos has stated that Lyme disease may imitate MS.[11] Lana-Peixoto
has reported positive serology for Lyme disease in MS.[12] Garcia-Monco has found antibodies to
myelin basic protein in Lyme disease.[13] Martin has demonstrated that Borrelia burgdorferi can
act as a trigger for autoimmune T-cell reactions within the central nervous system.[14] A possible
explanation for this phenomenon is that Borrelia burgdorferi exhibits molecular mimicry with
human nervous tissue.[16] Molecular mimicry might evoke antimyelin T-cells against myelin
basic protein.[14] Baig, et al demonstrated cells secreting antibodies to myelin basic protein in
cerebrospinal fluid in patients with Lyme neuroborelliosis.[17] Gay and Dick have referred to
literature that suggests that Borrelia burgdorferi can cause demyelination as a sequel Lyme
disease.[18,19,20] They stated, “It seems inevitable that some patients, especially in area where

Lyme disease is endemic, who have been labeled as “possible MS”, will ultimately be shown to
have Lyme demyelinating encephalopathy.” They further suggested that, “A search for these
patients should be undertaken in endemic areas.”[18]
The endeavors reported here appear to have
supported their suggestions