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### Neurologists Take Heed Lyme Neuroborreliosis Mimicry is Afoot

*“Therefore, a Neurologist needs to always be cognizant of the fact that neurological manifestations, could be resulting from Neuroborrelial mimicry”*



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#### COLUMN ARTICLE

It has been stated, “Like its close spirochetal cousin *Treponema pallidum* (the bacterium that causes syphilis), *B. burgdorferi* can cause disabling neurologic manifestations and present a puzzling diagnostic challenge” [1].

Coyle noted that in Lyme disease, “neurological involvement occurs in up to 40% of symptomatic infections and includes both CNS and PNS involvement” [2].

The literature further states that, “Many healthcare practitioners mistakenly believe LD (Lyme disease) is not endemic to their state (in USA), causing them to omit the diagnosis from their differential or discount the patient’s concerns in this regard. Unfortunately, practitioners may not realize that LD has been found in every state. In states where *B. burgdorferi* carrying ticks are not highly prevalent, the ticks may be carried in on the bodies of birds, pets, wild animals, or people. Furthermore, practitioners often do not obtain a thorough patient travel history that might raise suspicion to test for LD” [1,2,3].

The literature also points out that, “Some LD patients are misdiagnosed with serious, untreatable chronic conditions (i.e. ALS, MS, Early Onset Alzheimer’s Disease, Epilepsy, Parkinson Disease) with no hope for recovery. When patients

are assumed to have an autoimmune disease, they are even put on corticosteroids to suppress their immune system, an unfortunate outcome for a patient with a chronic infection” [1,2].

The emotional trauma to patients and their loved ones that result when Lyme Neuroborreliosis has been misdiagnosed with an untreatable neurological disease is devastating!! Thus, the greatest effort must be made by a Neurologist to avoid such a misdiagnosis.

The noted country and western music star Kris Kristofferson has become a Poster Child for Lyme Neuroborreliosis which had misdiagnosed as Alzheimer’s Disease (CBSNews July 2. 2016: Kris Kristofferson’s Lyme disease misdiagnosed as Alzheimer’s).

In Virginia Savely’s monograph on the diagnostic dilemma posed by Lyme Disease, the author states that “patients are often labeled “atypical” in their disease presentation, meaning that their disease process greatly resembles the given diagnosis but is missing some of the diagnostic features. Anyone with an atypical presentation of any of the diseases discussed below should be thoroughly evaluated to rule out the possibility of disseminated LD” [1]. Therefore, a Neurologist needs to always be cognizant of the fact that neurological manifestations, could be resulting from Neuroborrelial mimicry [1-4].

The problems associated misdiagnosis as relates Lyme Disease were considered so serious a concern that, when the Department of Health of the Commonwealth of Pennsylvania (I was a member of this special committee) convened the special Task Force Committee on Tick and Lyme Disease in August of 2014 (the Pa Task Force group worked from Sept 2014 to Sept 2015). To create guidelines and recommendations on how the Pennsylvania deal with the ramifications associated with Lyme Disease and related tick borne infections, Misdiagnosis was discussed by the special task force committee and recommendations were put forward in order to promote a of general awareness for both the public and healthcare professionals on the long-term effects of misdiagnosis of Lyme Disease and its many serious ramifications.

Makhani., *et al.* have stated that “There is no “gold standard” diagnostic test for Lyme Neuroborreliosis. Direct culture of *Borrelia species* and PCR are of low sensitivity; therefore, laboratory diagnosis instead relies on the detection of anti-*Borrelia* antibodies [5]. In North America, testin follows a two-step algorithm. Serum samples are screene for antibodies with an ELISA, a relatively sensitive, but no specific test. Confirmatory testing is then performed using Western blotting, which is specific, but not sensitive. The sensitivity of the two-step approach is well described to increase in later stages of the disease for both European and North American acquired borreliosis. While sensitivity may be less than 40% in cases of acute stage 1 Lyme disease, both retrospective and prospective studies from New England have found the sensitivity of the two-step approach to be 85% to 100% in cases of stage 2 acute “Neuroborreli-

Dersche., *et al.* stated that, “Patients with LNB and neurosyphilis showed significantly higher CXCL13 levels in their CSF compared to MS patients ( $p < 0.05$ ,  $p < 0.001$ , respectively). CXCL13 levels in the CSF declined during treatment” [6].

Research investigations as relates to the development of accurate lab based assays as relates to improved early and accurate detection of Neuroborrelial infections and the ability to monitor the efficacy of therapy as a function of

changes in the pathogen load and eventual clearance of the pathogen from the patient are needed.

Borreliar pathogens like their cousin *Treponema pallidum* can mimic a variety of illnesses and can invade the CNS [3]. It is therefore incumbent upon all Neurologists to be ready to think “out of the box” when it comes to considering the possibility of a potential microbial agent as the “raison d’être” of Neuropathology whether one practices in a known endemic or non-endemic region for infections such as Lyme Neuroborreliosis which is commonly associated with tick bites, but can be also transmitted by other non-tick related vectors.

## BIBLIOGRAPHY

1. Savely V. “Lyme disease: a diagnostic dilemma”. *The Nurse Practitioner* 35.7 (2010): 44-50.
2. Markeljevic J., *et al.* “Tremor, seizures and psychosis as presenting symptoms in a patient with chronic lyme neuroborreliosis(LNB)”. *Collegium Antropologicum* 25 (2011): 313-318.
3. Coyle PL. “Lyme Disease”. In: Central Nervous System Infectious Diseases and Therapy. Karen L Roos and Marcel Dekker (1997): 213-236.
4. De Cauwer H., *et al.* “Motor neuron disease features in a patient with neuroborreliosis and a cervical anterior horn lesion”. *Acta Clinica Belgica* 64.3 (2009): 225-227.
5. Makhani N., *et al.* “A Twist on Lyme: The Challenge of Diagnosing European Lyme Neuroborreliosis”. *Journal of Clinical Microbiology* 49.1 (2011): 455-457.
6. Dersch R., *et al.* “The chemokine CXCL13 is elevated in the cerebrospinal fluid of patients with neurosyphilis”. *Fluids Barriers CNS* (2015): 12-12.

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