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Editorial

Lyme Disease, Chronic Fatigue and Fibromyalgia

Sam T Donta*
Department of Medicine, Falmouth Hospital, USA

***Corresponding author:** Sam T Donta, Department of medicine, Falmouth Hospital, USA

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Editorial

Lyme disease is caused by a bacterium named after its discoverer, Willy Burgdorfer [1]. In its initial characterization, it was found to be responsible for what was thought to be a form of juvenile rheumatoid arthritis [2]. As more information became available, it was appreciated that, while there can be an acute presentation of an expanding rash, with or without other non-specific symptoms, symptoms and or signs might not occur, or recur, weeks or months later. The disease was then described as occurring in stages, i.e. I-acute Lyme disease, II-neurologic or cardiac abnormalities, III-intermittent arthritis/late Lyme disease. Subsequently, it was appreciated that Lyme disease can span a spectrum from early Lyme disease, disseminated Lyme disease, and late Lyme disease, without distinct stages.

It is known that the Lyme spirochetes can be cultured from the initial typical erythema migrans rash and in the circulating blood over the subsequent 4-8 weeks [3]. At some point after that, it has not been possible to find the bacteria in any body fluid, despite the presence of various symptoms and signs. Serologic responses are currently the mainstay of laboratory diagnosis, and can be helpful when positive, indicating exposure to the organisms, but negative serologists cannot be used to exclude the diagnosis. The currently recommended 2-tier system, in which the Western Blot is done only if the initial screening test (e.g. ELISA, EIA), is positive, is flawed both because of the lack of sensitivity and specificity of screening tests. In addition, the requirement on Western Blotting for 5/10 IgG reactions or 2/3 IgM reactions, is not based on assessment of clinical patients who have persisting symptoms without objective signs. Those criteria were initially used for sero-epidemiologic purposes, and were never intended for clinical use, but became a standard for the laboratory diagnosis of Lyme disease in the absence of any other available test. Further analysis of the Western blotting results discloses that it is not the numbers of bands that are important, but whether there are any reactions to highly specific proteins of the causative borreliae, e.g. 23kd, 39kd, 83/93kd proteins [4]. Hence, if patients have one or more reactions to these highly specific proteins, it is likely that they have been infected by the Lyme bacteria. Additionally, the interpretations included in test results that if there are IgM reactions in patients with ongoing symptoms beyond the initial infection, these reactions should be considered false positives, even though there is no evidence that they are indeed false positives. It is illogical to, on the one hand, use IgM positivity to support the diagnosis of early Lyme disease, but then dismiss the IgM reactivity in patients with persisting or recurring symptoms as being false positive. It is likely that in patients who have IgM reactivity in relapsing illness have ongoing infection, in lieu of there being any currently available test or means to determine that the infection is still present or absent.

It was hoped that PCR-DNA technology would provide the needed sensitivity and specificity to support the clinical diagnosis, but that has not proved to be the case. There are untreated patients who have oligoarthritis who often have positive PCR-DNA tests for Lyme disease in their synovial fluid [5]. Even in those patients, culture of the fluid has been negative. It might be that differing DNA probes would provide greater sensitivity in various body fluids, but those probes have not been developed or tested as yet.

Patients with persisting or recurring symptoms present the greatest challenge to physicians in terms of diagnosis and treatment. These patients typically have ongoing symptoms, with few if any objective signs, similar to patients with chronic fatigue syndrome and fibromyalgia, and are frequently given those diagnoses or a diagnosis of post-Lyme disease. The use of the term post-Lyme disease implies that there had been adequate treatment of Lyme disease, and the ongoing symptoms, even if identical to those included in the original diagnosis, is not due to any persisting infection. This diagnosis is misleading, as previously stated, because there is currently no means to determine that the infection is or is not present. Cultures and PCR-DNA technology are not positive after the initial infection, and have not been positive during the course of ongoing disease; therefore one cannot reach the conclusion that the infection is no longer present. Similarly, serologic tests cannot be used to determine the presence or absence of the causative organisms. If and when there are actual means or tests to determine whether infection is or is not present, the diagnosis of persistent or recurrent Lyme disease, often termed chronic, rests on the clinical picture, and the results of treatments using effective regimens. There are many antibiotics that are effective in vitro, but some antibiotics are not clinically effective, for likely pharmacologic properties. Certain antibiotic regimens over longer periods of time do appear to be effective [6,7]. It might be that some persisting symptoms are due to residual auto-immune reactions, but that has not been yet been shown to be the case; even so, if what appear to be curative regimens resolve the symptoms, that would indicate that the underlying infection had been eradicated. Additional research will be needed to resolve these important questions.

Of considerable interest as well is the question whether patients who are diagnosed as having chronic fatigue syndrome or fibromyalgia might have Lyme disease as the underlying cause, given the overlap of symptoms. It would seem unlikely that most or all cases of these other disorders are due to Lyme disease, but, especially in Lyme-endemic areas, it would seem reasonable to evaluate patients who have chronic fatigue syndrome or fibromyalgia for the possibility that they have Lyme disease, using a combination of clinical criteria-including exposure history, laboratory tests, MRI of the brain-looking for white matter foci, brain SPECT scans [8], and treatment trials.

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