Correspondence

Pathogenesis of Post-Lyme Disease Symptoms

To the Editor—In an effort to understand the pathogenesis of post-Lyme disease symptoms, the article by Strle et al estimated the levels of 15 cytokines and an additional 11 chemokines in erythema migrans patients from Slovenia [1]. Their finding of elevated levels of the type 17 helper T-cell (T_{H17})–associated interleukin 23 (IL-23) in some of the patients with persistent symptoms is a valuable addition to attempts to understand the pathogenesis of the perplexing phenomena of post-Lyme disease symptoms following the apparently successful antibiotic treatment of Borrelia burgdorferi sensu lato (B. afzelii and B. garinii). The few markedly elevated levels of the type 1 helper T-cell chemokines CXCL9 and CXCL10 prior to receiving antibiotics were associated with the absence of symptoms on subsequent visits [1, Fig. 3A]. No other abnormalities in cytokines or chemokines were found. As noted in the Strle article, other instances of the stimulation of innate and adaptive immune response following Borrelia infection have been reported. More recently, Jacek et al studied patients with post-Lyme disease symptoms following treatment of the B. burgdorferi sensu stricto present in the United States [2]. They found that persistent symptoms were associated with activation of specific target genes of the innate immune system, suggesting increased interferon-alpha activity. However, the observations by Strle et al of normal levels of cytokines other than IL-23 considerably restricts the variety of cytokines likely to be involved with post-Lyme disease symptoms.

Still a complete understanding of the pathogenesis of post-Lyme disease symptoms remains an elusive goal. Strle et al found that 18/510 (3.5%) patients had symptoms that were present at 12 months. Perhaps, unfortunately, the severity and type of these symptoms were not discussed. As can be calculated from data in the article, 6 of the patients with persistent symptoms never had elevated IL-23 levels. Accordingly, other factors are likely to be involved, at least in some cases. Variations in the pathogenic potential of the bacteria, the genotype of the host, and symptoms unrelated to Borrelia infection are plausible contenders. Precedence for variation in virulence of Borrelia strains exists in the propensity for hematogenous dissemination during early Lyme disease. J Infect Dis 2014; 58:372–80.

Note

Potential conflicts of interest. All authors: No potential conflicts of interest. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

References


Correspondence: Stephen J. Seligman, MD, Department of Microbiology and Immunology, New York Medical College Valhalla, 10595 (stephen_seligman@nymc.edu).

Clinical Infectious Diseases 2014:59(5):747
© The Author 2014. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: permissions@oup.com.
DOI: 10.1093/cid/ciu341