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Autoimmune Disorders following Lyme disease

Dr. Sheila Arvikar is a physician scientist in the Division of Rheumatology, Allergy, and Immunology and Center for Immunology and Inflammatory Diseases at the Massachusetts General Hospital (MGH), and an Instructor of Medicine at Harvard Medical School. She is a graduate of the University of Massachusetts Medical School. She completed her internal medicine and rheumatology training at MGH. She joined Allen Steere’s research group during her rheumatology fellowship training. She now staffs the MGH Lyme arthritis clinic with Dr. Steere and is dedicated to improving the clinical outcomes of patients with Lyme arthritis. Her translational and clinical research interests include infectious triggers of rheumatoid arthritis, autoimmunity in Lyme disease, treatment and outcomes in Lyme arthritis, and imaging of Lyme arthritis. She has received numerous awards for her research including grants from the Arthritis Foundation and the American College of Rheumatology-Rheumatology Research Foundation.
One of many challenges in Lyme disease is the symptoms which may persist despite treatment. Musculoskeletal complaints are prominent among these symptoms, and may range from arthralgia (joint pains without inflammation) seen in Post-Treatment Lyme disease Syndrome to frank arthritis with inflammation and swelling as in Lyme arthritis. There may be multiple mechanisms for these phenomena, including immune dysregulation and autoimmunity. With Lyme arthritis, an immune-mediated inflammatory arthritis may persist after antibiotic treatment. However this is usually confined to a single previously infected joint, without systemic symptoms.

We have recently described a cohort of 30 patients who developed new-onset rheumatoid arthritis, psoriatic arthritis or peripheral spondyloarthropathy, a median of 4 months after antibiotic treatment for Lyme disease. In the majority, the rheumatic disease followed an early manifestation of Lyme disease such as erythema migrans. These patients often had distinguishing clinical features from Lyme arthritis patients including family history of autoimmunity, involvement of many joints, involvement of the spine and entheses (tendon insertion sites), and the onset of skin psoriasis. They also had distinguishing laboratory features from Lyme arthritis patients such as rheumatoid arthritis-specific biomarkers, significantly lower titers of antibodies to *B. burgdorferi*, and lower frequency of Lyme-associated autoantibodies. The patients were treated with typical inflammatory therapies which are the standard of care for these diseases, resulting in improvement. In addition to this cohort, we have seen other types of systemic autoimmune diseases follow Lyme disease including systemic lupus and thyroid disease. Autoimmune neurologic conditions have also been reported.

Although systemic autoimmune diseases may follow Lyme disease
by chance, onset within months suggests that *B. burgdorferi* infection may be a pro-inflammatory trigger. It is important for clinicians to be aware of this possibility when evaluating patients with post-infectious symptoms. Although this may not be a common outcome, given that Lyme disease is now epidemic in parts of the U.S., awareness of autoimmune disease in the spectrum of post-Lyme syndromes is essential in preventing delays in appropriate diagnosis and treatment.