Antiphospholipid Antibody Syndrome and Lyme Disease: A Possible Association

Kevin J. Cross, BS and Michael A. Patmas†, MD, MS, FACP*

ABSTRACT

Antiphospholipid Antibody Syndrome (APS) and Lyme disease both result in affected patients having elevations in anticardiolipin antibodies (ACAS). The literature suggests that these antibodies lead to characteristic clinical findings in APS only, and have no clinical significance in Lyme disease. We present a patient who had symptoms suggestive of APS and elevated ACAS levels to support the diagnosis. Her antibody levels decreased upon treatment for Lyme disease, however, suggesting the symptoms were actually a result of a Lyme disease related elevation in her ACAS levels. The possible association between Lyme disease and APS warrants further research.

Key words: Lyme disease, Antiphospholipid Antibody Syndrome, anticardiolipin antibodies

Lyme disease is a multisystem infection caused by the spirochete *Borrelia burgdorferi*. Lipids on the surface of *B burgdorferi* may cross-react with IgM and IgG anticardiolipin antibodies (ACAS), causing elevations in the levels of one or both of these antibodies on ELISA screens.[1] Antiphospholipid Antibody Syndrome (APS), an autoimmune mediated disease that is distinct from Lyme disease in its clinical presentation, also results in the elevation of these ACAS. In APS, the elevated level of ACAS has been linked to its clinical manifestations, which include cerebrovascular changes, thrombotic events, and spontaneous and recurrent abortions.[2] To date, this has not been found to be the case when ACAS are high in patients with Lyme disease.[3] Herein, we report a case of a patient with a longstanding documented diagnosis of APS in whom both elevated ACAS as well as clinical evidence of the disease existed. The patient was later found to have Lyme disease and underwent treatment for it. Surprisingly, during her antibiotic therapy, her ACAS fell to within normal limits, suggesting that these levels were most likely elevated because of the presence of *B burgdorferi* antigens and thus not a result of APS. This being the case, it is also likely that her clinical symptoms, originally attributed to APS, are also a result of Lyme disease related elevations of ACAS.

*From the *University of Pennsylvania School of Medicine, †University of Pennsylvania Health System—Clinical Care Associates, Philadelphia, Pennsylvania.*

Address correspondence to: Michael A. Patmas, MD, MS, FACP, Anchorage Square, 1749 Hooper Avenue, Suite 101, Toms River, NJ 08753.

CASE REPORT

A 37-year-old white female was referred for treatment after she was found to have a positive Western blot, which met the Center for Disease Control and Prevention criteria for the diagnosis of Lyme disease. The patient reported the history of a successful pregnancy and delivery of a healthy child. A few years later two episodes of fetal wastage and miscarriages occurred, despite Heparin therapy during pregnancy. At that time, after testing positive for elevated levels of IgM and IgG ACAS, she was given the diagnosis of APS and referred to a hematologist for evaluation of the disease. Her hematologist tested her and found her to have Lyme disease.

Upon our questioning, the patient reported no recollection of a specific tick bite or erythema migrans rash and did not show clinical signs or symptoms of Lyme disease including joint pain, headaches, confusion, arthralgias,

myalgias, or disabling fatigue. She did, however, have many risk factors for its acquisition. She grew up in and now lives in areas endemic for Lyme disease and has family members in other areas endemic for the disease. Her husband works as a biologist in a nearby state park and has had as many as 100 deer ticks on him after coming home from work. He very likely could have acted as a vector for the transmission of the tick to the patient.

The physical examination revealed a very pleasant, healthy appearing woman. Her vital signs were stable and within normal limits. Her head, ears, eyes, nose, and throat examinations were normal. Her cranial nerves were intact and her neck was supple and without lymphadenopathy. The pulmonary examination was normal. On cardiac examination, normal S1 and S2 were heard without evidence of a gallop, murmur, or rub. Her abdominal exam was normal without organomegaly, masses, or tenderness. Her extremities were grossly normal. There was no evidence of synovitis or effusions, and her joints were mobile and without pain. Neurologic examination did not reveal any lateralization, and her cutaneous examination was normal.

A laboratory evaluation resulted in a normal complete blood count. The patient is blood type O and Rh negative. Her coagulation profile was normal. Our blood test found her antiphospholipid IgM levels to be positive at 13 MPL units and her Lyme ELISA IgM and IgG levels to be at an index of 1.33 and 1.37, respectively. She was Lupus anticoagulant negative, rheumatoid factor negative, micro somal antibody negative, and antinuclear antibody negative. Although her Lyme Western blot IgG was negative, her IgM Western blot was positive with bands noted at 66, 41, and 23 kd.

Based on the epidemiological risk factors and laboratory results, the diagnosis of infection with *B burgdorferi* was made. After declining intravenous antibiotic treatment the patient was started on intramuscular Bicillin injections at 1.2 million units IM every four weeks. Other than a severe Jarisch-Herxheimer reaction, the drug therapy was tolerated well. After four months of IM injections, the patient's anticardiolipin IgM fell to 7 MPL units, and after seven months of treatment, it was normal at 5 MPL units. The IgM antibody levels have remained negative.

DISCUSSION

The literature states that patients whose anticardiolipin antibody levels are elevated as a result of Lyme disease, rarely, if ever, present with clinical symptoms like those found in patients with APS elevated anticardiolipin antibody levels.[4] In our patient, given her history and presentation, this may not be the case. She had a healthy, full-term delivery followed by two miscarriages. These miscarriages were temporally associated with elevated ACAS. When the patient was later diagnosed with and treated for Lyme disease, her anticardiolipin antibody levels fell to within normal limits. Because of this, we believe if indeed her miscarriages were a result of her high ACAS, Lyme disease was the cause rather than APS. Furthermore, we propose that like our patient, other patients currently carrying the diagnosis of APS may instead be suffering from Lyme disease masquerading as APS. Further study should be done on the possible association between Lyme disease and the symptoms of APS. This could include the use of Lyme disease screening in diagnosed APS patients who live in areas endemic for Lyme disease, or the use of tests that distinguish autoimmune ACAS from nonautoimmune ACAS.[2,5]

REFERENCES

1. Mackworth-Young CG, Harris EN, Steere AC, Rizvi F, Malawista SE, Hughes GRV, Gharavi AE. Anticardiolipin antibodies in Lyme disease. Arthritis Rheum 1998;31(8):1052-1056.

2. Goel N. Antiphospholipid antibody syndrome: current concepts. Hosp Pract 1998;33(2):129-140.

3. Duray PH, Steere AC. Clinical pathologic correlations of Lyme disease by stage. Ann N Y Acad Sci 1998;539:65-79.

4. Garcia Monco JC, Wheeler CM, Benach JL, Furie RA, Lukehart SA, Stanek G, Steere AC. Reactivity of neuroborreliosis patients (Lyme disease) to cardiolipin and gangliosides. J Neurol 1993;117:206-214.

5. Roubey RA. Immunology of the Antiphospholipid Antibody Syndrome. Arthritis and Rheum 1996;39(9):1444-1454.