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Reply to the Letter by Fern’s i Tortajada et al.: Relapsing Neuroborreliosis

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Ferris i Tortajada et al. present an interesting case of seronegative Lyme disease. Their letter, however, does not provide documentation for ongoing infection after treatment, which is essential for the diagnosis of chronic Lyme disease.

The HLA Dr 2, DQW1 association [1] was the only connection with the treatment-refractory neuroborreliosis in our patient. She had no history of any other unusual or frequent infections. Her humoral immune system appeared intact, as judged by normal results of the following tests: quantitative immune globulins; IgG subclasses 1–4; normal absolute lymphocyte count and CD3, CD4, CD8 profile; and she developed antibodies to hepatitis B following four vaccine injections; isohemagglutinins titers were normal. Her cellular immunity also appeared intact inasmuch as the diagnosis of borreliosis was made by finding a strongly positive *Borrelia burgdorferi* blastogenesis test, with a stimulation index of 50 [2]. Although the patient was anergic when symptomatic, after 4 months of therapy with clarithromycin her skin tests to TB, Candida and mumps were again positive.

A brief further follow-up of the patient reported in our paper may be of interest. After starting clarithromycin in August 1992, the patient improved steadily and, by February 1993, became asymptomatic without new neuromuscular or sensory symptoms.

As a prelude to discontinuing antibiotics, in January 1994 we obtained a repeat lumbar puncture (No. 9) in which the CSF protein was 68 mg°/o. The PCR of the CSF was negative [Godfroid, personal commun.] but a Western blot of the fluid revealed a prominent band of *B. burgdorferi* OspA and a strongly positive antibody capture ELISA for OspA [3]. We considered this as evidence for ongoing infection and continued clarithromycin. In January 1995 her CSF (No. 10) had a protein of 60 mg% and was still positive for OspA by ELISA and Western blot. In April 1995 the patient developed a partial right central facial weakness. In May 1995 she was hospitalized for the 7th time, was premedicated with intravenous solumedrol and motrin, and was given 2 g of ceftriaxone daily for 10 days. Within 24 h she had a mild Herxheimer reaction but was well enough to go home by day 4. Following the ceftriaxone she was started on azithromycin 250 mg daily. She has remained well since initiation of azithromycin therapy and every 4th month receives 4 daily doses of 2 g of ceftriaxone i.v.

We emphasize the importance of documenting active infection in patients being considered for retreatment for Lyme disease. In our patient, the OspA positivity by ELISA and Western blot, and the Herxheimer reactions after intravenous therapy, provide evidence of ongoing infection despite continued antibiotic therapy.

References

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