REINFECTION WITH BORRELIA BURGDORFERI

SIR,—Lyme borreliosis, a tick-borne multisystem disorder caused by the spirochaete *Borrelia burgdorferi*, typically begins with erythema chronicum migrans and is sometimes followed by involvement of the heart, joints, and nervous system.¹ Neurological manifestations include meningoradiculitis (Bannwarth's syndrome), meningitis, and encephalitis.²-⁴ The clinical diagnosis can be confirmed by finding antibodies to *B burgdorferi* by indirect immunofluorescence or ELISA.56 We describe here a serologically and bacteriologically confirmed case of possible reinfection.

In September, 1983, 2 weeks after a tick bite on the right arm, a 59-year-old woman had erythema migrans around the bite site. She presented on Nov 15 with a painful meningoradiculitis and bilateral papilloedema. CSF analysis showed a lymphocytic pleocytosis (330 cells/µl) and an increase in total protein (82 mg/dl). Her serum IgG antibody titre against *B burgdorferi* rose from less than 16 to 256 within 4 weeks (figure). IgM antibodies were not detected, and borreliae could not be isolated from the CSF. The patient was treated with intravenous penicillin (20 megaunits daily for 5 days, then 10 megaunits daily for 5 days) and oral methylprednisolone (70 mg daily over 2 weeks with decreasing dosage). On discharge (Dec 12) she was free of pain, with a slightly improved vision. Follow-up examinations showed residual bilateral papillatrophy. In October, 1984, the CSF was normal (1 cell/µl, total protein 37 mg/dl).

On Oct 2, 1985, the patient visited a forest. She did not recall any arthropod bites but on Oct 5 a painful redness developed on the right side of her chest. She presented on Oct 7 with an erythema chronicum migrans, about 20 × 14 cm. Spirochaetes were isolated at biopsy of the skin around the lesion after 4 weeks' incubation in modified Kelly's medium. Her serum IgG antibody titre against B burgdorferi had been 16 on June 27, 1985 but had risen to 64 on Oct 17 without a corresponding IgM increase. Treatment with minocycline 200 mg daily by mouth for 14 days was successful.

In 1958 Hollström reported a case of recurrent erythema migrans 5 years after primary infection. Sköldenberg et al in 1983 described a patient with two episodes, 20 years apart, of erythema migrans and meningitis after a tick bite. Serological tests were not done on these patients. In Weber and colleagues' two patients with erythema migrans and reinfection after 5 and 7 years antibody titres against B burgdorferi did not increase.

In 1983 our patient had Lyme borreliosis with Bannwarth's syndrome. Her serum IgG titre against *B burgdorferi* became negative within 10 months. Erythema chronicum migrans recurred 2 years later. The absence of IgM antibodies in acute manifestations of Lyme borreliosis has been described before. The 1985 episode presented after the patient had stayed for a long time in a forest in an area known to be endemic for *B burgdorferi* infection. Despite the absence of a known tick bite we think that the second attack was a reinfection and not a recurrence. The skin sites involved were different in the two episodes.

Patients with Bannwarth's syndrome usually retain a significant IgG titre against *B burgdorferi* for several years. Our patient's antibody titre was insignificant by 10 months. The antibiotic and/or corticosteroid treatment given for that first attack may explain the short-lived immunity, permitting reinfection after only 2 years.

Departments of Neurology and Dermatology, University of Munich, Klinikum Grosshadern, 8000 Munich 70, West Germany; and Max von Pettenkofer Institute for Hygiene and Medical Microbiology, University of Munich

HANS-WALTER PFISTER
UWE NEUBERT
BETTINA WILSKE
VERA PREAC-MURSIC
KARL MAX EINHÄUPL
GIAN DOMENICO BORASIO

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