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J Oksi M Marjamäki K Koski J Nikoskelainen M K Viljanen

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Bilateral facial palsy and meningitis caused by borrelia double infection

SIR—Facial nerve paresis, especially bilateral facial palsy, is a well-known manifestation of Lyme borreliosis. Patients may have meningeal involvement with mild pleocytosis and other inflammatory signs in the cerebrospinal fluid (CSF). *Borrelia burgdorferi* has been cultivated from the CSF and blood of patients with facial palsy.[1] Three genospecies of *B burgdorferi* sensu lato occurring in Europe are now known to cause human infections.[2,3] We describe simultaneous detection of two genospecies by culture in a single patient.

A 52-year-old previously healthy man living in an area where Lyme borreliosis is endemic removed an engorged tick from his thigh in the autumn of 1993. No erythema migrans developed. During the first months of 1994, persistent myalgia developed. Anti-inflammatory drugs and physiotherapy provided only marginal relief. In the summer of 1994, he had intermittent photophobia. On Aug 12, 1994, the patient suddenly developed bilateral facial palsy without any other new symptoms. The palsy disappeared completely in 2 weeks. However, daily episodes of myalgia persisted. On Aug 31, 1994, CSF analysis revealed lymphocytic meningitis (table) with greatly raised protein (with 3–4 subfractions) and an increased IgG index (0·88; normal value <0·60). CSF angiotensin-converting enzyme was normal, whereas lysozyme was raised (0·61; normal value <0·1). Magnetic resonance imaging of the brain was unremarkable. Results of antibodies against *B burgdorferi* and of PCR tests (plasma, CSF, Bardour-Stoenner-Kelly II [BSK-II] medium) are shown in the table. On Sept 12, 1994, treatment with cefixime (200 mg orally every 8 h) combined with probenecid (500 mg orally every 8 h) was instituted and continued for 100 days. While on antibiotics he developed mild bilateral iritis. The myalgia resolved in 1 month. CSF and serum samples on Dec 30, 1994, 1 week after the end of antibiotic therapy, showed only borderline abnormalities (table).

	Aug 12, 1994	Aug 31, 1994	Dec 30, 1994
Serum antibodies against:			
B burgdorferi (ss, whole sonicated, ELISA)			
IgM	..	Pos	Neg
IgG	..	Neg	Neg
B burgdorferi flagellin*			
IgM	..	Borderline	Neg
IgG	..	Neg	Neg
PCR† for *B burgdorferi* from plasma	Neg	Pos	Neg
CSF results			
CSF lymphocytes	..	474	7
CSF protein (mg/L)‡	..	2250	763
CSF antibodies against *B burgdorferi* flagellin§ (index)	..	Pos (14·4) for IgM, neg for IgG	Borderline*
PCR† for *B burgdorferi* sl from CSF	..	Neg	Neg
CSF culture in BSK-II medium	..	Pos	Neg
PCR from BSK-II medium			
For *B burgdorferi* sl†¶	..	Pos	..
For *B burgdorferi* ss¶	..	Neg	..
For *B garinii*¶	..	Pos	..
For *B afzelii*¶	..	Pos	..

ss=sensu stricto, sl=sensu lato, Pos=positive, Neg=negative, others not tested. *Lyme borreliosis ELISA kit, 2nd generation, DAKO A/S, Glostrup, Denmark; †with primers for DNA fragment from flagellin gene; ‡normal value 290–665 mg/L; §DAKO ELISA, Lyme neuroborreliosis kit, Glostrup, Denmark, indices above 0·3 show intrathecal antibody production; ¶with four primer pairs selected from gene encoding 16S rRNA of *B burgdorferi*.[4]

Table: **Laboratory findings**

Our patient had concomitant, culture-proven infections with two genospecies of *B burgdorferi* sensu lato, *B garinii* and *B afzelii*. PCR-based evidence of simultaneous infection by two or even three genospecies of *B burgdorferi* sensu lato has been reported.[3] We have found several local ticks harbouring more than one genospecies of *B burgdorferi* sensu lato (unpublished). It is possible that double infections with borreliae are not very rare, and could be transmitted even by the bite of a single tick. It has been suggested that the three European genospecies, *B burgdorferi* sensu stricto, *B garinii*, and *B afzelii*, have different organotropisms.[5] *B garinii* has been proposed as the main causative agent of Lyme neuroborreliosis.[3] It is impossible to say which one of the genospecies in our case was responsible for the clinical disease, or whether both were responsible. CSF and blood may give positive PCR results only transiently or intermittently. The reason for this might be the minimal amounts of spirochaetes and their degradation products occurring sporadically in the CSF and blood. It is also possible that spirochaetes are shed to the circulation only intermittently from their hiding places. Both possibilities stress the importance of repeated testing. The first symptom of infection in our patient was persistent myalgia. Transient photophobia, occurring 2 months before definitive diagnosis, was obviously an ocular manifestation of Lyme borreliosis. It is remarkable that the patient, with marked pleocytosis in the CSF, had no meningeal signs or headache. Without the transient bilateral facial palsy 1 year after onset of the infection, the patient's central-nervous-system infection would have remained unrecognised and progressed towards more serious sequelae. On the basis of this case, we advocate analysing the CSF of patients even when symptoms are mild.

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J Oksi, M Marjamäki, K Koski, J Nikoskelainen, M K Viljanen
Department of Internal Medicine, Turku University Central Hospital; *Department of Medical Microbiology, Turku University, FIN-20520 Turku, Finland; and National Public Health Institute, Department in Turku; and Pulssi Neurocentre, Turku

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