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CASE REPORT

Lyme borreliosis associated with complete flaccid paraplegia

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Summary

We report the case of a patient with concomitant Lyme borreliosis and acute paraplegia. The paraplegia was complete, flaccid and of upper motor neurone type. The diagnosis of borreliosis was based on the detection of large amounts of IgM and IgG borrelia antibodies in the acute phase serum and on the complete disappearance of IgM antibody during the review period. IgG borrelia antibodies were also detected in the CSF, but leakage of antibodies from the blood to the intrathecal space could not be ruled out. Lymphocytosis and increased total protein concentration in the CSF were signs compatible with neuroborreliosis. Ceftriaxone therapy effected dramatic recovery of the patient. This case suggests that borreliosis should be considered a possible cause of acute flaccid paraplegia.

Introduction

Neurological manifestations are a prominent feature of Lyme borreliosis. Cranial neuritis, aseptic meningitis and polyradiculitis (Bannwarth's syndrome) are typical subacute presentations of the disease. Late neurological manifestations are extremely varied.[1] Dementia, encephalomyelitis, and peripheral nervous system involvement have been described frequently.[2] In contrast, only sporadic cases of transverse myelitis and pure motor paraparesis have been reported.[3–7] In these few cases, the course of the myelitic disease has usually been subacute or chronic, and paraparesis has always been spastic.[8] The prognosis of transverse myelopathy is generally poor, especially if tendon reflexes and posterior column function are absent.[9]

We describe a patient with concomitant occurrence of Lyme borreliosis and acute, flaccid and total paraplegia. Although the patient presented with poor prognostic signs, he recovered rapidly and completely after antibiotic treatment.

Case report

On March 13 1989, a previously healthy 72-year-old Finnish man was admitted to the neurology outpatient clinic of Turku University Hospital, because of increasingly impaired gait. The symptoms had appeared 7 days earlier. The patient did not recall any tick bites or erythema migrans.

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However, he lives in an area where *Ixodes ricinus* is found and borreliosis is endemic.

Neurological examination revealed extensor plantar responses, brisk patellar reflexes and absent ankle jerks. All sensory qualities were decreased up to the level of T 10. The patient could stand but not walk, and he had urinary retention. Examination of the cranial nerves and the upper extremities was normal. Both thoracic and cervical myelography, as well as thoracic magnetic resonance imaging, were normal. The ESR was slightly elevated (32 mm/h, normal < 20 mm/h). The CSF analysis revealed increased total protein concentration (1100 mg/l, normal range 525 to 790 mg/l); and lymphocytic pleocytosis (30×10^6/l). The protein increase was mainly due to increased albumin, indicating impairment of the blood-CSF barrier. Total IgG concentration was slightly raised (90 mg/l, normal < 40 mg/l). The IgG index between serum and CSF was normal, and no oligoclonal IgG could be demonstrated in the electrophoresis of the CSF. Virus serological tests from serum and CSF were negative.

By March 16, the patient had developed a complete flaccid paraplegia with absent tendon reflexes and extensor plantar responses. He was unable to move his legs in the lying position. A working diagnosis of transverse myelitis was made, and dexamethasone (4 mg four times daily i.m.) was started. During the next week, the patient's condition improved. He was able to lift his legs in the lying position and stand when assisted, but he could not walk.

On March 17, serum and CSF specimens were collected for estimation of IgM and IgG antibodies to *Borrelia burgdorferi* using two EIA systems: One used sonicated borrelia organisms as antigen;[10] the other was a commercial EIA, with 41 kDa flagellar protein as antigen (Dako Lyme Borreliosis Kit; Dakopatts A/S, Glostrup, Denmark). Both systems revealed considerable amounts of IgM and IgG antibodies in the serum, indicating active borreliosis (Table I). IgG antibodies were also detected in the CSF, using both EIA systems (Table I). However, quantitative comparison of amounts of antibody in the serum and CSF was not possible. Thus, leakage of antibodies from the blood to the intrathecal space cannot be ruled out as an explanation of the presence of antibodies in the CSF. Lues serology (VDRL) yielded a negative result from serum and CSF.

The patient's motor and sensory nerve velocities and electromyography were normal. Somatosensory evoked potentials (SEPs) from the upper extremities with median nerve stimulation were normal. In contrast, no somatosensory potentials could be evoked from the lower extremities with tibial nerve stimulation. This finding indicated a lesion in somatosensory pathways from the lower extremities.

On March 23, ceftriaxone (2 g twice daily i.v.) was started and continued for 2 weeks. The treatment's favourable effect on the patient's clinical condition was almost immediate. On March 26, dexamethasone was discontinued. After 2 weeks ceftriaxone, the patient was able to walk and climb stairs without difficulty. Plantar reflexes were flexor. He still had some hyperaesthesia in the lower extremities. The neurological defect of longest duration was urinary retention which lasted a month and required intermittent self-catheterisation.

On April 4, the ESR was 8 mm/h. The CSF protein concentration was

Table I *IgM and IgG antibodies against* Borrelia burgdorferi *in the serum and CSF specimens of the patient, as measured by EIA systems with sonicated antigen (S-EIA) or 41 kDa flagellar protein antigen (F-EIA).*

		IgM		IgG	
Date	Specimen	S-EIA EIU*	F-EIA OD_{405}†	S-EIA EIU	F-EIA OD_{405}
17/3/89	Serum‡	75 (++)	1·263 (+++)	114 (+++)	1·470 (+++)
04/4/89	Serum	68 (++)	1·036 (++)	98 (+++)	0·761 (+)
31/1/90	Serum	0	nd	105 (+++)	nd
17/3/89	CSF§	0	0	37	0·191
04/4/89	CSF	0	0	3·6	0·091

nd, not determined.

Grading of antibody levels in the parentheses: +, low positive; ++, medium positive; +++, strongly positive.

* EIU is a relative enzyme immunoassay unit, where 1 EIU is 1:100 of the corresponding antibody concentration in the reference serum. The positivity limit for both IgM and IgG antibodies was 30 EIU. This level was the mean+2 S.D. of a control material of 100 healthy persons.

† The positivity limit, stipulated by the manufacturer of the test kit, was OD_{405} 0·500.

‡ Serum specimens were assayed at a dilution of 1:100.

§ CSF specimens were assayed at a dilution of 1:10.

normal (390 mg/l), and there was no lymphocytic pleocytosis. Serum concentrations of IgM and IgG borrelia antibodies, especially those measured by the EIA with flagellar antigen, were less than those determined before the onset of antimicrobial therapy (Table I). Nevertheless, with both EIA systems, positive results were observed. In contrast, IgG antibodies in the CSF were almost undetectable (Table I).

On January 31 1990, the patient was examined again. He was asymptomatic, and neurological examination revealed normal function. Amounts of borrelia antibodies in the serum were estimated only by the sonicate EIA. IgM antibodies were no longer detected, whereas IgG antibodies were present in the same amount as that observed in the acute phase serum more than 10 months earlier (Table I).

Discussion

This case further supports earlier findings that neuroborreliosis may present as transverse myelopathy. Its noteworthy aspect was flaccid and rapidly progressive paresis. In the patients reported previously, the paresis had been spastic; and the progress of disease mostly subacute or chronic.[3,4,5,7] In only one of these cases had it evolved as rapidly as in our patient.[6] Even so, in that case, the paresis was spastic.

Our patient's symptoms appeared in March which suggests that the primary infection may have occurred more than 6 months earlier. Thus, his symptoms obviously represent late manifestations of borreliosis which are usually chronic or subacute and often less dramatic than when they occur early in the disease.[11]

There is no doubt that the patient had Lyme borreliosis. Two EIA systems revealed large amounts of IgM and IgG antibodies in the acute phase sera. The complete disappearance of IgM antibodies during the review period further confirmed the diagnosis. Nevertheless, the evidence for neuroborreliosis was circumstantial, since the present study protocol did not allow confirmation of the intrathecal origin of IgG borrelia antibodies detected in the CSF. Lymphocytosis and increased protein concentration in the CSF are compatible with neuroborreliosis, but not specific to it.

The dramatic favourable effect of antimicrobial therapy on the neurological functions and laboratory parameters is, however, difficult to explain by mechanisms other than direct destruction of the bacteria infecting the nervous system. Immunopathological processes triggered by the spirochaetes certainly play a role in the rapid development of clinical symptoms.[12] The moderate improvement observed after dexamethasone may have resulted from inhibition of local inflammatory reactions caused by immune attack against the causative organisms.

The prognosis of transverse myelopathy is generally poor, and treatment has only marginal effect on the outcome.[13] Especially poor is the prognosis of those patients who retain no tendon reflexes and posterior column function.[9] This was the case in our patient, yet he recovered rapidly after receiving antimicrobial therapy. Clinicians should consider borreliosis as a potential cause of paraplegia which may show rapid and complete response to treatment.

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