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MRI in Lyme disease of the spinal cord

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Abstract We report a case of Lyme myelitis in a 31-year-old man, presenting with a conus medullaris syndrome. MRI demonstrated contrast enhancement on the pial surface of the lower thoracic cord and conus medullaris. Elevated blood immunoglobulins and IgM antibodies against *Borrelia burgdorferi* in the cerebrospinal fluid (CSF) were found. Leptomeningitis may be the first stage of spinal infection in Lyme disease, preceding parenchymal infection leading to myelitis. Vasculitis is probably the major mechanism. MRI findings are nonspecific and the diagnosis is given by serum and CSF analyses. Early treatment with antibiotics and high doses steroids may result in complete recovery, as in this case.

Keywords Myelitis · Leptomeningitis · Lyme disease · Magnetic resonance imaging

Introduction

Lyme disease is a multisystem infectious disease transmitted by the tick-borne spirochaete *Borrelia burgdorferi (B. burgdorferi)*. The nervous system is involved in 15% of cases. Neurological symptoms are extremely variable and diagnosis is often difficult, especially since a history of a tick-bite is lacking in the majority of cases. Bell's palsy, cranial neuropathies, encephalomyelitis, radiculopathies, meningitis and transverse myelitis have been described. We report the clinical and imaging findings of an infectious myelitis due to *B. burgdorferi* in a adult.

Case report

A 31-year-old white man developed a sore throat with headache, fever and lethargy, but no cutaneous abnormality. He then complained of painful paraesthesiae of both legs. He presented 1 month later with marked muscle fatigue, backache and dysuria, with sphincter disturbances. Examination showed weakness of the legs, hyperreflexia and extensor plantar reflexes. There was no upper limbs or cranial nerve abnormality. The clinical findings were consistent with a conus medullaris syndrome.

MRI was performed at 1.5 tesla, using phased-array coils. Sagittal T2- and T1-weighted images showed no abnormality (Fig. 1 a), but contrast enhancement on the pial surface of the lower thoracic cord and conus medullaris was seen on sagittal and axial images (Fig. 1 b, c). The findings were consistent with leptomeningitis of the conus medullaris and lower thoracic cord. The brain

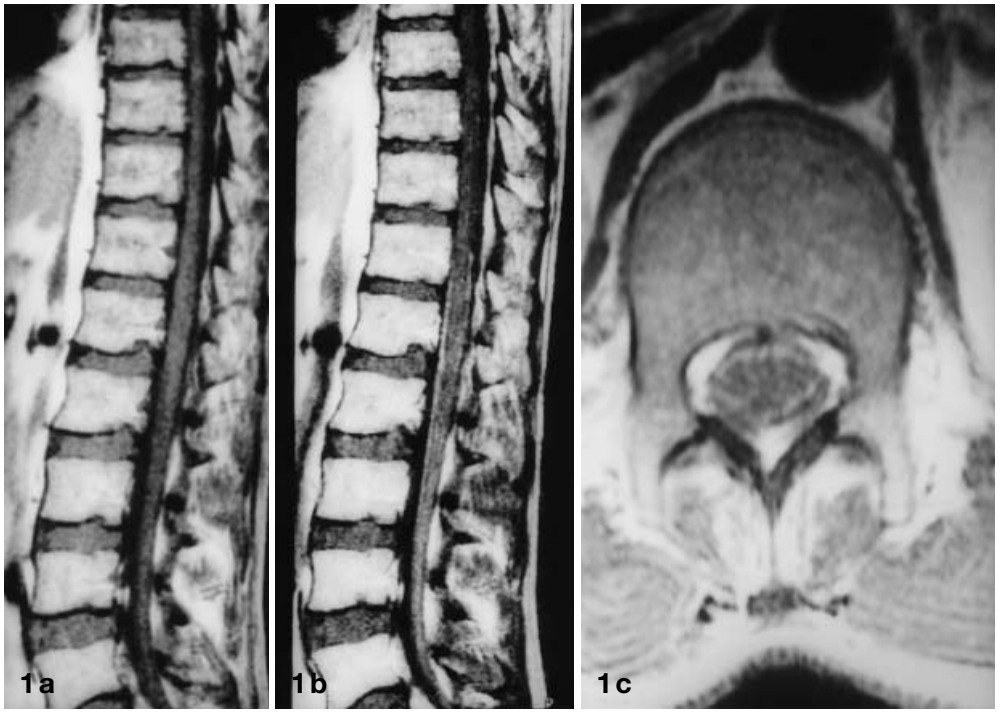

Fig. 1 **a** Sagittal T1-weighted image showing no abnormality. **b** Sagittal and **c** axial contrast enhanced images of the thoracolumbar spine, showing strong leptomeningeal enhancement on the surface of the lower thoracic cord and conus medullaris

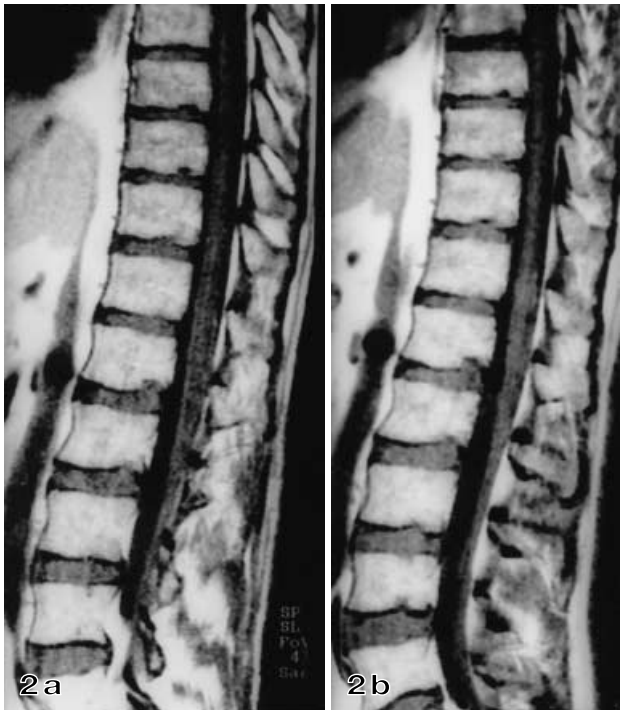

Fig. 2 **a** Sagittal T1 weighted contrast-enhanced image 2 months after Fig. 1, showing significant decrease in the leptomeningeal enhancement. **b** A similar image at 6-month follow-up, showing no abnormality

was not examined at that time because of the absence of relevant symptoms or signs.

Blood sedimentation rate was normal at 4 mm/h. Cerebrospinal fluid (CSF) obtained at lumbar puncture showed a pleocytosis with 54 white cells, elevated total protein of 1.96 g/l and normal glucose 3.2 mmol/l. HIV serology was negative. Elevated blood immunoglobulins and IgM antibodies against *B. burgdorferi* were found in the CSF, indicating intrathecal production. Total immunoglobulin in serum was 1/512. The clinical picture, MRI findings, serology and CSF examination were consistent with Lyme myelitis due to *B. burgdorferi*.

Treatment with antibiotics (Ceftriaxone) and high-dose corticosteroids was given for 2 weeks and the patient was discharged. His course was favourable and 2 months later the neurological ex-

amination was normal. The blood showed persistently positive serology. MRI showed a significant decrease in the leptomeningeal contrast enhancement (Fig. 2a); T2-weighted images were again normal. MRI 6 months later was completely normal (Fig. 2b); the patient was symptom-free and his serology was normal.

Discussion

Lyme borreliosis is an infectious disease transmitted from an animal reservoir to humans through a tick-bite. It was first recognised in 1909 by Afzelins [in 1], who described erythema chronicum migrans, a skin lesion caused by a tick-bite. In 1922, Garin-Bujadoux [2] described a case of meningoradiculoneuritis, the first recorded neurological manifestation, known as Bannwarth syndrome. It was not until the 1970s that the disease was recognised as a nosological entity with neurological, rheumatological and cardiac components. The name Lyme disease derives from the town in Connecticut where a juvenile rheumatoid arthritis was described. In 1983, a spirochaete was identified in a tick in the United States; it was found by culture and serological studies to be the organism causing Lyme disease [3,4].

The disease is world-wide. In the northern hemisphere, most infections occur during the warm season, from May to July. In 60–80 per cent of cases, a skin lesion at the site of a tick-bite is the initial manifestation. Diagnosis is not difficult during the summer season where the disease is endemic and/or when all clinical manifestations are present, but in many cases, the cutaneous lesion is unnoticed or forgotten.

Lyme disease has been compared with other spirochaetal infections of the nervous system, such as syphilis. All these spirochaetoses induce a chronic illness, with ill-defined stages, affecting many organs, with a high level of neurotropism. As in syphilis, the nervous system is invaded early in a form of asymptomatic meningitis [1]. Later, neurological abnormalities appear, mainly due to chronic meningitis. Neurological presentation is extremely variable: facial palsy and other cranial neuropathies [5], encephalomyelitis, radiculopathies, demyelinating polyneuropathies and transverse myelitis [6,7] have been reported.

Diagnosis of central nervous system (CNS) involvement is made by blood serology and CSF examination [8]: there is often a mononuclear pleocytosis with elevated protein, but the glucose is normal. *B. burgdorferi*-specific antibodies are demonstrated in the CSF and blood by immunofluorescent assay (IFA) and enzyme-linked immunosorbent assay (ELISA). Demonstration of production of specific antibodies in the CSF appears the most effective test.

MRI findings are nonspecific. In the brain, multiple areas can be affected, but with a predilection for subcortical white matter suggesting demyelinating lesions [9]. Meningitis occurs with direct invasion of the nervous system by *B. burgdorferi*. Inflammation of the subarachnoid space may be manifest by meningeal contrast enhancement [8, 10,11]. Few cases of Lyme myelitis or radiculomyelitis have been reported [6, 7, 12,13], and the radiological findings have also been nonspecific. Isolated foci of spinal inflammation seem exceedingly rare. Neurological disturbance limited to the spinal cord can manifest clinically as acute transverse myelitis [14]. Focal enlargement of the cervical spinal cord with nodular high signal on T2-weighted images has been reported in a patient with a Brown-Séquard syndrome due to *B. burgdorferi* [15]. In another case of acute transverse myelitis in childhood, MRI demonstrated an enlarged cervical spinal cord with marked contrast enhancement [16]. Demaerel et al. [11] reported a 10-year-old boy with low-back pain and a cauda equina syndrome in whom MRI showed subtle thickening and strong contrast enhancement of all the nerve roots of the cauda equina. Like many infections, Lyme borreliosis can be responsible for a syrinx cavity: a case of subacute transverse myelitis with spinal cord cavitation has been reported [17].

In our case, the clinical features suggested a lesion of the lower thoracic cord and conus medullaris, but MRI T1- and T2-weighted images of this region were normal. However, leptomeningeal enhancement was consistent with leptomeningitis. Lyme infection of the spinal cord seems to affect the leptomeninges more frequently than the parenchyma [18]. We may assume that, as in other infections, leptomeningitis represents the initial stage of spinal infection by *B. burgdorferi* and precedes the parenchyma infection leading to myelitis. As the neurological picture is nonspecific, contrast enhancement of the spinal leptomeninges may give rise to a differential diagnosis including: granulomatous (sarcoidosis, toxoplasmosis), bacterial and viral infectious (HIV, cytomegalovirus), lupus erythematosus, Behçet's disease and lymphoma.

Postmortem findings in Borrelia rhombencephalomyelopathy have shown slight thickening of the leptomeninges of the spinal cord [19]. Microscopic examination revealed fibrosis with chronic inflammation restricted to the leptomeninges; the infiltrate was concentrated around the vessels. Basal meningitis with obliterative vasculitis in Lyme disease has been shown and may represent the same histopathological mechanism [20]. Spinal subarachnoid haemorrhage involving the spinal cord, caused by *B. burgdorferi*, has been reported and vasculitis was suggested in that case [21].

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