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Lyme Disease in Childhood Presenting as Primary Leptomeningeal Enhancement without Parenchymal Findings on MR

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Summary: We report the MR appearance of a rare case of Lyme disease presenting as diffuse leptomeningeal enhancement in the absence of parenchymal lesions. In the appropriate clinical setting, one should consider Lyme disease in the differential diagnosis of meningeal enhancement.

Index terms: Lyme disease; Spinal cord, infection; Spinal cord, inflammation; Spinal cord, magnetic resonance; Pediatric neuroradiology

Lyme disease is an inflammatory disease with multisystem manifestations. Involvement of the brain has been reported by several authors. However, meningeal enhancement is rare. We present a case with diffuse enhancement of the pial lining of the brain stem and whole spinal cord in the absence of parenchymal lesions.

Case Report

A 12-year-old boy was admitted in August with a 2-week history of fatigue and neck stiffness. For 5 days he had complained of difficulty closing his right eye and occasional pains in the right arm. A month earlier he had been on a holiday at the French Mediterranean coast, during which he remembered having removed an "insect" from his back.

On admission the boy was afebrile and looked moderately ill. He had lost 4 kilograms. There was a paralysis of the right facial nerve, but the muscle strength of arms and legs was normal. Neck stiffness was present, but the Kernig sign was negative.

Electromyography confirmed the right-sided peripheral seventh nerve palsy with subtotal motor deficit. An unenhanced cranial computed tomography (CT) scan and a high-resolution CT scan of the petrous bones were normal. Spin-echo T1- and T2-weighted images of the brain were normal. After intravenous administration of gadopentetate dimeglumine, a diffuse leptomeningeal enhancement was seen surrounding the brain stem and the spinal cord from C1 to the conus medullaris (Fig 1).

Examination of the spinal fluid showed 355 lymphocytes/mm³, protein of 216 mg/dL, and normal glucose.

Analysis of serum and spinal fluid for antibodies against *Borrelia burgdorferi* showed evidence of an acute infection (Table 1). No virus could be isolated from the spinal fluid by standard isolation techniques. In the blood there was no serologic evidence for a recent infection with Coxsackievirus B1-6, cytomegalovirus, Epstein-Barr virus, lymphocytic choriomeningitis virus, *Mycoplasma pneumoniae*, mumpsvirus or poliovirus. Against a number of viruses for which serologic evidence of past infection was present, cerebrospinal fluid antibodies were measured and found to be negative.

The boy was treated with a 2-week course of intravenous ceftriaxone and recovered fully during that period.

Discussion

Lyme disease is a multisystem inflammatory disease caused by *Borrelia burgdorferi* (1). The vector in Europe is most frequently *Ixodes ricinus*. The disease, first described at the beginning of this century, was named after the town of Old Lyme, Connecticut, where 39 children were diagnosed as having a tick-borne arthritis. Lyme disease is endemic in several European countries, including France (1). Central nervous system involvement in Lyme disease occurs in 10% to 15% of the cases (2).

The highest incidence of infection is seen in patients younger than 15 years and between 25 and 44 years of age. Three stages are usually differentiated in the disease process: the acute stage I, 2 to 30 days after the tick bite, with flu-like symptoms and an expanding skin lesion; stage II, 1 to 4 months after infection, with cardiac and neurological abnormalities; and stage III, up to a few years later, with arthritis and chronic neurological symptoms (2).

The neurological presentations may include peripheral neuropathies, radiculopathies, myelopathies, encephalitis, lymphocytic meningitis, pain

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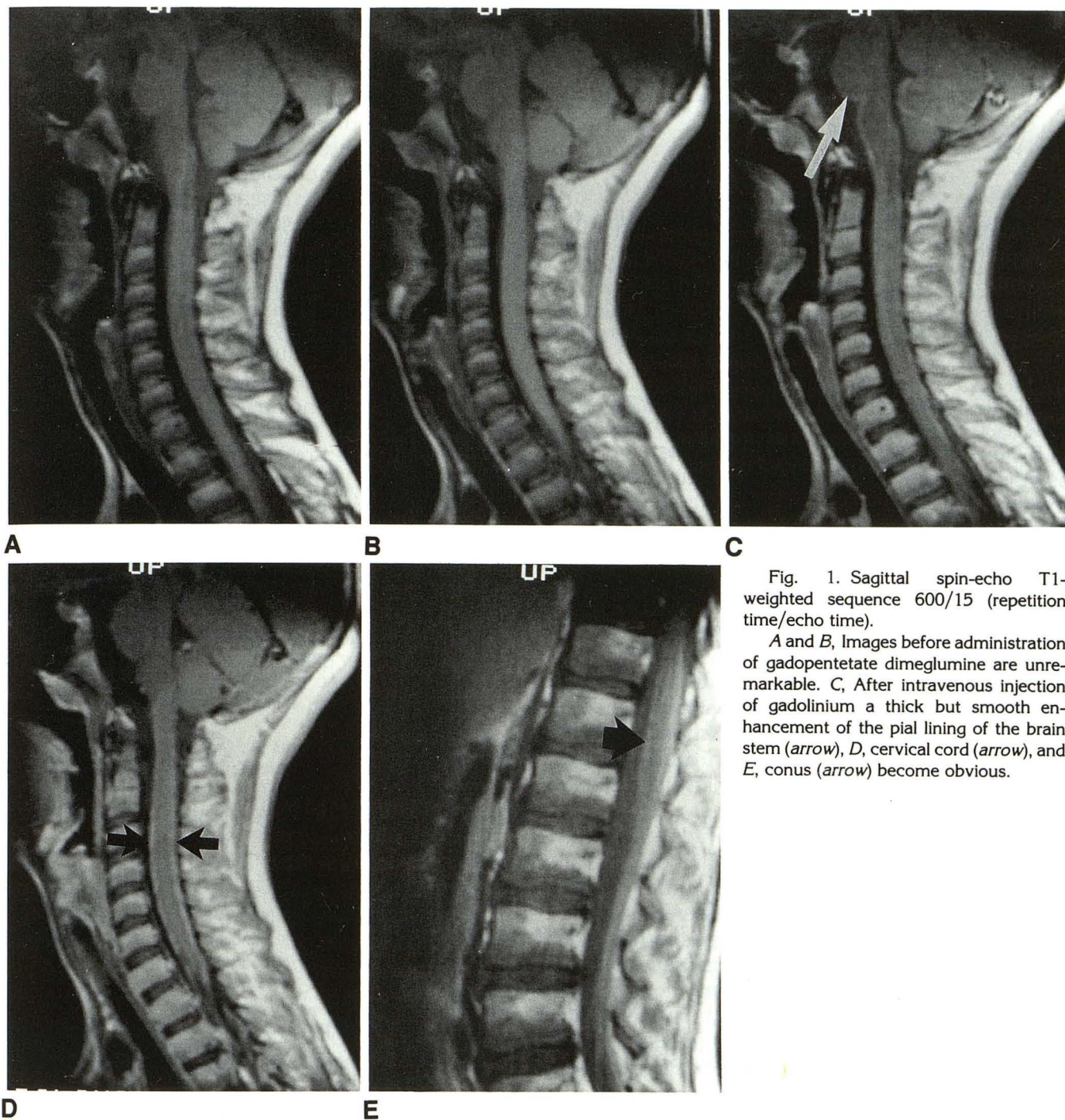


Fig. 1. Sagittal spin-echo T1-weighted sequence 600/15 (repetition time/echo time).

A and B, Images before administration of gadopentetate dimeglumine are unremarkable. C, After intravenous injection of gadolinium a thick but smooth enhancement of the pial lining of the brain stem (arrow), D, cervical cord (arrow), and E, conus (arrow) become obvious.

syndromes, cerebellar signs, cognitive disorders, movement disorders, and seventh cranial nerve palsy (2-4).

A definitive diagnosis may be difficult to obtain because active infection is not detectable with routine tests and a positive serology does not exclude other causes (5). This problem may be resolved with the advances in molecular genetics and the application of polymerase chain reactions

(2). The measurement of the intrathecal production of *B. burgdorferi* antibodies is considered sensitive, highly specific, and broadly available (6). The treatment consists of antibiotics sometimes in association with steroids (2).

The mechanism of central nervous system involvement is not clear and could be the result of direct invasion or a vasculitic process.

Several studies have reviewed the CT and

TABLE 1: *Borrelia burgdorferi* antibody titers in serum and cerebrospinal fluid

	Date			
	8/13	8/18	8/25	9/5
Serum				
EIA	3.1	5.4	—	1.4
IF	Negative	1/128	—	1/128
CSF				
EIA	5.6	5.1	4.5	4.1
IF	1/16	1/32	1/32	1/8

Note: IF: immunofluorescent antibody titers; EIA: antibodies by enzyme immunoassay, expressed as the ratio of the optical densities [patient serum or CSF-borderline positive control serum or CSF]; CSF, cerebrospinal fluid.

magnetic resonance (MR) findings in Lyme disease (3, 6, 7). Fernandez et al found abnormalities in 57% of the cases (7). Most of the lesions were seen in the cerebral white matter, but also in the thalami and the pons. Gadopentetate dimeglumine had not been administered.

Enhancing white matter lesions have been reported on MR and on CT scans in a few case reports (5, 8, 9). Rafto et al (5) also described leptomeningeal interpeduncular gadolinium enhancement. Krüger et al (3) reviewed 96 cases of adult neuroborreliosis. Brain and/or spinal cord signs were seen in 15 of these patients (3). The head CT scan was normal ($n = 7$), showed edema ($n = 2$), or ischemic lesions ($n = 6$). Brain MR, obtained in three cases, showed multiple sclerosis-like lesions.

Halperin et al (6) reviewed 35 adult patients. MR showed white matter lesions in 16 of 22 cases with acute encephalitis but gadolinium enhancement was only seen in 1 of 17 cases. In 10 of 13 cases with chronic progressive spastic hemi- or paraparesis, white matter abnormalities were observed. Spinal cord MR and/or myelogram appeared normal in all cases. In 41 patients with encephalopathy, MR was obtained in 24 cases and abnormalities were seen in only 8 cases. The

lesions were smaller and less conspicuous. No gadolinium enhancement was seen.

Our case of diffuse brain stem and spinal cord leptomeningeal enhancement in Lyme disease is unusual and demonstrates the importance of including inflammatory diseases such as Lyme disease in the differential diagnosis. In children, pial spread of metastatic disease must be excluded. Pial involvement by spinal sarcoidosis has also been described (10). Clinical correlation and cerebrospinal fluid analysis including cytology are necessary to reach a specific diagnosis. It is clear from this case that postcontrast MR is an excellent method for evaluation of the spinal cord lesions of Lyme disease. Early institution of therapy may minimize the neurological complications and sequelae.

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