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CRANIAL AND SPINAL NEUROPATHY IN LYME NEUROBORRELIOSIS

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ABSTRACT

Introduction: Lyme borreliosis is a multisystem inflammatory disease caused by the spirochete Borrelia burgdorferi, transmitted to humans by infected ticks. The neurological involvement most frequently presents with various types of neuropathy or may include symptoms and syndromes caused by cerebral and spinal cord damage.

Objective: to study the clinical manifestations and outcome of cranial and spinal neuropathies in patients with Lyme neuroborreliosis.

Material and methods: Twelve patients (6M/6F, between 22 to 57 years of age) with cranial and spinal neuropathy of various types were included in the study. The diagnosis of neuroborreliosis was based on the medical history, neurological examination, EMG, and specific laboratory tests. Up to 3 years patients' follow-up was done.

Results: Two patients had optic neuritis with asymmetrical reduction of vision in both eyes. Two patients presented with involvement of cranial nerves and two with unilateral facial nerve palsy. Eight of all patients demonstrated clinical features of asymmetrical sensory-motor polineuropathy with distal paresthesias and loss of vibration sensation. EMG study showed axonal degeneration in eight patients, segment demyelination - in three, and involvement of central motor neuron - in one.

Conclusion: Our study focus on cases with various types of isolated and rarer concomitant cranial and spinal neuropathies. Special attention should be paid to patients with vasculitis-associated opticopathy, particularly with regard to the prognosis of severe papilledema development. Although the benign clinical course of spinal neuropathies, we emphasize on the medico-social significance of neuroborreliosis as a potential cause for neurological disabilities.

Key words: Lyme neuroborreliosis, neuropathy, clinical manifestation, outcome

INTRODUCTION

Lyme disease (LD) is a vector-borne, multisystem inflammatory disease caused by the spirochete Borrelia burgdorferi, which is transmitted to humans by infected ticks of the Ixodes genus (1, 8, 14, 18). The clinical presentation of LD generally follows three stages of disease progression: early localized, early disseminated, and chronic disseminated (1, 13, 14, 17). Neurological involvement is known as neuroborreliosis. Previous studies suggest that a broad variety of peripheral nerve disorders, including single or multiple cranial neuropathies, painful radiculopathies, and diffuse polyneuropathies are typical for the second and third stages of LD (3, 5, 6, 11, 12, 16). Not so rare, neuroborreliosis may involve symptoms and syndromes caused by cerebral and spinal cord damage (2, 4, 17). Diagnosis is made by a history of exposure, previous systemic or acute neurological manifestations of Lyme borreliosis, and determinations of antibodies to Borrelia burgdorferi by enzyme-linked immunosorbent assay and immunoblot analysis (5, 7, 8, 13, 15). Prognosis after therapy is good, but neurological recovery is slower for chronic than acute Lyme radiculoneuropathy (2, 10, 14, 16).

Recently, as a result of rapid rise in incidence and protean neurological manifestations, LD achieves an important medico-social significance (9, 14). It is also a challenge to the specialists as a potential cause for serious neurological discrepancies. Therefore, we decided to study the clinical manifestations and outcome of spinal and cranial neuropathies in patients with early disseminated and chronic LD.

MATERIAL AND METHODS

Twelve patients (6M/6F, between 22 to 57 years of age) with cranial and spinal neuropathy of various types were included in the study. The diagnosis of neuroborreliosis was based on the medical history of previous tick bite, neurological examination, electromyography (EMG), and specific serologic/ cerebrospinal fluid (CSF) laboratory tests. Up to 3 years patients' follow-up was done.

RESULTS

All patients had different degrees of peripheral nerves abnormalities. Two patients presented with optic neuritis and neuro-ophthalmological findings of asymmetrical reduction of vision in both eyes: 0.6 for the worse eye in the first patient and 0.04 for both eyes in the second one. Two patients had simultaneous involvement of III, IV and VI cranial nerves. Unilateral facial nerve (Bell's) palsy was found in two cases. Eight of all patients had clinical features of asymmetrical sensory-motor polineuropathy with distal paresthesias and loss of vibration sensation. EMG study demonstrated axonal degeneration in eight cases, segment demyelination - in three, and syndrome of amyotrophic lateral sclerosis (ALS) - in one.

Up to three years patients' follow-up established complete recovery of vision in the first patient, partial vision recovery in one eye combined with definite blindness as a result of subsequent papilla atrophy in the second one, no residual symptoms from multiple cranial neuropathy in two patients, and moderate mimic muscles insufficiency in two cases with Bell's palsy. Clinical features of spinal neuropathy had improved in all eight patients, especially the reduction of irritative sensory feelings and recovery of motor deficit. Two of the patients, respectively with ALS syndrome and additional myelitis have worse recovery of motor function, presenting with serious gait impairment.

DISCUSSION

Recently, neuroborreliosis has become the most frequent arthropod-borne infection in North America and Europe (9). It is a tick-borne disorder associated with a wide variety of neurological manifestations (1, 5, 8, 14). The involvement of both central and peripheral nervous system can be divided into acute and chronic forms. Within weeks after disease onset, approximately 15% of untreated patients develop an acute radiculoneuritis, weakness, and sensory loss, often associated with cranial neuropathy (6, 11, 12, 15, 17, 18). Months to years later on, patients may develop chronic polyradiculoneuropathy with sensory symptoms (1, 4, 7, 14, 16, 18). In correspondence to these findings we illustrate twelve cases with different types of cranial and spinal neuropathy associated with LD. An acute peripheral nervous involvement was found in five patients and chronic form of the disease - in seven.

Although the mechanism underlying these neuropathies remains unclear, the neurological involvement can be due either to the direct action of the spirochetes on neural cells or to the indirect immunologic reactions (1, 7, 9, 13, 14, 17). In agreement with these speculations, we suppose development of vasculitis-associated opticopathy in cases with severe optic disc swelling.

Diagnosis is made by a history of preceding exposure, recognition of characteristic clinical features, serologic or CSF evidence of antibodies to B. burgdorferi infection, and EMG assessment of nerves conduction (1, 8, 9, 13, 14, 16). Our clinical findings reveal history of tick bite within 2-3 weeks in four patients and 6-18 months in eight cases. All patients are seropositive and six of them have CSF antibodies. EMG data are equivocal with previous electrophysiological studies which most frequently establish an axonal degeneration in patients presenting with peripheral neuropathy (14, 16).

The systemic literature review suggests a good prognosis after antibiotic, anti-inflammatory, and physical therapy. (2, 9, 10, 13, 15). Our follow-up validates the slower and worse neurological recovery in patients with chronic form of spinal and cranial neuropathy, especially in cases with additional central motor neuron injury.

CONCLUSION

Our study focuses clinical interest on patients with neuroborreliosis, presenting with isolated and rarer concomitant cranial and spinal neuropathies. We find as a remarkable the high frequency of cranial nerves involvement. Special attention should be paid also to optic neuritis, particularly to the prognosis of papilledema development with elements of vasculitis-associated opticopathy. Although literature review reveals benign clinical course of spinal neuropathies, we emphasize on medico-social significance of Lyme disease as a potential cause for neurological disability.

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