CASE REPORT

Lyme disease: an unusual case of peripheral nerve palsy

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Lyme disease is a vector-borne multisystem inflammatory disease caused by the spirochete Borrelia burgdorferi sensu lato. This disease is frequently seen in North America and to a lesser degree in Europe. However, its presence in England is uncommon and we present a case in which the patient developed a palsy of the common peroneal nerve.

The common causes of sensory and motor changes in the lower limbs are either a central lesion, such as a prolapsed intervertebral disc, or a peripheral lesion which may be associated with trauma or follow arthroplasty, ligament reconstruction or osteotomies.

The majority of nerve palsies are neuropraxias which recover spontaneously. Nerve conduction studies can confirm the diagnosis, or be used if recovery is prolonged. Surgical exploration is recommended for loss of continuity of the nerve, or if recovery is prolonged beyond several months.1

In the absence of an appropriate clinical history and evaluation, alternative causes for an isolated nerve palsy must be sought, as this may avoid surgical exploration and any associated morbidity.

We present a rare cause of common peroneal nerve palsy and a review of the associated literature.

Case report

A 51-year-old man presented to his general practitioner with a short history of weakness, paraesthesia and pain affecting his right lower leg. He had no relevant past medical history nor history of injury or recent travel. An insect bite in the popliteal fossa, which was followed by an annular rash, was reported to have occurred several weeks earlier. This was present for approximately ten days before resolving spontaneously.

The patient was initially referred to a musculoskeletal assessment centre with a suspected prolapsed lumbar disc. Clinical examination showed power of 1/5 on the MRC grade in tibialis anterior with reduced sensation in the L5 dermatome. The remainder of the neurological examination was normal. An MR scan showed a small prolapsed disc at the L4-5 level, but no evidence of nerve compression.

Nerve conduction studies demonstrated absent conduction of the motor and sensory components of the peroneal nerve. However, the tibial (motor) and sural (sensory) nerves were normal (Table I). The sensory action potential in the superficial peroneal nerve was absent, suggesting a peripheral (post-ganglionic) rather than a spinal root (pre-ganglionic) lesion.

Electromyography (EMG) demonstrated active degeneration in tibialis anterior and peroneus longus, whereas biceps femoris and tibia-lis posterior were normal. High electrical stimulation demonstrated that only a few motor units were recoverable in tibialis anterior.

The patient also began to complain of lethargy and mild headaches. The combination of mononeuropathy, lethargy, and an apparent history of an insect bite and a subsequent annular rash raised suspicions of an infective tick-borne disease. The headaches suggested meningism, which is recognised in patients with Lyme disease.2 Serological examination confirmed exposure to Borrelia burgdorferi, and thereby the diagnosis of Lyme disease. The patient was started on amoxicillin 500 mg four times per day for four weeks.

Nerve conduction studies and an EMG were repeated six months afterwards and demonstrated that the motor nerves distal to the common peroneal nerve had reduced latency, velocity and amplitude compared to those above the knee. The right sural and tibial nerves demonstrated normal latency, velocity and amplitude (Table I).

EMG studies suggested some active degeneration, considerable voluntary activity and some re-innervation. Clinical examination
suggested that extensor hallucis longus and tibialis anterior had a power of 3 to 4/5 on the MRC grade.

The patient’s neurological condition was unchanged ten months after the onset of disease and the antibiotic treatment.

**Discussion**

Lyme disease is a vector-borne multisystem inflammatory disease caused by the spirochaete *Borrelia burgdorferi* sensu lato. In the 1920s Garin and Bujadoux described a similar condition which they felt was due to spirochaete infection. Although probably known previously in Europe under a variety of names, the now familiar term Lyme disease has been used following an outbreak of juvenile arthritis in the town of Old Lyme, Connecticut, in the mid-1970s. Further investigation led to the isolation of *B. burgdorferi* from the local deer tick, *Ixodes scapularis*.

This case represents the first case of common peroneal nerve palsy in Lyme disease described in the literature. Indeed, mononeuropathy attributable to this condition is reported infrequently.

The disease typically has three phases: early localised, early disseminated and late. Early localised infection is characterised by erythema migrans, an expanding annular rash with a central macule or papule with varying degrees of central clearing. Patients may also experience flu-like symptoms in the early stages.

In early disseminated infection additional outbreaks of erythema migrans may appear in sites remote from the original bite, and in European cases a borrelial lymphocytoma may be seen. Systemic and focal central neurological symptoms such as meningitis, fatigue, Bell’s palsy, myalgia, iritis or optic neuritis, are common.

In late Lyme disease, non-specific neurological disturbances such as impaired cognitive function, encephalitis and encephalopathy may be present. Furthermore, arthropathy is common, typically affecting the knee.

The clinical presentation varies between Europe and North America, with a higher incidence of peripheral neurological manifestations in Europe and arthropathy and central neurological symptoms in North America. These differences can be partly explained by the different species of *B. burgdorferi*.

The complex pathology of the disease is well described. Substances in the tick bite saliva interfere with the normal host inflammatory response, thereby reducing local neutrophil migration and enabling haematogenous dissemination of the spirochaete to different sites. In nervous tissue, *B. burgdorferi* can induce proliferation and subsequent apoptosis of astrocytes. Quinolonic acid is also released, as are the cytokines TNF-α and IL-6, which are toxic to cells. Antibody clearance is hindered by the alteration of expression of cell surface protein and interference with activation of complement.

Owing to the diverse and predominantly non-specific nature of the symptoms associated with Lyme disease, clinical diagnosis can be difficult, particularly in the absence of a skin rash. Although erythema migrans is thought to be present in up to 80% of infected patients, variations in the macroscopic appearance should be noted. The Centre for Disease Control recommends serological diagnosis as a two-test technique involving high-specificity Enzyme-linked immunosorbent assay, followed by the highly sensitive Western immunoblot technique.

Following confirmation of the infection, antibiotic therapy is indicated with appropriate agents, often doxycycline. A four-week course of parenteral medication is recommended for those with severe debilitating disease such as meningitis, central nervous system neuropathy or myocarditis.

Early appropriate treatment improves the condition, but 30% of patients have persisting symptoms, although recent evidence suggests that a response may take six to 12 months following completion of therapy. Patients with late Lyme disease pose further challenges with regard to treatment, and there is controversy as to whether *B. burgdorferi* is the causative factor in those with persisting morbidity. In such cases, prolonged therapy of beyond four weeks is not recommended, and the role of multiple repeated regimes of treatment is controversial.

All clinicians must be aware of alternative causes of a peripheral neuropathy. This is particularly important with a history of an insect bite and rash, suggesting infection by *B. burgdorferi*, as prompt treatment can prevent progression to the chronic form of Lyme disease with its associated morbidity.
References