Twelve cases of Lyme's disease with neurological complications are reported. Seven patients had meningoradiculitis of the Garin-Bujadoux-Bannwarth type, with facial palsy in 2 cases. In 1 case the radiculitis involved only the cauda equina. Two more patients had meningomyelitis. Of the remaining 3, 1 had subacute inflammatory polyneuritis with albumino-cytologic dissociation, 1 had probable dorsal epiduritis, and the last one developed parkinsonism and communicating hydrocephalus after an otherwise classical meningoradiculitis.

Three patients recalled a tick bite but only one a cutaneous eruption. No arthritis or cardiac involvement were observed. In 2 cases the CSF contained pseudo-neoplastic cells. Severe pain was a prominent feature in most cases. Pain consistently and rapidly improved on high-dose intravenous penicillin, while other signs or symptoms (e.g. paresthesias or fatigue) often lasted several months.

Parkinsonism and hydrocephalus were not influenced by penicillin, and both required specific therapy. Isolated neurological (both central and peripheral) involvement is not unusual in Lyme's disease and may give rise to a wide range of signs and symptoms. This diagnosis is to be considered even when other features of Borrelia burgdorferi infection are lacking.

PMID: 2662339 [PubMed - indexed for MEDLINE]
INTRODUCTION: Many different neurological conditions may be seen in the later stages of Lyme's Disease, such as blindness, epileptic crises, CVA, extrapyramidal disorders, amyotrophic lateral sclerosis, and dementia may be yet another form of presentation of chronic infection due to Borrelia burgdorferi (Bb).

Progressive Supranuclear Paralysis (PSP), a disorder of unknown aetiology, considered to be the commonest cause of Parkinsonism-plus, one of the symptoms of which is dementia, has never been mentioned in this type of differential diagnosis.

CLINICAL CASE: We present the case of a 78 year old man with sub-acute mental deterioration, Bb positive serology in both plasma and CSF, and with clinical and epidemiological features compatible with Lyme's Disease. Complementary tests were negative. The syndrome corresponded to Lyme's Disease and improved after treatment with ceftriaxona.

CONCLUSIONS: We consider aspects of the aetiology of PSP which are still not clear. In our patient, the aetiology seemed to be Bb infection, according to the criteria of the original description of the disease and in view of the neuropathological findings which have shown Bb in the substancia nigra of the mid-brain and the existence of an animal model in which Bb shows a particular tendency to colonize infratentorial structures.

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Lyme-associated parkinsonism: a neuropathologic case study and review of the literature.

- **Cassarino DS, Quezado MM, Ghatak NR, Duray PH.**

Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Bethesda, Md 20892, USA.

Neurological complications of Lyme disease include meningitis, encephalitis, dementia, and, rarely, parkinsonism. We present a case of striatonigral degeneration, a form of multiple system atrophy, in Lyme-associated parkinsonism.

A 63-year-old man presented with erythema migrans rash, joint pains, and tremors. Serum and cerebrospinal fluid antibodies and polymerase chain reaction for Borrelia burgdorferi were positive. Clinical parkinsonism was diagnosed by several neurologists.

Despite treatment, the patient continued to decline, with progressive disability, cognitive dysfunction, rigidity, and pulmonary failure. At autopsy, the brain showed mild basal ganglia atrophy and substantia nigra depigmentation, with extensive striatal and substantia nigral neuronal loss and astrogliosis. No Lewy bodies were identified; however, ubiquitin-positive glial cytoplasmic inclusions were identified in striatal and nigral oligodendroglia. There were no perivascular or meningeal infiltrates, the classic findings of neuroborreliosis.

To our knowledge, this is the first report of striatonigral degeneration in a patient with B burgdorferi infection of the central nervous system and clinical Lyme-associated parkinsonism.

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