Dynamic Chiropractic

CHRONIC / ACUTE CONDITIONS

Lyme Disease: The Great Imitator

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Lyme disease is a multisystem disease with prominent neurological involvement. Lyme disease is caused by spirochete Borrelia burgdorferi which is transmitted to humans through the bite of Ixodes ticks. The disease has been documented in 47 out of 50 states in the United States but is thought to be more common in New England, Wisconsin, Minnesota, and in the Pacific Coast area. The disease has been broken down into three distinct stages based on the presenting symptoms.

Stage I

Stage I of Lyme disease usually begins three days to one month after a tick bite with erythema migrans (a red papule or macula that starts at the site of the bite and expands outward to form an annular red lesion with central clearing) in approximately 50 percent of affected patients. The lesions average 15 centimeters in diameter, although some may be larger. The lesion is asymptomatic in two-thirds of affected patients and may cause itching or burning in the remaining third. The lesion usually fades in three to four weeks. In over one-half of untreated patients, multiple secondary lesions may develop within a few days of the primary lesion which are smaller, lack central induration, and may appear anywhere on the body, but generally spare the soles and palms. Additionally, the patient may manifest the following flu-lke symptoms which are associated with the skin lesion: polyarthralgia, myalgia, fatigue, headache, stiff neck, sore throat, fever, chills and lymphadenopathy. Approximately 10 percent will have nausea, vomiting, and right upper quadrant pain suggestive of hepatitis. Later, in Stage I, conjunctivitis, urticaria, and/or a malar rash may develop.

Stage II

Neurological manifestations and cardiac abnormalities are the hallmark of Stage II of Lyme disease. Nervous system symptoms may manifest while erythema migrans is present but usually begin one to six months after the erythema migrans has faded. Additionally, the following systemic symptoms and signs (in decreasing order of frequency) may be present at the onset of the neurological abnormalities associated with Stage II of the disease: headache, fatigue, fever, myalgia, neck stiffness, nausea, vomiting, arthralgia, photophobia, and arthritis. The neural involvement in Lyme disease commonly takes the form of a triad of neurological complaints comprised of lymphocytic meningitis, cranial, and peripheral neuropathies, and radiculopathies. This triad of complaints is also known as Banwarth's syndrome. Neurological abnormalities are seen in 15 percent of Lyme disease patients and may occur in the absence of Stage I symptoms and signs.

Lymphocytic meningitis is the most common neurological abnormality seen in Stage II of the disease and is preceded by erythema migrans in 40 percent of cases, may follow the erythema migrans by two to 10 weeks, or may be the presenting sign of the disease process. Headache is the most common symptom (seen in 50 percent) of the lymphocytic meningitis, with the patient complaining of frontal or occipital headache varying in intensity from mild to disabling. Extraocular eye movements may cause pain to be increased. The patient may also exhibit mild neck stiffness, seen usually on flexion, with

Kernig's and Brudzinski's signs rarely seen. Photophobia has been noted in 10 percent of cases, nausea and vomiting in 25 percent, and low grade fever in 30 percent of cases. The symptoms may persist for up to one to two months and then resolve gradually over several weeks.

Cerebral symptoms which may accompany the meningitis include somnolence, emotional liability, depression, impaired memory and concentration, behavioral changes, and fatigue. Associated cerebral symptoms are generally mild; however, more severe cerebral symptoms may result from encephalitis associated with the Lyme disease. In cases of encephalitis secondary to Lyme disease, the symptoms develop rapidly over hours to days. Patients may experience severe somnolence, hallucinatory delirium , disorientation, paranoid psychosis, catatonia, confusion, irritability, agitation, coma, as well as all types of seizures. Focal neurological signs in the form of hemiparesis, cerebellar ataxia, chorea, dystonia, athetosis, tremor, or possible Parkinsonia symptoms may accompany cerebral symptoms of encephalitis and may be gradual or sudden in onset.

Myelitis is also associated with Lyme disease and is the most frequent severe central nervous system abnormality seen in Stage II of the disease process. The myelitis may take the form of an acute or subacute transverse myelitis which develops over hours to days. The symptoms of myelitis include: bilateral spastic paraparesis, Babinski signs, and loss of superficial reflexes; a sensory level between T4 and T10; and bowel and bladder incontinence or urinary retention.

Cranial neuropathies develop in approximately 60 percent of Lyme disease patients and are commonly seen three weeks after the onset of the erythema migrans. Multiple cranial neuropathies are common. The most common cranial nerve involved is the facial nerve (CN VII). Facial nerve involvement accounts for 70-80 percent of all Stage II cranial nerve palsies. Weakness usually begins in the summer months over one to two days and proceeds to bilateral involvement in over one-third of affected patients, with both sides of the face becoming paralyzed within a few days to three weeks of each other. Outcome for this form of facial nerve paralysis is usually favorable with most patients experiencing a complete recovery within a one to two months period. The facial paralysis of Lyme disease is differentiated from that of Bell's palsy by the following three factors:

Summer onset
Bilateral involvement
History of tick bite or erythema migranes

The optic nerve (CN II) may be involved in Stage II of Lyme disease due to increased intracranial pressure, inflammation along the meninges of the optic nerve, or by optic neuritis. Recovery is incomplete if antibiotic therapy is delayed. Involvement of the trigeminal nerve (CN V) is in the form of sensory symptoms such as facial paresthesia, numbness, and pain that may resemble trigeminal neuralgia. The corneal reflex is generally unaffected and the patient usually recovers spontaneously within a few weeks to months. Cranial nerves III, IV, and VI involvement may lead to diplopia. Cranial nerve VIII involvement may lead to a hearing loss and vertigo. Permanent hearing losses have been reported, even with antibiotic treatment. There have also been reports of cranial nerve IX, X, XI, and XII, although incidences are extremely low.

Radiculopathy is the last portion of the triad of neurological symptoms associated with Lyme disease. Spinal and radicular pain associated with Lyme disease is generally worse at night and resistant to analgesics. The spinal pain has been characterized as having a generalized burning, gnawing or tearing quality that migrates. Once the pain is established it may last for months and eventually

subside completely within a six weeks to three months period. Motor symptoms that may be associated with the intense radicular pain may include motor loss to the lower extremities twice as frequently as the upper extremities, diaphragm paralysis, cauda equina syndrome, or abdominal wall paralysis. Sensory losses are less common than motor losses and tend to be dermatomal and located in the lower cervical and in the T8-T12 regions.

Stage III

In this stage of Lyme disease, the patient may experience arthritis weeks to years after the initial onset of the disease. The rheumatological symptoms begin as migratory musculoskeletal discomfort involving the joints, bursae, and tendons. Joint swelling and pain appear several months later with the knee being the most affected joint. Destruction of cartilage and bone may occur in individuals with long-standing Lyme disease and lead to chronically enlarged joints.

As one can see, Lyme disease presents a diagnostic dilemma to the chiropractic physician due to the myriad of neurological conditions that may be mimicked by the disease. An important aspect of the history in any patient with signs or symptoms similar to those discussed should be the investigation into the possibility of a tick bite in the recent past or the presence of the erythema migrans rash. Diagnostic testing for Lyme disease as well as available therapies will be discussed in my November article.

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