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Lyme disease misdiagnosed as a temporomandibular joint disorder

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Craniomandibular disorders cause many pleomorphic and seemingly unrelated clinical manifestations that mimic other more serious medical problems and thus can present physicians and dentists with a challenge that invites misdiagnosis and improper treatment planning. Conversely, misdiagnosis and ineffective treatment planning are facilitated when serious medical problems manifest a range of signs and symptoms that are clinically similar to temporomandibular joint muscle dysfunction. At times, the patient's response to therapy may be the best method of corroborating a diagnosis, as illustrated in this report of a patient with Lyme disease that was misdiagnosed as a temporomandibular joint disorder. Lyme disease has already reached epidemic proportions in several parts of the United States and its geographic distribution is spreading. Because Lyme disease is a life-threatening illness whose clinical manifestations can mimic temporomandibular joint/myofascial pain-dysfunction, it is the responsibility of every dentist who treats craniomandibular disorders to become familiar with the clinical presentations of Lyme disease and more proficient in its differential diagnosis. (J PROSTHET DENT 1990;63:82-5.)

Temporomandibular joint (TMJ) disorders appear to be common multicausal problems[1-6] characterized by numerous signs and symptoms.[7-10] These disorders cause many pleomorphic and seemingly unrelated clinical manifestations that mimic other more serious medical problems. Physicians and dentists are therefore faced with a diagnostic challenge[11] that may lead to misdiagnosis and improper treatment planning.

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Conversely, misdiagnosis and ineffective treatment planning are facilitated when medical problems manifest a range of signs and symptoms that are clinically similar to TMJ disorders.

At times, the patient's response to therapy may be the best method of corroborating a diagnosis, as illustrated in this history of a patient with Lyme disease that was misdiagnosed as a TMJ disorder. The potential consequences of misdiagnosis in this patient are considerable, because Lyme disease can cause irreversible neurologic changes if not treated early, and in its advanced stage the illness is sometimes fatal.[12]

LYME DISEASE

The clinical manifestations of Lyme disease can mimic those of a TMJ disorder. Lyme disease is not an obscure malady. Indeed, it is the most common tick-borne illness in the United States and has become a major public health problem, particularly in New York State and eastern Long Island where more than 55% of all reported cases of the disease occur.[13-15]

Of the 5731 cases of Lyme disease reported to the Centers for Disease Control between 1980 and 1986, 86% were contracted in seven states: the five Atlantic seaboard states of New York, New Jersey, Connecticut, Rhode Island, and Massachusetts and in Wisconsin and Minnesota.[16]

The geographic range of Lyme disease is spreading. Once seen only in New England, the illness is now recognized in 33 states, in every country in Europe, in the People's Republic of China, and in Australia.[17] In New York, the number of reported cases of Lyme disease has doubled every year since 1982.[16]

Lyme disease is evenly distributed between males and females and it affects all age groups, although only 2.5% of all cases of Lyme disease in the United States have been diagnosed in black patients.[12] The disease was first recognized in Old Lyme, Connecticut in 1975 when a cluster of patients developed an illness resembling juvenile rheumatoid arthritis.[18]

Lyme disease is caused by the spirochete *Borrelia burgdorferi*.[19] The usual incubation period of the disease varies between 3 to 40 days,[16] but the spirochete can remain dormant in its host for several months to several decades.

Any blood-sucking insect (mosquitos, horse flies) can transport the Lyme spirochete,[20] but the deer tick *Ixodes dammini* is the most common vector.[13] In contrast to the adult tick, which prefers to feed on large animals (especially white-tail deer), the young adults, or nymphs, prefer mice, domestic animals, and humans. Infected ticks rest on grasses and shrubs in wooded areas and are transmitted to animals and humans when they brush by. Although Lyme disease may be contracted throughout the year, most individuals are infected during the warmer months from May to August when the nymph population is most abundant.[13]

Lyme disease is a multisystem disorder involving the skin, joints, nervous system, and heart.[12] Although the disease most often affects large joints such as the knee and hip, [13] the TMJ can also become involved.[12]

SIGNS AND SYMPTOMS

The appearance of a slowly expanding red rash termed *Erythema chronicum migrans*, or ECM lesion, is the most reliable method of diagnosing Lyme disease and is its most characteristic sign.[12] However, ECM lesions only appear in approximately 60% of all infected patients.[13] When present, the rash develops as a host response to spirochetes in the skin and subcutaneous tissues and typically begins as a small red macule at the site of the tick bite several days to several weeks after infection,[13] although in some patients multifocal lesions may appear. The lesion then expands slowly in a circular pattern to become a painless erythematous patch approximately 8 to 40 cm in diameter. The ECM rash can vary greatly in appearance from patient to patient, but it generally emerges as an expanding red ring with a clear center or uniform red blotchy area that persists for several days or remains for several months before gradually fading.[13]

Other early symptoms of Lyme disease[12] include generalized myalgia and arthralgia, atypical facial pain, joint swelling, synovitis, joint stiffness, headaches, neck stiffness, fatigue, vertigo, short-term memory loss, numbness and tingling in the extremities, hearing impairment, impaired concentration, loss of appetite, carpal tunnel syndrome, and facial paralysis that is most often bilateral.[17]

Since many of the preliminary signs and symptoms of Lyme disease are similar to those of a TMJ disorder, an early differential diagnosis can be difficult. This difficulty often leads to misdiagnosis, mistreatment, and further progression of the disease. In the late stages of the illness, spirochetes may settle in various organ systems[19] and neurologic complications[21] such aseptic meningitis, encephalitis, facial palsy, cranial nerve palsy, sensory and/or motor peripheral neuropathy, and myelitis with demylination, and cardiac complications[22] such as myocarditis, irregular heart beat, and complete heart block can occur. Neurologic complications occur in 20% to 25% of all untreated patients,[21] and cardiac abnormalities appear in approximately 7% of untreated patients.[22] Lyme-related cranial neuropathies most frequently involve the facial (VIII) nerve,[17] and bilateral facial palsy (Bell's palsy) is a common complication of the disease.[12] Lyme neuropathies can also occur unilaterally, as can the development of unilateral Bell's palsy as a complication of TMJ syndrome.

A positive diagnosis of Lyme disease is confirmed by the appearance of one or more ECM lesions, by using serum antibody detection with either an immunofluorescent assay (IFA titer) or an enzyme-linked immunoassay (ELISA) method, with histochemical methods, and with in vitro culture of spirochetes from biopsy tissues and synnovial fluid.[23] Serologic tests are unreliable, however, because of their inability to detect antibodies in the early stages of the disease and because approximately 40% to 60% of those infected never produce antibodies to the spirochete.[17] Thus, a negative seroloy finding does not rule out a diagnosis of Lyme disease with certainty.

PATIENT HISTORY

The subject of this report is a 33-year-old white woman with a noncontributory past medical history. The patient's symptoms began in 1986 with the onset of recurring episodes of frontal headache. In a short time, she also developed tingling and paresthesia in the posterior cervical region, in both hands, and in all fingers. The dysesthesias began to fade gradually approximately 1 week after their

onset, but the patient's neck soon became stiff and painful. A chiropractor was then consulted in the belief that spinal adjustments would ease her headaches and cervical symptoms. She discontinued chiropractic therapy 9 months later when repeated treatments failed to provide symptomatic relief, and her right TMJ became painful and developed an intermittant click. In the weeks that followed, the patient consulted her family physician and a neurologist because she was confident that they would find the cause of her complaints. However, all of the medical, neurologic, and radiographic tests, including CT scans of the skull and cervical spine, and brain scans were unremarkable. The patient's complete blood count, sequential multiple analysis with computer thyroid profile, sedimentation rate, and urinalysis were also within normal limits. The physicians eventually prescribed a total of 12 medications including ibuprofen, naproxen (Anaprox), hydroxyzine pamoate, methocarbamol, cyclobenzaprine, amitriptyline, carisoprodol, propoxyphene napsylate, acetaminophen with 1 grain of codeine, oxycodone with aspirin, and diazepam. All of the medications were either ineffective or produced adverse reactions with the exception of the oxycodone preparation (Percodan), which "made the pain more bearable."

When the patient's physicians were unable to identify a medical cause for her chief complaints, they suggested she might be suffering from a TMJ disorder and advised her to consult a dentist. The patient's family dentist also attributed her complaints to the presence of a TMJ dysfunction and instituted treatment with a flat-plane mandibular splint. After insertion of the splint, the patient's headaches, neck stiffness, clicking, and TMJ pain decreased slightly, but several days later her symptoms returned with increased severity. The splint failed to provide relief and was eventually discarded.

The patient was finally referred to our office for evaluation and treatment in February 1988. Her chief complaints at the time of her initial visit included recurring mild to moderate protracted headaches in the occipital, temporal, and frontal regions, neck and shoulder pain, occasional episodes of pain in the lower back, intermittent episodes of tinnitus in the right ear, recurring bouts of vertigo with postural imbalance, and bilateral pain in the masseter muscles, TMJs, preauricular region, and zygoma. The patient also complained of diffuse bilateral nonsegmental paresthesia in her hands and fingers and episodes of forgetfulness.

Clinical examination revealed that the patient had a full complement of natural teeth in a class II division I relationship. Maximum interincisal opening measured 42 mm. There were no deviations or deflections in the mandibular midline on opening, closing, or protrussive movements, and mandibular excursions to the left and right were unrestricted. Pain was elicited on intrameatal and lateral palpation of the right and left TMJs, and a soft reciprocal click was auscultated in the left joint. Pain was also elicited bilaterally on palpation of the anterior, middle, and posterior fibers of the temporal, superficial and deep fibers of the masseter, medial pterygoid, lateral pterygoid, sternocleidomastoid, levator scapula, scalenes, and posterior digastric muscles. The right paravertebral and posterior cervical muscles were also painful when palpated. Transcranial radiographs of the left and right TMJs revealed no developmental or osseous abnormalities, although both condyles were displaced posterosuperiorly in the articular fossa.

Because no organic cause could be identified as a source of the patient's chief complaints, and even though her family dentist was unable to effectively manage her symptoms with a mandibular orthopedic appliance, a tentative diagnosis of myofascial pain-dysfunction syndrome was entertained. The vertical dimension and occlusal contact pattern of the patient's existing mandibular appliance was adjusted, and she was instructed to wear it continuously except for eating and cleaning. A cervical pillow, moist heat pack, soft diet, and minor tranquilizer (7.5 mg of clorazepate q.i.d.) were also prescribed, and the patient was given an appointment for reevaluation in 10 days.

When the patient returned 10 days later, she stated that the medication, cervical pillow, moist heat, soft diet, and continued use of the mandibular appliance failed to provide significant relief.

A protocol of physical therapy consisting of ultrasound, high-voltage electrogalvanic stimulation, transcutaneous electrical nerve stimulation, and trigger-point injections with 2% lidocaine without epinephrine was then instituted. The tranquilizer was discontinued and a 7-day protocol of oral prednisilone therapy was prescribed in its place. The patient's myalgic and arthralgic pain was moderately reduced during the steroid protocol, but the pain returned to its original intensity 2 days after the prednisilone trial was completed.

When the patient failed to experience appreciable improvement after receiving physical therapy 2 to 3 times weekly for 16 visits, it became obvious that a TMJ dysfunction or muscle imbalance were not the cause of her complaints. A diagnosis of Lyme disease was then considered in view of its high incidence on Long Island. When the patient was asked a series of questions focused on the presentations of Lyme disease, she denied a previous history of an unusual skin rash or to her knowledge, a tick bite.

The patient was referred to the laboratory for a Lyme serology test that subsequently proved negative for exposure to *Borrelia burgdorferei*. However, in view of the high incidence of false negative serology tests, and because not all patient's with Lyme disease develop ECM lesions, the negative serology test result was not considered conclusive, and she was referred to a group of physicians specializing in the diagnosis and treatment of Lyme disease for further evaluation.

The specialists concluded that there was a great likelihood that the patient had contracted Lyme disease, and they instituted a trial protocol of oral penicillin therapy,

which dramatically reduced her muscle and joint pain within 7 days. In view of the patient's favorable response to penicillin, the antibiotic was continued, and her pain and other associated complaints progressively diminished. The patient's antibiotic therapy was supplemented by periodic visits to our office where lidocaine injections were administered into trigger points in the craniocervical muscles, which continued to ache. Her positive response to antibiotic therapy further corroborated the diagnosis of Lyme disease, as did a subsequent Lyme serology test that was positive for exposure to *Borrelia burgdorferi.*

SUMMARY AND CONCLUSIONS

Because of the high incidence of Lyme disease, the ease with which it can be misdiagnosed, and its potential for causing irreversible neurologic or cardiac complications and fatalities if left untreated, all patients living in known epidemic areas who manifest intractable facial pain, or what appears to be a case of TMJ syndrome that does not respond to therapy, should be tested for Lyme disease. However, not all patients with active Lyme disease produce antibodies, and it is thus imperative for the clinician to obtain a detailed patient history with a focused series of questions directed at the known presentations of the disease, with specific emphasis placed on the prior appearance of an ECM lesion.

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