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Case Report

Spinal Cord Stimulation for Chronic Pain Originating from Lyme Disease

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Background: Neuropathic pain is a relatively common outcome of Lyme disease. Pain management options for these patients have been limited to pharmaceutical treatments.

Objective: We present a case of chronic pain following Lyme disease treated successfully using spinal cord stimulation (SCS).

Study Design: Case report.

Setting: Pain management clinic.

Methods: A 62-year-old patient presented with a 5-year history of bilateral foot pain following Lyme disease that failed to respond to medication and physical therapy. The patient was treated by a trial of SCS at the clinic and then implanted with a spinal cord stimulator. The Visual Analog Scale (VAS) assessed pain before and after SCS.

Results: The patient reported significant pain relief and improved foot function. The 10 point VAS score was reduced from 8–10 to 1–3.

Limitations: Single case report.

Conclusion: Spinal cord stimulation may be an effective option for relieving chronic pain originating from Lyme disease.

Key words: Spinal cord stimulation, Lyme disease, chronic pain.

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Lyme disease is a tick-borne illness caused by the spirochete Borrelia burgdorferi. The specific nervous system syndromes of Lyme disease can be divided into those with fairly abrupt onset and dramatic presentation, such as meningitis and facial nerve palsy, and those with more indolent onset and protracted course, particularly diffuse polyneuropathy. Neuropathic pain is often a prominent element of diffuse polyneuropathy. Spinal cord stimulation (SCS) has been used with varying degrees of success for the treatment of neuropathic pain syndromes, including radiculopathy, peripheral nerve injury, postherpetic neuralgia, diabetic neuropathy, and chemotherapy-induced neuropathy (1). To our knowledge, this is the first report describing the use of SCS for treatment of chronic pain as a consequence of Lyme disease.

Case Description

A 62-year-old man with a 5-year history of bilateral foot pain was referred to our clinic. Five years prior to the current examination, he presented with erythema migrans and flu-like symptoms. He received cephalexin for 10 days and became symptom-free. However, 3 weeks later he reported pain and numbness in bilateral forearms, hands, and feet. The pain was described as a sporadic, mild, cramp-like pain that continued for about half an hour after onset. The numbness was limited to the hands and feet. Two months after this second examination, the patient reported arthralgia and edema of the knees, accompanied by more intense pain in the extremities. Both serum and cerebral spinal fluid (CSF) were positive for B. burgdorferi IgG and IgM antibodies as indicated by enzyme immunoassay and confirmed by Western blot. He was diagnosed with Lyme disease and received intravenous penicillin G (24 million U/d) for 4 weeks.

Following this treatment, specific antibodies against B. burgdorferi were not detected in serum or CSF, and the arthralgia and edema of the knees, as well as pain in the upper extremities, were significantly reduced. Conversely, the foot pain persisted, characterized by recurrent attacks of cramp-like and sharp stabbing pains in both feet, with a pain level of 8–10 on the 10 point visual analog scale (VAS). Each pain episode lasted 5–6 hours and episodes recurred every 2–3 hours. During these episodes, the patient was unable to walk. In addition, his sleep and daily quality of life were severely impaired. During the 5 years prior to the current admission, carbamazepine, amitriptyline, topical capsaicin and lidocaine cream, gabapentin, and physical therapy were all tried without effect. The patient was then treated with an opioid, which did provide relief but caused intolerable lethargy. He was then prescribed 4–6 tablets daily of oxycodone/acetaminophen (5/325 mg) with only minimal pain relief. Finally, the patient was referred to our pain management clinic.

A neurological examination revealed hypoesthesia of the low extremities, no paresthesia or hyperesthesia of the skin, mildly decreased muscle tone in the calves, and bilaterally reduced patellar and plantar reflexes. Cranial and lumber contrast-enhanced magnetic resonance imaging scans were normal (as were those obtained on the first presentation 5 years prior to the current examination). Electrophysiological analyses, including measurement of nerve conduction velocity and electromyography, revealed peripheral nerve injury to both the upper and lower extremities with sensory nerve involvement. Briefly, all motor nerves tested exhibited normal latencies, amplitudes, and conduction velocities. In contrast, the amplitudes of the median and ulnar nerve responses were decreased by about 97%. The conduction velocity of the median nerve was 20% below normal, while no responses were elicited from the tibial and peroneal nerves.

A trial of spinal cord stimulation was initiated in an attempt to provide pain relief. Two quadrapolar electrode arrays (Medtronic Inc., Minneapolis, MN) were positioned in the dorsal epidural space. The needles were placed at the level of L2–3 and L3–4 and the leads advanced to the mid T12 vertebral body as viewed by anteroposterior fluoroscopy (Fig. 1). Electrical stimulation parameters were 1.2 V left side amplitude, 0.7 V right side amplitude, 250 μs pulse width, and a frequency of 100 Hz. During the trial, the patient reported greater than 80% pain relief (10 point VAS of 1–2, compared to 8–10 prior to stimulation) without oral pain medication. Furthermore, the duration of these pain episodes decreased from 4 hours to less than one hour. In addition, sleep quality and mood improved noticeably. After a successful 7-day trial, an SCS system was implanted. On subsequent follow-up visits, the patient reported complete satisfaction with pain relief and coverage. One year after implantation, VAS was 2–3. The patient continues to report satisfactory pain manage-

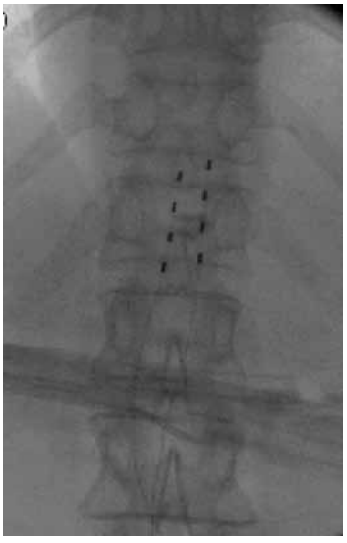

Fig. 1 *Radiograph showing epidural spinal cord stimulation lead placement*

ment and does not require opioid medications. When necessary, acetaminophen is sufficient to successfully alleviate pain.

Discussion

Lyme disease is caused by 3 pathogenic species of the tick spirochete Borrelia. It is a multisystem infectious disease, but several organ systems appear to be more commonly affected. The nervous system is the third most common site, with nervous system syndromes reported in 10–15% of infected adults (2). Neurological syndromes include both abrupt onset conditions (meningitis, facial nerve palsy, radiculoneuritis, focal encephalitis) and chronic disorders like diffuse polyneuropathy.

In our patient, the main symptom was neuropathic pain primarily restricted to the feet. While the patient's CSF was transiently positive for Borrelia antibodies during the early stage of the illness (and responsive to penicillin G), no current symptoms and signs supported central nervous damage. The pain was due to polyneuropathy (primarily afflicting the median, ulnar, tibial and peroneal nerves) caused by Lyme disease. In most Lyme disease cases, neurologic symptoms disappear following early antibiotic therapy and there is no delayed neuropathy. However, our patient's foot pain had persisted for 5 years after initial diagnosis. Studies in patients (3) and experimentally infected rhesus macaque monkeys (4) indicate peripheral nerve involvement in Lyme disease, primarily multifocal axonal damage. Indeed, measurement of nerve conduction velocity and electromyographic data supported multifocal axonal damage in our patient.

Pain resulting from multifocal axonal damage associated with diabetic polyneuropathy is treated with antidepressants, anticonvulsants, and/or opioids (5). Our patient was treated with all 3 without significant benefit. Spinal cord stimulation was first used for pain control in 1967 by neurosurgeon Dr. Norman Shealy (6). It is based on the gate-control theory (7), which postulates that SCS inhibits transmission through the pain-conducting spinothalamic tract by activating the dorsal columns. Yakhnitsa et al (8) demonstrated that SCS may induce a significant and long-lasting inhibition of both the exaggerated principal response and the after-discharges in wide-dynamic range dorsal horn neurons. Spinal cord stimulation may also alleviate chronic pain at the supraspinal level (9-12). In our patient, both the first SCS trial and subsequent implantation of a spinal cord stimulator reduced the pain score by 80%, and oral pain medication (acetaminophen) is now only required on occasion. One year following implantation, pain control is still satisfactory.

A systematic review and meta-analysis support the use of SCS in patients with refractory neuropathic back and leg pain associated with failed back surgery syndrome (Grade B evidence) and complex regional pain syndrome (CRPS) type I (Grade A evidence) and type II (Grade D evidence). Spinal cord stimulation reduces pain, improves quality of life, reduces the need for analgesics with significant adverse events (e.g., opioid-induced lethargy), and may also result in significant cost savings over time (13). For the treatment of CRPS, the International Association for the Study of Pain Expert Group recommends that SCS be initiated within 12 to 16 weeks if conventional treatments fail (14). We suggest that SCS may also be a suitable early treatment for intractable chronic pain due to Lyme disease if other therapies do not provide satisfactory pain relief.

The use of SCS presents minimal risk to Lyme disease patients. Animal models of Lyme disease indicate that although B. burgdorferi can persist despite antibiotics, they usually persist in very small numbers (15). Moreover, the electrode was inserted into the epidural space, not the subarachnoid space, so there was very little risk of central nervous system infection. Another rare but serious outcome of Lyme disease is cardiac Lyme borreliosis, characterized by acute onset atrioventricular (I–III) conduction disturbances, rhythm disturbances, and occasionally myocarditis or pericarditis (16). For these patients, before SCS, detailed preoperational evaluation of the cardiac conduction system is recommended.

Conclusion

The patient reported significant pain relief and improved foot function. The 10 point VAS score was reduced from 8–10 to 1–3.

Spinal cord stimulation may offer an effective therapeutic alternative for patients with intractable neuropathic pain resulting from Lyme disease.

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