Clinical Report

Reflex Sympathetic Dystrophy Associated With Multiple Lumbar Laminectomies

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Abstract: Reflex sympathetic dystrophy (RSD) is an often devastating chronic pain condition that can develop following relatively trivial traumatic events. The precise mechanism and predisposing factors governing the development and progression of this syndrome are not completely understood. However, RSD most commonly presents distally in an extremity following injury to the limb. Rarely has it been reported following lumbar laminectomy or ruptured lumbar disc (1, 2). Of those cases reported, the RSD was relatively acute, mild, and unilateral (1-3). We present a case of severe, chronic, bilateral RSD following multiple laminectomies and propose a possible mechanism to explain these findings. Key Words: Reflex sympathetic dystrophy-Causalgia-Sudek's atrophy-Lumbar laminectomy

CASE REPORT

A 34-year-old man presented to the Pain Control Center with a 6-year history of low back pain radiating into the left leg. The pain began acutely following a twisting fall at work. Conservative therapy consisting of muscle relaxants, narcotic analgesics and physical therapy failed to alleviate the pain. Orthopedic evaluation, computer tomography (CT) scan and myelography of the lumbar spine revealed no evidence of pathology. Discography of the L5 disc demonstrated diffuse bulging without extrusion of disc material. He was not considered a candidate for surgery or chymopapain injection and conservative modalities were resumed. Sixteen months following the injury, lumbar laminectomy at L5-S1 was performed in an attempt to reduce the pain. No significant relief was obtained following surgery or with subsequent acupuncture and transcutaneous nerve stimulation. Over the next 3 years the patient underwent bilateral L5-S1 spinal fusion, epidural steroid injections, and scleral therapy, all without improvement. Work up for multiple sclerosis and connective tissue disease which included a muscle biopsy, was negative. Repeat lumbar spine CT scan and myelography demonstrated epidural adhesions extending into the left S1 nerve root sleeve. Electromyography (EMG) revealed mild left L5-S1 radiculitis. A 3-day trial of intravenous procaine with vitamin B complex and physical therapy failed to reduce the pain. Exploratory laminectomy with lysis of

adhesions was undertaken without substantial pain relief. Over the next 2 years, two additional laminectomies for lysis of adhesions were performed without significant improvement of the patient's symptoms.

The patient presented to the Pain Clinic complaining of a constant burning ache located in the left lumbar area, radiating down the posterior left leg to the plantar aspect of his foot. Several times monthly he experienced lancinating exacerbations into the left leg described as "electrical jolts" followed by severe, debilitating muscle spasm for several hours.

Burning pain in the right leg was present, hut to a much lesser degree. He was taking 60 mg Amitriptyline and six to eight tablets containing acetamenophen 325 mg with codeine 30 mg daily for pain relief.

Physical examination revealed severe limitations of motion in the lumbar spine. The skin of both calves was pale and shiny with severe scaling. Allodynia was present in a stocking distribution to the thigh on the left and to the knee on the right. The left leg was 2.8°C colder than the right with atrophy and weakness. Deep tendon reflexes were minimally decreased on the left. Straight leg raising was positive for radicular pain at 30° and 45° in the left and right legs, respectively. There was no sensory loss noted.

A recent CT scan and myelography of the lumbar spine demonstrated persistence of the epidural adhesions at the left S1 nerve root. EMG remained positive for mild left L5-S1 radiculitis. Liquid crystal thermography confirmed the temperature discrepancy between the legs. McGill pain rating index and present pain intensity were 40 and 3, respectively. A visual analog scale consisting of a 10-cm line with I-cm graduations measured 7.5.

A retrograde differential spinal nerve block using 1 cc of 10% procaine with 1 cc of CSF indicated that the pain was primarily sympathetically mediated with a mild somatic component. Over the next 2 months, the patient underwent a series of two lumbar epidural and one caudal injection of 10 cc 0.25% bupivicaine with depomethylprednisolone, followed by five lumbar paravertebral sympathetic ganglion blocks with 15 cc of 0.25% bupivicaine. On follow-up examination I month later, he reported 25% improvement of pain, no further episodes of leg spasms, complete withdrawal from the acetomenophen with codeine and improved mobility and improved sleep. The McGill pain rating index and present pain intensity improved to 17 and 2 respectively. However, the visual analog scale remained at 7.5. The leg temperatures had equalized and the allodynia was markedly improved. At present he continues to be treated at the Pain Control Center using a multidisciplinary approach in-eluding temperature biofeedback.

DISCUSSION

RSD is a group of syndromes characterized by alteration in peripheral sympathetic activity with severe persistent burning pain, vasomotor and sudomotor changes, hyperesthesia, allodynia and trophic changes most commonly seen in a distal extremity following trauma (4). Mitchell was the first to describe the burning pain associated with peripheral nerve injuries sustained by soldiers during the Civil War, using the term causalgia (5). Sudek, in 1900, defined the osteoporosis and radiographic changes seen with RSD (6). The International Association for the Study of Pain and others have suggested using the term causalgia specifically only if a demonstrable nerve lesion is present and RSD for the syndrome in general (7).

Several theories have been proposed to explain the pathophysiology involved. The proponents of the peripheral hypothesis contend that the pain and associated physiological changes are due to a lesion present in a peripheral nerve (8, 9). At the level of the lesion, sprouting occurs from damaged axons which may form neuromas or ephapses between sympathetic efferents and sensory or visceral afferents (8). The membrane of regenerating sprouts are very unstable and may depolarize spontaneously (10). The chemosensitivity and mechanosensitivity of these membranes is altered and may be stimulated by catecholamines, touch, and blood flow (10). In addition, there is evidence suggesting that with high velocity missile injuries, large myelinated fibers can be selectively damaged. This causes a loss of the normal inhibition of the smaller unmyelinated pain fibers at the level of the dorsal horn (9). Consequently, the rostral passage of these more slowly conducted impulses creates abnormal activity in the CNS.

Those that postulate a central mechanism believe that the abnormal activity present in the dorsal horn and higher centers arises due to the convergence of peripheral nociceptors and mechanoreceptors on wide dynamic range dorsal horn cells leading to loss of inhibition or misinterpretation of stimuli (11). This may cause retrograde neuronal activity or turbulence in the dorsal horn as suggested by Sutherland (12). Melzack believes there is loss of the central biasing mechanism involving the brain stem reticular formation allowing normally inhibited impulses to reach higher centers (13). Some authorities feel that there is a common underlying mechanism to all forms of RSD involving both peripheral and central pathophysiology (4, 14).

The relationship between lumbar spine surgery and RSD has rarely been reported (1, 2, 3, 15). The syndrome has been reported following myelography and chymopapain therapy (16, 17). Previously described cases have been relatively acute, i.e., present for <6 months, mild in severity and unilateral. Our patient presented with severe, chronic, bilateral RSD following multiple lumbar laminectomies.

The mechanism of bilateral RSD in this patient could be explained on either a peripheral or a central basis. Below L3 the ventral and dorsal roots extending into the neural foramen do not normally contain preganglionic sympathetic or peripherally directed postganglionic sympathetic fibers (18). These structures would be necessarily involved in a peripheral nerve injury for classic RSD (8). However, the sinu-vertebral nerve (recurrent meningeal nerve of Luschka) innervates structures within the epidural space and carries both afferent sensory and efferent postganglionic sympathetic fibers below L3 (19). Bilateral damage to the sinu-vertebral nerve from previous surgery or epidural adhesions could have caused RSD on a peripheral basis in this patient.

A central mechanism can also be postulated. Increased afferent traffic from damaged neural structures within the epidural space may lead to abnormal activity in the dorsal horn causing a loss of the central biasing mechanism and allowing normally inhibited impulses to reach higher centers. These pathways decussate at various levels within the CNS. Thus, a unilateral lesion could, theoretically, develop into bilateral symptoms. The patient had extensive epidural adhesions from L5 to S1. These adhesions extended into the left S1 neural foramen and could have damaged the left sinu-vertebral nerve at that level. Furthermore, the patient had severe depression and this may have caused increased susceptibility to pain as suggested by Pak (20).

Several treatment modalities have been suggested for RSD including sympathetic blockade, physical therapy, TENS, acupuncture, biofeedback, and various drug therapies. However, most authorities agree that prompt diagnosis and sympathetic blockade of the involved extremity offers the best prognosis (1, 4, 7).

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