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UROLOGICAL SYMPTOMATOLOGY IN PATIENTS WITH REFLEX SYMPATHETIC DYSTROPHY

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ABSTRACT

Purpose: We determined the effect of reflex sympathetic dystrophy on lower urinary tract function.

Materials and Methods: A total of 20 consecutive patients (16 women and 4 men) with neurologically verified reflex sympathetic dystrophy was referred for voiding symptoms, including urgency, frequency, incontinence and urinary retention. No patient had had voiding symptoms before the initial trauma that induced reflex sympathetic dystrophy. Evaluation included medical history, physical examination, video urodynamic testing and cystoscopy.

Results: Mean patient age was 43.4 ± 10.2 years (range 28 to 58) and mean duration of urological symptoms was 4.9 ± 3.6 years (range 1 to 14). Urodynamic study demonstrated a mean cystometric bladder capacity of 417 ± 182 ml. (range 120 to 700). The urodynamic diagnoses included detrusor hyperreflexia in 8 patients, detrusor areflexia in 8, sensory urgency in 3 and detrusor hyperreflexia with detrusor-external sphincter dyssynergia in 1. In 4 women genuine stress urinary incontinence was also documented urodynamically.

Conclusions: Reflex sympathetic dystrophy may have a profound effect on detrusor and sphincter function.

KEY WORDS: reflex sympathetic dystrophy; cystitis; urodynamics; bladder, neurogenic; urinary incontinence

Reflex sympathetic dystrophy is a disabling syndrome characterized by severe pain with autonomic changes, such as vasomotor disturbances. Reflex sympathetic dystrophy is associated with dystrophic changes of the skin, bones or joints, which usually improve with sympathetic denervation.[1] The condition usually follows traumatic injury, such as that occurring in a motor vehicle accident. Other precipitating factors, including infection, malignancy and surgical procedures, have been implicated as precipitating factors. Although reflex sympathetic dystrophy most commonly affects the extremities, pelvic and perineal pain has also been attributed to this syndrome.[2,3] Galloway et al recently proposed reflex sympathetic dystrophy as a potential etiology for the syndrome of chronic interstitial cystitis. As urologists at an institution serving as a regional center for the treatment of reflex sympathetic dystrophy, we have noted that voiding dysfunction is a significant complaint in some patients. We report urological symptomatology in patients with reflex sympathetic dystrophy.

METHODS

A total of 20 consecutive patients (16 women and 4 men) with neurologically verified reflex sympathetic dystrophy was referred by the regional reflex sympathetic dystrophy center for urological evaluation of severe voiding symptoms, including frequency, urgency and/or urinary incontinence. In each case lower urinary tract symptomatology did not occur until after the initial injury to which the development of reflex sympathetic dystrophy was attributed. None of the patients was diagnosed with reflex sympathetic dystrophy because of urological complaints.

The original injury that caused reflex sympathetic dystrophy included blunt foot injury in 4 patients, blunt leg injury in 5, blunt hand and arm injury in 2, blunt groin injury in 1, minor lower extremities orthopedic surgery in 2, cervical and lumbosacral sprain in 5 and a fall on the coccyx without fracture in 1. None of the patients had voiding symptoms immediately after the initial injury. Urological symptoms became noticeable as reflex sympathetic dystrophy manifested and progressed.

A history was obtained, and physical examination, urinalysis and urine culture, renal ultrasound, cystoscopy using local anesthesia and video urodynamic evaluation were done. Urine cytology was performed in 6 patients with significant urgency. Urinary tract infection was ruled out in all patients before urodynamic evaluation, which included simultaneous video, pressure and electromyographic components. The urodynamic diagnoses were established based on International Continence Society standards.[5] Filling pressures were considered normal if they were less than 20 cm. water at bladder capacity. Detrusor hyperreflexia was considered when involuntary detrusor contraction occurred at 15 cm. water or more. Detrusor-external sphincter dyssynergia was defined as persistent and involuntary electromyographic activity during an involuntary detrusor contraction. Oral terazosin (5 mg.) was given nightly for 1 month to patients 16 and 19 with detrusor hyperreflexia (see table).

RESULTS

Mean patient age was 43.4 ± 10.2 years (range 28 to 58) and mean duration of urological symptoms was 4.9 ± 3.6 years (range 1 to 14). None of the patients had had voiding symptoms before the initial trauma that induced reflex sympathetic dystrophy. The distribution of urological complaints varied, including frequency, urgency, urinary retention and incontinence. Painful bladder symptoms were not significant (see table).

Endoscopic evaluation failed to reveal bladder tumors, ulceration, pinpoint bleeding at cystoscopic capacity or other intravesical pathology and, therefore, bladder biopsy was not routinely performed. Urine cytology showed no malignancies. No upper tract pathology, including hydronephrosis, nephrolithiasis or solid masses, was seen on renal ultrasound. Uro-

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Patient demographics

Pt.—Age—Sex No.	Reflex Sympathetic Dystrophy Duration (yrs.)	Previous Intervention	Chief Urinary Complaint	Cystometric Capacity (ml.)	Urodynamic Diagnosis	Urological Treatment
1 — 51 — F	1	Anticholinergics, pelvic floor stimulator	Urgency	120	Detrusor hyperreflexia	Epidural block
2 — 39 — F	9	Multiple orthopedic operations, dorsal column stimulator	Urinary retention	675	Detrusor areflexia	Intermittent self-catheterization
3 — 28 — F	1	Sympathetic blocks	Urge incontinence	300	Detrusor hyperreflexia, urethral hypermobility stress urinary incontinence	Anticholinergics, pelvic floor exercises
4 — 53 — M	6	Anticholinergics	Urgency, post-void fullness	240	Detrusor hyperreflexia, detrusor-external sphincter dyssynergia	External sphincterotomy
5 — 45 — F	5	Epidural blocks	Urge incontinence	200	Detrusor hyperreflexia	Anticholinergics, timed voiding
6 — 50 — M	3	Lumbar laminectomy	Urinary retention	500	Detrusor areflexia	Intermittent self-catheterization
7 — 31 — F	14	Dorsal column stimulator	Mixed incontinence	200	Detrusor hyperreflexia, urethral hypermobility stress urinary incontinence	Anticholinergics, pelvic floor exercises
8 — 55 — F	7	Multiple orthopedic operations	Urgency	250	Sensory urgency	Anticholinergics, timed voiding
9 — 42 — F	1	Epidural block	Urinary retention	570	Detrusor areflexia	Intermittent self-catheterization
10 — 28 — M	4	Epidural blocks	Urgency, urge incontinence	300	Detrusor hyperreflexia	Anticholinergics, timed voiding
11 — 33 — F	8	Laminectomy	Urgency	263	Sensory urgency	Anticholinergics, timed voiding
12 — 58 — M	7	Multiple orthopedic operations	Nocturia 10 times/night	563	Detrusor areflexia	Intermittent self-catheterization
13 — 54 — F	10	Multiple orthopedic operations	Frequency, dysuria	600	Sensory urgency	Anticholinergics, timed voiding, fluid restriction
14 — 50 — F	7	Multiple orthopedic operations	Urinary retention	600	Detrusor areflexia	Intermittent self-catheterization
15 — 32 — F	4	Multiple orthopedic operations	Urinary retention	700	Detrusor areflexia	Intermittent self-catheterization
16 — 51 — F	5	Total abdominal hysterectomy	Urgency	370	Detrusor hyperreflexia, urethral hypermobility stress urinary incontinence	Anticholinergics, terazosin
17 — 42 — F	1	Epidural block	Urinary retention	570	Detrusor areflexia	Intermittent self-catheterization
18 — 29 — F	2	Lumbar laminectomy	Urinary retention	600	Detrusor areflexia	Intermittent self-catheterization
19 — 42 — F	1	Sympathetic blocks	Urge, sensation of incomplete emptying	416	Detrusor hyperreflexia	Anticholinergics, terazosin, timed voiding
20 — 55 — F	2	Epidural blocks	Urgency, severe incontinence	300	Detrusor hyperreflexia, intrinsic sphincteric deficiency stress urinary incontinence	Anticholinergics, periurethral collagen injection

dynamic evaluation demonstrated detrusor hyperreflexia in 8 patients, detrusor areflexia in 8, sensory urgency in 3 and detrusor hyperreflexia with detrusor-external sphincter dyssynergia in 1. Mean cystometric bladder capacity was 417 ± 182 ml. (range 120 to 700). In 4 women genuine stress urinary incontinence was also documented urodynamically.

Urological treatment included anticholinergic medications, timed voiding, moderate fluid restriction, pelvic floor exercises, terazosin, periurethral collagen injection, an epidural block and external sphincterotomy. The 2 women who were given terazosin for voiding symptoms tolerated the drug well without side effects but they reported no subjective improvement.

DISCUSSION

Pain, edema and autonomic dysfunction are the chief symptoms of early reflex sympathetic dystrophy. In later stages movement disorders and topical changes are prominent. Bone changes, ranging from osteopenia to marked demineralization or ankylosis, are often described.[1] Reflex sympathetic dystrophy differs from autonomic dysreflexia, which is the sympathetic nervous system dysfunction most commonly encountered by urologists in patients with spinal cord injury above the T7 level.[6]

In cases of autonomic dysreflexia an acute noxious stimulus applied to the body below the level of the injury, such as bladder or bowel distension, results in a sudden massive unchecked sympathetic discharge, which causes vasoconstriction below the level of the spinal cord injury. It manifests as a dramatic increase in systemic blood pressure. Parasympathetic reaction above the level of the lesion may cause facial flushing, diaphoresis and reflex bradycardia.[7]

In contrast, reflex sympathetic dystrophy is a chronic sympathetic nervous system dysfunction, which progresses steadily in a series of identifiable stages.[1] The initiating event is often a traumatic injury, such as the sudden deceleration encountered in a motor vehicle accident. Stage I reflex sympathetic dystrophy, the acute stage, is characterized by pain that is out of proportion to that expected for the initiating injury. It is usually described as localized, deep burning or aching exacerbated by movement or emotional disturbance. Edema, hyperthermia or hypothermia and increased nail growth are common. Roentgenography may show evidence of demineralization even at this early stage.

In stage II reflex sympathetic dystrophy, the dystrophic phase, burning pain increases and emotional disturbances are common, such as anxiety and depression. Edematous

areas may progress to the point of induration. The skin may appear shiny, bronzed and cool with cyanosis and mottling. Alopecia may develop in previously hair-bearing skin while the nails become severely dull and brittle.

Stage III, the atrophic phase, is characterized by further increases of pain resulting in severe, often crippling hyperasthesia. Motor changes are common, including weakness, tremor, spasm, dystonia and increased deep tendon reflexes. The fascial tissues lose thickness, and cartilage, muscle and joints atrophy, resulting in contracture of the extremities. Roentgenography often reveals marked bony demineralization in the fully manifested syndrome.

The diagnosis of reflex sympathetic dystrophy is clinical and currently there is no consensus for its diagnosis.[8] Roentgenography, bone scintigraphy and differential sympathetic neural blockade have been used to support the diagnosis. A number of pain syndromes, such as causalgia, Sudeck's atrophy and algoneurodystrophy, are considered clinical variants of reflex sympathetic dystrophy.[1]

The etiology and pathogenesis of this disorder are unclear. Numerous theories have been suggested to account for the manifestations of the disease process. The common features of reflex sympathetic dystrophy (burning pain, hyperalgesia and dystrophic changes accompanied by vasomotor disturbances after nerve plexus or soft tissue injury) may improve with sympathetic intervention. Effective therapy includes the blockade of sympathetic activity using epidural or regional injections, systemic sympathetic antagonists or surgical sympathectomy.[1]

There are few previous reports of the urological manifestations of reflex sympathetic dystrophy. To our knowledge no previous evaluations of patients with reflex sympathetic dystrophy have included a thorough documentation of urodynamic diagnoses of the voiding dysfunction that may be associated with reflex sympathetic dystrophy. Chalkley et al described a case of suspected reflex sympathetic dystrophy of the penis that developed 1 year after transurethral prostatectomy.[9] The symptoms were disabling burning pain in the penis accompanied by penile hypothermia. This condition was successfully treated using epidural nerve blocks. Olson reported on another patient with apparent reflex sympathetic dystrophy who had a severe perineal and penile burning sensation after the treatment of metastatic colon cancer with surgery and radiation.[2] Symptoms were alleviated by bilateral lumbar sacral sympathectomy. Recently Stevens et al reported on a patient with a sympathetically maintained pain syndrome who was given terazosin orally.[10] The condition responded well to sympathetic blocks for short periods but not to oral opioids, anti-inflammatory medications and muscle relaxants. Symptoms rapidly resolved with the initiation of terazosin therapy. The implication of abnormal α-receptor activity in these chronic pain syndromes is supported by the finding of increased responses of various α-adrenoceptors to locally infused norepinephrine in the affected compared to unaffected limbs and the normal contralateral limb of these patients.[11]

Ghostine et al reported on 40 patients with sympathetically mediated pain who were treated with 40 to 120 mg. phenoxybenzamine for 6 to 8 weeks with significant resolution of symptoms.[12] Followup ranged from 6 months to 6 years with no reported recurrences. When reflex sympathetic dystrophy has been relieved by sympathetectomy, intradermal injection of phenylephrine hydrochloride (predominantly α1 agonist) but not clonidine hydrochloride (predominantly α2 agonist) into the formerly painful area causes reactivation of pain, suggesting that α1 receptors may mediate sympathetically maintained pain. Although most common in extremities, manifestations of reflex sympathetic dystrophy have been described in the head, neck and trunk. Terazosin is reportedly effective in the treatment of autonomic dysreflexia[13] but it was not effective for empirical therapy of detrusor hyperreflexia in the 2 women with reflex sympathetic dystrophy in our series.

To our knowledge our report represents the first series of urodynamically verified neurourological dysfunction associated with reflex sympathetic dystrophy. Our study illustrates that significant lower urinary tract dysfunction may develop as a direct result of or in association with sympathetic nervous system dysfunction. Our patients had complex histories, and underwent a number of invasive and noninvasive treatment modalities for pain. It is possible that previous injuries and invasive therapies rather than reflex sympathetic dystrophy alone contributed to the voiding symptoms. We attempted to include only those in whom voiding symptoms developed concurrently with progressive reflex sympathetic dystrophy symptoms. Patients with reflex sympathetic dystrophy but more severe initial injury, causing herniated intervertebral disks in the cervical, thoracic, lumbar or sacral spine, were not included. Furthermore, patients with acute development of voiding symptoms after back surgery were not included. Treatment for reflex sympathetic dystrophy, including anticholinergic therapy for patients with detrusor hyperreflexia and intermittent catheterization programs for those with detrusor areflexia, was based on urodynamic results and was largely successful. The 2 most common treatment options in our series were anticholinergic drugs and intermittent self-catheterization.[14]

A theory of the pathogenesis of reflex sympathetic dystrophy pain is that tissue injury sensitizes C-fiber nociceptors via α1-adrenoceptors. In undamaged nerves sympathetic stimulation has a suppressive effect on C-fiber activity. Involved tissue may have an extra increase in adrenoceptors to increase the discharge rate in response to sympathetic stimulation.[15] We are presently studying intravesical capsaicin, a C-fiber neurotoxin, for the treatment of reflex sympathetic dystrophy and interstitial cystitis.

The urological symptoms and urodynamic findings of the reflex sympathetic dystrophy patients in our study are similar to those of other neurologically impaired patients with voiding symptoms. Notably pelvic or suprapubic pain was not a significant complaint. There is no proved explanation for the urological dysfunction among our patients. However, our study suggests that sympathetic dysfunction can result in the development of neurogenic lower urinary tract dysfunction.

Parallels may be drawn to the autonomic neuropathy of diabetes mellitus, which can affect the bladder and urethral sphincter. Detrusor hyperreflexia and especially areflexia are common urological sequelae of diabetes mellitus.[16, 17] The implication of detrusor hyperreflexia in diabetes cystopathy is that cortical or spinal regulatory tracts have been affected.

In conclusion, reflex sympathetic dystrophy may have a profound effect on detrusor and sphincter function. The spectrum and severity of lower tract dysfunction in reflex sympathetic dystrophy patients vary markedly.

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Spinal Cord Stimulation in the Treatment of Complex Regional Pain Syndrome (CRPS) of the Lower Extremity: A Case Report

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Complex regional pain syndrome (CRPS) is a condition that is often associated with the extremities. This chronic pain syndrome, when localized to the lower extremity, includes peripheral changes such as edema, temperature alterations, limited range of motion, loss of or excessive perspiration, pain out of proportion to any stimulus, and trophic alterations of the skin, hair, and nails. In this report, we describe the case of a patient who developed complex regional pain syndrome following an ankle injury and surgery. This case report highlights treatment options that are available to patients experiencing complex regional pain, including the use of a spinal cord stimulator. Level of Clinical Evidence: 4 (The Journal of Foot & Ankle Surgery 48(1):52–55, 2009)

Key Words: causalgia, complex regional pain syndrome, CRPS, reflex sympathetic dystrophy, spinal cord stimulation

Complex regional pain syndrome (CRPS), formerly known as reflex sympathetic dystrophy, is often a devastating neuropathic condition that has, in recent years, been recognized with increasing frequency in the lower extremities. Patients with CRPS, who are not diagnosed and treated in a timely fashion, may worsen to such a degree that the individual may never return to a satisfactory and productive life. Spinal cord stimulation (SCS) has been used in the treatment of neuropathic pain since 1967 (1). Although it was infrequently used during the 1970s and 1980s, SCS has gained in popularity over the past 15 years because of technological progress that has directly impacted the use of implantable systems. Before the use of SCS, patients who failed to respond to conservative modalities such as physical therapy and pharmacological interventions were left to deal with continued and debilitating pain. Currently, the treatment of CRPS includes early intervention and implantation of a spinal cord stimulator, as soon as it becomes apparent that less invasive modalities fail to diminish and provide pain relief (2). Unfortunately, delaying the diagnosis and treatment of CRPS can adversely affect the response to treatment (2). Many clinicians feel that an early and accurate diagnosis of the condition provides the greatest chance of a full recovery and an improved quality of life.

Case Report

A 69-year-old female presented to her podiatric physician for a work-related crush injury to the left foot. The injury was initially treated by a different physician, who had been appointed by the patient's workers' compensation adjustor. The initial surgeon had already performed a primary repair of the patient's ruptured lateral ankle ligaments. After the initial surgical intervention and subsequent recovery, the patient was discharged from the previous surgeon's practice. Several months thereafter, she presented to our practice with a complaint of continued pain and instability involving her left ankle. Following historical interview and physical examination, a diagnosis of chronic left ankle lateral ligamentous instability was made and, after considering treatment options, the patient underwent revisional lateral ankle

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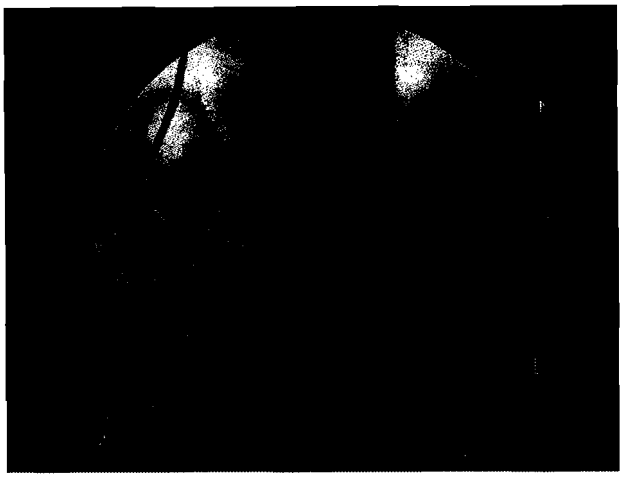

FIGURE 1 Anteroposterior fluoroscopic view showing spinal cord stimulator lead in the epidural space at the cephalic aspect of the 10th thoracic vertebra.

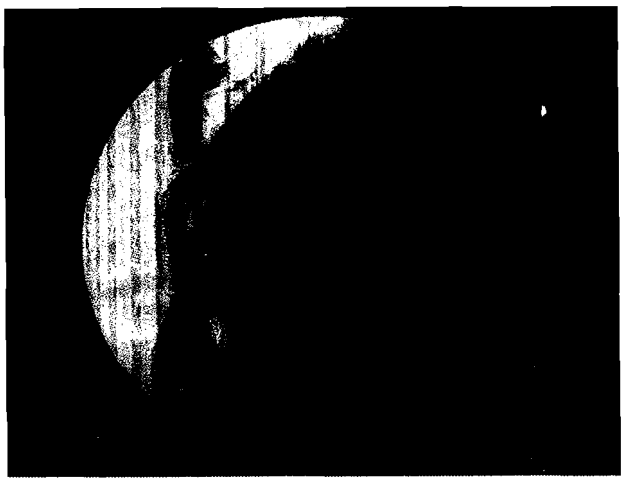

FIGURE 2 Lateral fluoroscopic view showing the spinal cord stimulator lead in the posterior epidural space.

ligamentous reconstruction using a split peroneus brevis tendon graft. Initially, the patient appeared to respond favorably to the procedure and temporarily returned to work. Over time, however, she displayed increased anxiety and her left foot exhibited persistently worsening edema and discoloration. She subsequently reported progressive burning pain in the left foot. Despite extensive physical therapy, anxiolytic therapy, peripheral nerve blocks, and adjunctive pharmacological management, her condition failed to improve. The presence of hyperalgesia, edema, allodynia, and skin discoloration, raised concerns about the possibility of CRPS, and the patient was referred to an interventional pain medicine physician for further evaluation and treatment at approximately 4 weeks following the second left ankle operation.

At the time of her initial evaluation with the pain medicine specialist, a diagnosis of CRPS was confirmed. Her pre-procedural subjective visual analog scale (VAS) pain core was reported by the patient to be 8 out of 10 in intensity. Chronic pain therapy was initiated with the use of an anticonvulsant (gabapentin titrated over 3 weeks, up to 600 mg orally 3 times daily), a tricyclic antidepressant (nortriptylene, 50 mg orally at bedtime), topical compounded analgesic cream (ketamine, clonidine, capsaicin amitriptyline, and ketoprofen applied to the affected area 3 to 5 times daily), an opioid analgesic (methadone, 5 mg orally every 8 hours), and 4 separate lumbar sympathetic nerve blocks (LSB). These treatment interventions failed to provide satisfactory pain relief after 12 weeks, and she experienced a number of adverse side effects related to the medications. Moreover, she experienced only temporary pain relief following LSB. In an effort to improve her response to therapy, the decision was made to perform a 5-day SCS trial using the Precision Plus (Boston Scientific Corporation, Valencia, CA) SCS. The patient responded favorably to the SCS trial, reporting a 75% reduction of her pain. Based on her response to the SCS trial, a permanent SCS (Figures 1 and 2) was implanted 8 weeks following removal of the trial leads. Over the ensuing several weeks, the patient reported clinically significant pain reduction, improved sleep, and increased activity level after implantation of the permanent SCS system. At the time of her last follow-up evaluation, 6 months following implantation of the permanent SCS, she related that her subjective VAS pain scale score was 2 out of 10.

Discussion

CRPS can be debilitating, and is often difficult to treat. The controversial role of sympathetic nervous system involvement in reflex sympathetic dystrophy (RSD), lack of evidence for a reflex mechanism, and the small subgroup of patients who present with dystrophy, led to a revision of the terminology used to describe this condition (3) and, in 1994, the International Association for the Study of Pain (IASP) changed the terminology so that RSD would thereafter be referred to as complex regional pain syndrome type I (CRPS I), and causalgia would thereafter be referred to as CRPS type II (4).

It is interesting, moreover, to note that although CRPS I has been a recognized clinical entity for more than a century, even today early diagnosis is often missed. One of the key clinical features of CRPS I is the presence of pain out of proportion to the stimulus, with a nondermatomal distribution. It was once thought that these patients had a psychogenic disorder, but to date no empirical evidence has substantiated this claim (5). A retrospective study found that patients with CRPS had, on average, seen 4.8 different

physicians and had received an average of 5 different types of treatments before being referred to a pain center, and the mean duration of symptoms before evaluation by a pain specialist was approximately 30 months (6).

Currently, there is limited epidemiological data pertaining to the incidence of CRPS. A population-based study at the Mayo Clinic found that the median age of onset of CRPS was 46 years, and that it occurred 4 times more frequently in females than males (7). Furthermore, the development of CRPS is usually associated with trauma or surgery, although the condition can arise without any precipitating traumatic event. According to the IASP, the clinical features to be taken into account for diagnosing CRPS type I include the presence of regional and continued pain disproportionate to any inciting event, sensory changes such as allodynia and hyperalgesia, sudomotor alterations, edema, vasomotor instability (temperature changes and skin discoloration), and exclusion of any other condition that would account for the above-mentioned signs and symptoms. CRPS II (causalgia) includes the aforementioned features accompanied by a specific peripheral nerve lesion (3), and the sensory changes that usually accompany this diagnosis include burning, aching, pain to light touch, and an exaggerated response to noxious stimuli. As CRPS persists, trophic nail and hair alterations, as well as skeletal muscle weakness, tremor, and dystonia may develop, and radiographs often display patchy demineralization of long bones. In severe cases involving the lower extremity, contractures may be observed, and dystonia may present with resultant equinovarus position of the foot. Still further, sudomotor dysfunction, manifested as hyper- or hypohidrosis (3), as well as peripheral edema, which conveys a glossy, swollen appearance.

The signs and symptoms of CRPS result from dysfunction of the peripheral and central components of the nervous system. Nociceptors are peripheral nerve fibers that transmit pain signals to the spinal cord. These fibers are capable of releasing inflammatory mediators, such as substance P and calcitonin gene-related peptides, into the peripheral tissues. The release of these mediators is believed to trigger neurogenic inflammation through capillary leakage and activation of inflammatory cells in peripheral tissues (8). Abnormal interactions subsequently develop between the sensory and mechanical nociceptors that are responsible for normal sensations such as touch and vibration. Allodynia results when abnormal connections are established between the axons of nociceptors and mechanosensory fibers, resulting in nociceptor excitation from tactile fiber stimulation (8). These abnormal interactions result in the interpretation of nonpainful stimuli as painful, with amplified pain perception following an injury; such interactions can evolve following even relatively minor trauma.

Various opioids and adjuvant medications have been successfully used to treat CRPS. Adjuvant medications include anticonvulsants, tricyclic antidepressants, nonsteroidal anti-inflammatory drugs (NSAIDS), corticosteroids, and topical compounded creams. Intrathecal therapy may also be effective in certain cases where intolerable side effects occur with high doses of opioids. Sympathetic nerve blocks have also been used to reduce the pain associated with CRPS, to facilitate physical therapy, and to aid in the determination of whether or not the sympathetic nervous system is involved in the maintenance of pain. Sympathetic blocks are performed under fluoroscopic guidance by injecting a local anesthetic agent on the stellate ganglion for treatment of the upper extremity, or on the lumbar sympathetic chain for treatment of the lower extremities. In some cases, surgical sympathectomy has been shown to be beneficial in the treatment of sympathetically maintained pain (9); however, this procedure is generally reserved for cases in which extensive conservative treatment has failed. Physical therapy, as well as cognitive and behavioral therapies are important adjunct modalities that should also be considered in the multidisciplinary approach to the treatment of CRPS.

Since its first use in 1967 by Shealy et al (1), SCS has been widely used for the treatment of chronic pain. A spinal cord stimulator is an implantable medical device that generates electrical pulses that stimulate the dorsal column fibers of the spinal cord. The electrical current produced by an implantable pulse generator (IPG) is carried through either a single- or dual-lead cathode to the spinal cord. The location of the lead(s) in the epidural space effects stimulation of the desired dermatome. Following the SCS trial, the percutaneous leads are removed from the patient and, as such, are not considered a permanent implant. SCS technology has been used effectively in the management of chronic pain related to diabetic peripheral neuropathy (DPN), failed back surgery syndrome (FBSS), CRPS, phantom limb pain, postamputation stump pain, and arachnoiditis; and, after more than 30 years of experience, SCS has come to be a first line intervention for cases of CRPS that have not satisfactorily responded after 12 to 16 weeks of conservative therapy (2).

Foot and ankle surgeons faced with patients who fail to progress as anticipated after surgical intervention, and who display persistent pain, pain out of proportion to a stimulus, burning sensation, edema, and limited range of motion, should be alerted to the possibility that the patient may be developing CRPS. If CRPS is suspected, then consideration should be given to the potential benefits of a timely referral to an interventional pain medicine physician for further evaluation and potential management. The treatment of CRPS is considered by many clinicians to be most effective when it is multifaceted and undertaken as early as possible. As demonstrated in the patient described in this case report, SCS can provide a safe and minimally invasive modality for the treatment of CRPS.

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