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CASE REPORT
Intractable Pruritus After Traumatic Spinal Cord Injury

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Abstract

Background: This report describes a young woman with incomplete traumatic cervical spinal cord injury and intractable pruritus involving her dorsal forearm.

Method: Case report.

Findings: Anatomic distribution of the pruritus corresponded to the dermatomal distribution of her level of spinal cord injury and vertebral fusion. Symptoms were attributed to the spinal cord injury and possible cervical root injury. Pruritus was refractory to all treatments, including topical lidocaine, gabapentin, transcutaneous electrical nerve stimulation, intravenous Bier block, stellate ganglion block, and acupuncture.

Conclusions: Further understanding of neuropathic pruritus is needed. Diagnostic workup of intractable pruritus should include advanced imaging to detect ongoing nerve root compression. If diagnostic studies suggest radiculopathy, epidural steroid injection should be considered. Because the autonomic nervous system may be involved in complex chronic pain or pruritic syndromes, sympatholysis via such techniques as stellate ganglion block might be effective.

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Key Words: Tetraplegia; Brachioradial pruritus; Pain, neuropathic; Pruritus, neuropathic; Spinal cord injuries; Gabapentin; Lidocaine; Transcutaneous electrical nerve stimulation; Bier block, intravenous; Stellate ganglion block; Ropivacaine

INTRODUCTION

In the 17th century, Samuel Hafenreffer defined pruritus as an ''unpleasant sensation that elicits the desire or reflex to scratch.'' Chronic pruritus is likened to pain due to its complex nature and unpleasant sensory experience (1). The distinguishing factor between chronic pruritus and pain is the behavior that the two perceptions evoke. Pain triggers withdrawal, whereas pruritus triggers both reflex and conscious mechanical stimulation (eg, scratching) of the affected area (2).

Pathologic pruritus occurs most commonly in the setting of damage to peripheral and central sensorineural cells that carry pruritus signals. Damage anywhere along this pathway may cause neuropathic pruritus, which may occur independently or be accompanied by neuropathic pain (2,3). Notalgia paresthetica, a peripheral sensory neuropathy characterized by asymmetrical pruritus on the back within the distribution of spinal nerves T2 through T6 (4), and postherpetic neuralgia, a poorly studied and understood chronic pruritus observed in patients who have had shingles often affecting the head or neck and colocalizing with reduction in protective pain sensation (5), are, perhaps, the best known examples of neuropathic pruritus (2). Reports of pruritus associated with neuropathic features (eg, burning, stinging, pain) support a neurogenic origin (6).

There are approximately 30 reported cases of central neuropathic pruritus caused by multiple sclerosis, brain tumor, abscesses, and strokes (2). Although pruritus has been associated with spinal cord lesions (multiple sclerosis, pilocytic astrocytoma, and cavernous hemangioma) (2), to our knowledge there have been no reports of pruritus associated with traumatic spinal cord injury (SCI).

CASE REPORT

An 18-year-old woman with C6 AIS C (C6 motor and sensory level bilaterally at acute rehabilitation discharge) tetraplegia sustained a cervical flexion distraction injury in a motor vehicle collision. She sustained an unstable anterior subluxation disrupting the posterior longitudinal ligament, a right-sided paramedian disk protrusion, and a displaced right C6 transverse process fracture. Two

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days later, she underwent open reduction of C6-C7 3-column spinal injury and C6-C7 posterior instrumentation and fusion. Three months after the injury (while completing inpatient rehabilitation), she complained of episodic aching in her left upper extremity in the C6 dermatomal distribution. She was administered gabapentin 300 mg nightly but was unable to tolerate a daytime dose due to its sedative effects. Seven months after the injury, she presented to the clinic complaining of bilateral lower extremity ''burning'' pain, left upper extremity ''itching,'' and right upper extremity ''hypersensitivity to touch.'' She was already taking 1,200 mg of gabapentin 4 times daily (higher than the recommended maximum daily dosage) and occasional bedtime oxycodone and using a transcutaneous electrical nerve stimulation unit for neuropathic pain. These therapies were providing moderate relief.

Ten months after the injury, she returned to the clinic complaining of constant pruritus in her left arm that was most severe at night and not relieved with use of the transcutaneous electrical nerve stimulation unit. She was given lidocaine patches to be applied to the pruritic area of her left forearm. Seventeen months after the injury, she reported that her neuropathic pain had responded well to the 1,200 mg of gabapentin 3 times daily, but she continued to experience intractable itching of her dorsal left forearm and digits in C6 and C7 dermatomal distributions. Her itching was refractory to gabapentin, transcutaneous electrical nerve stimulation and lidocaine patches. Subsequently, she underwent intravenous Bier block with injection of a solution containing 200 mg of lidocaine, 100 μg of clonidine, 50 mg of methylprednisolone, and normal saline (total volume = 60 mL). She tolerated this procedure well but experienced no relief. Eight days after the Bier block, she underwent left stellate ganglion block, during which 10 mL of ropivacaine was injected. The procedure was tolerated well, and a definitive change in the temperature of her left upper extremity with some piloerection and conjunctival injection was noted. During the weeks surrounding these blocks, she also underwent one acupuncture therapy utilizing both laser acupuncture and needle stimulation. She reported relief from pruritus up to 2 days after stellate ganglion block, but it returned promptly.

Nineteen months after the injury, she still complained of left arm itching, describing it as 3 out of 10 in severity during the day and 10 out of 10 during the night. Approximately 29 months after the injury, still plagued with unrelenting pruritus, she underwent stellate ganglion catheter placement for 1 week based on the temporary relief achieved with the previous stellate ganglion block. Upon initiation of ropivacaine infusion through the catheter, she experienced some hoarseness. The infusion was stopped, the catheter was pulled back about 1 cm, and the infusion was restarted without return of hoarseness. Symptoms were controlled about 4 weeks, but the pruritus eventually returned at a milder level. Concurrently, her bedtime gabapentin dose was increased to 1,500 mg. Thirty-four months after the injury, she continued to suffer from the same intractable pruritus.

DISCUSSION

Both the etiology and treatment for neuropathic pruritus of seemingly central origin remain elusive. Our patient's symptoms were presumed to be due to her known central spinal cord lesion.

This clinical presentation has many similarities to a well-documented disorder known as brachioradial pruritus (BRP), a neuropathic pruritus localized on the dorsolateral aspect of the upper arm (7). There is debate about the cause of BRP. Some consider it a type of photodermatosis that occurs only on sun-exposed areas of the body, whereas others view it as a symptom of cervical radiculopathy (7,8). Current theories suggest that neurological damage from peripheral nerves (eg, solar radiation, local injury) and from central sensory pathways (eg, cervical spine disease) contributes to the variable presentation of the pruritus in individual patients (6,7).

A total of 30 cases of BRP associated with spinal cord disease have been reported in the medical literature (6). In 13 patients, location of pruritus correlated with alteration in sensory innervation of the involved segment of the skin; in 2 of these 30 patients, symptoms were produced by a spinal cord tumor (6). Tait et al (9) described 6 patients with BRP and a history of neck problems for whom cervical spine manipulation was helpful in BRP management. Changes in neck position triggered symptoms in 1 patient with BRP, and whiplash injury was a precipitant in 2 others (6). Heyl (10) argued that BRP was a result of cervical nerve compression after reporting 14 patients with BRP, none claiming aggravation from sun exposure. Four of these patients showed radiographic evidence of degenerative changes and osteoarthritis between the fourth and seventh cervical vertebrae; 1 already underwent C6-C7 fusion, and 3 achieved significant improvement with physical therapy and traction. Heyl suggested that for patients in whom osteoarthritic changes cannot be demonstrated, BRP might be due to compression of nerves by other structures. The symptomatic relief that patients experienced with cervical spine manipulation and traction suggests that cervical radiculopathy may be a major contributor to brachioradial pruritus.

There is substantial evidence that radiculopathy causes another form of pathologic pruritus, anogenital pruritus. In a study by Cohen et al (11), researchers sought a neuropathic etiology in patients with anogenital pruritus. After ruling out other causes for anogenital pruritus, radiographs of the lower spine and sacrum and electrodiagnostics were performed on 20 patients with ''idiopathic'' anogenital pruritus. In 15 patients (75%) nerve conduction studies demonstrated abnormal or not

excitable F-responses, interpreted as lumbosacral radiculopathy. Sixteen patients (80%) with ''idiopathic'' anogenital pruritus had lumbosacral radiculopathy, representing nerve or nerve root compression at the level of L4 to S2 vertebrae. The location of the pruritus was correlated with location of the neuropathic changes, as observed in electrodiagnostic studies. It is plausible that lumbosacral nerve or nerve root compression may manifest as pain in some patients and as pruritus in others. Researchers then performed paravertebral injections with a mixture of triamcinolone acetate and lidocaine at the level of L5-S1 that resulted in a significant reduction in pruritus 2 to 4 weeks after the injections.

The treatment of neuropathic pruritus is diverse and often disappointing. Potential therapies include topical agents (steroids, capsaicin cream, antihistamines, lidocaine patch, lidocaine/prilocaine cream), oral therapy that includes anti-inflammatory agents, tricyclic antidepressants (nortriptyline, amitriptyline, desipramine), selective serotonin reuptake inhibitors, antiepileptics (oxcarbazepine, carbamazepine, zonisamide, tiagabine, lamotrigine, and levetiracetam), nerve root blocks, intravenous lidocaine, local anesthetic blockade of the stellate ganglion, transcutaneous electrical nerve stimulation, acupuncture, cervical spine manipulation, neck traction, physical therapy, surgical resection of a cervical rib, and sunlight avoidance (2,6,7). Unfortunately, most of these are not very effective. There are isolated reports of successful treatment of neuropathic pruritus with lamotrigine and carbamazepine (7,12), but on the whole there seems to be little documented evidence for the efficacy of these treatments.

Substantial data suggest radiculopathy plays a major role in the etiology of some forms of neuropathic pruritus. Our patient suffered intractable pruritus in the left arm in the dermatomal distribution that corresponded to her level of traumatic SCI. One may assume that in addition to the SCI, she suffered some root level injury. Unfortunately, diagnosing a radiculopathy after SCI is exceedingly difficult. In a study seeking to electrophysiologically characterize the injury zone, Berman et al (13) found that cervical SCIs cause a loss of motor axons in regions several segments caudal to the rostral injury level. For example, in patients with C5 or higher injury levels, lower motor neuron damage, evident electrophysiologically, was most commonly observed several segments lower in the C8/T1 level. This demonstration makes it difficult to rely on physical findings to help diagnose radiculopathy. Obtaining relief from a cervical epidural steroid injection, however, would lend support to a radicular etiology of the symptoms.

Stellate ganglion block and intravenous Bier block have been used as therapeutic interventions in conditions, such as Complex Regional Pain Syndrome, that tend to have a complex etiology involving peripheral and central nervous system etiology (14). Bier block provides both peripheral block and sympatholysis. Involvement of autonomic nervous system in complex chronic pain syndrome (akin to chronic pruritus) led us to believe that sympatholysis using either of these techniques could provide her therapeutic benefits. Negative response to Bier block ruled out peripheral mechanisms in the pathogenesis of her pruritus. Amelioration of itch after stellate ganglion block may point towards a role of sympatholysis in these patients.

Hope exists for better treatments for neuropathic itch in the future. Pain is known to inhibit itch, and it is commonly experienced that an itch sensation can be reduced by the mildly painful sensation caused by scratching (1). Analgesia reduces this inhibition and, consequently, enhances itch. Conversely, certain opioid antagonists have been found to have antipruritic effects in experimental itch studies (1). New research demonstrates that gastrin-releasing peptide receptor plays an important part in mediating itch sensation in the dorsal horn of the spinal cord and, thus, may provide a central therapeutic target for antipruritic drug development (15).

CONCLUSION

Further understanding of neuropathic pruritus is needed. However, recommendations for the diagnostic workup for intractable pruritus almost certainly should include advanced imaging, such as magnetic resonance imaging, to detect ongoing nerve root compression.

Electrodiagnostics are unlikely to help identify radiculopathy in patients with SCI but may be diagnostic for patients without such injuries. If diagnostic studies suggest radiculopathy, then an epidural steroid injection should be considered, because this treatment has been effective for patients with anogenital pruritus due to lumbosacral radiculopathy. Because the autonomic nervous system is thought to have involvement in complex chronic pain or pruritic syndromes, sympatholysis, via such techniques as stellate ganglion block, might also be tried. In the case described here, this procedure provided temporary relief.

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