The American College of Chiropractic Orthopedists

e-JACCO
Vol. 30 No.2

JACCO

April 2010
Volume 30, Number 2
A Publication of the
American College of Chiropractic Orthopedists

Editor/Publisher
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Greetings …

from the Board of Directors and contributing members of the American College of Chiropractic Orthopedists. We have been working quite diligently on the upcoming conference: *Clinical Manifestations of Cervical Spine Injury*. This 24 CE neuron stimulating conference is April 23-25th, 2010 at Harvey's in South Lake Tahoe, Nevada. Once again, we have thoughtfully put together a conference which includes many of our professions top doctors and practitioners. We are fortunate in our profession to have doctors who are committed to continuing the excellent research and publishing literature that not only is pertinent to our profession but to the health profession as a whole.

Our slate of speakers can be viewed, along with their curriculum vitae at our website www.accoweb.org .

There is still time to reserve your seat and hotel room at the famous **Harvey's Resort** at the beautiful South Lake Tahoe. Brush away the midwinter blues, jazz yourself up for a weekend filled with stimulating conversation, professional comradery, great food and, as always, information that you can input into your clinic on Monday morning!

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Respectfully Yours in Health.

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Deformation of the Thecal Sac by Lumbosacral Epidural Lipomatosis

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Abstract

Objective: This paper describes a case of a 60 year old female presenting with low back pain and radicular symptoms to the right foot. A diagnosis of lumbosacral epidural lipomatosis was made following magnetic resonance imaging. A clinical presentation of the case, pathophysiology and treatment options are described with the intent to assist clinicians in effectively diagnosing and treating patients with spinal epidural lipomatosis.

Method: Case report of a 60 year old female patient with magnetic resonance imaging consistent with spinal epidural lipomatosis.

Discussion: Spinal epidural lipomatosis is defined as excessive, unencapsulated epidural fat deposits within the spinal canal. MRI is used to diagnose epidural lipomatosis and clearly depicts the deformation of the thecal sac by the excessive fat.

Conclusion: Epidural lipomatosis is rare and may present with symptoms of low back pain and radiculopathy.

Key words: Spinal epidural lipomatosis, magnetic resonance imaging, angiolipoma, meningovertebral ligaments

Introduction

We report a 60 year old female patient with lumbosacral epidural lipomatosis unrelated to corticosteroid therapy, a diagnosis of Cushing's syndrome or hypothyroidism.[1] Epidural lipomatosis has been reported in patients who have received transplants [2] or those having Paget's disease or macroprolactinoma.[3] There have been reports of epidural lipomatosis related to obesity.[1, 4-12] The first case of symptomatic epidural lipomatosis causing a compression of the thecal sac and subsequent neurological deficits was described in 1975 in a patient that had undergone a renal transplant. [13]

Epidural lipomatosis is reported to be a rare entity causing spinal cord or nerve root compression.[3] Eight cases of idiopathic lumbosacral epidural lipomatosis have been previously reported in the literature. [14] In a study of 525 patients with lumbosacral lipomatosis, the prevalence of lumbosacral epidural lipomatosis according to gender was found to be 2.7 times more common in men.[14] Exogenous steroid use accounts for 55.3% of the cases of spinal epidural lipomatosis while obesity was the second most

common cause at 24.5%. Idiopathic epidural lipomatosis was found in 17% of the cases. [11] Given the rare nature of epidural lipomatosis, the intent of this paper is to describe a recent case and provide an extensive review of the clinical presentation, discuss the pathophysiology of epidural lipomatosis and treatment options. Given this intent, radiological and clinical recognition of spinal epidural lipomatosis may assist in ensuring that patients receive appropriate and timely care.

Case Report

A 60 year old female presented with insidious low back pain of one month duration. The pain was reported by the patient to radiate down the right thigh and leg and most pronounced when the patient was supine. The patient reported that she experienced mild incontinence and urinary frequency due to her five pregnancies. Physical examination revealed a body mass index of 37.8 kg/m^2 placing the patient in the obese category. A neurological and lower extremity examination revealed no abnormalities. Palpation of the lumbar spine demonstrated hypertonic paraspinal musculature on the right and a prominent right posterior sacroiliac spine with decreased right sacroiliac motion posterior to anterior. Orthopedic examination demonstrated reproduction of the patient's chief complaints during Milgram's, straight leg raise, Gaenslen's and Nachlas' exams. The patient described painful right rotation and exhibited a decreased range of motion. Plain film imaging of the lumbar spine revealed multilevel discogenic spondylosis and apophyseal joint arthrosis and a 4% degenerative spondylolisthesis at L4/5.

Four months after the initial complaint of low back pain the patient presented to a urologist with a complaint of burning urination with hematuria and increased frequency.

Laboratory findings confirmed cystitis and urge incontinence was diagnosed. One month following treatment for cystitis, during an examination with a neurologist, the patient still reported being unable to empty the bladder and urinating more frequently. A urinalysis confirmed trace amounts of blood in the urine. Neurological examinations were negative, as well as a perineal sensation test. Palpation demonstrated pain at L5 with extension.

Prior to magnetic resonance imaging conducted five months after the initial onset of lumbar pain, the patient reported that the low back pain was increasingly worse since onset and the pain was newly described as burning. MRI examination revealed epidural lipomatosis at the L5/S1 level with deformation of the thecal sac (Figures 1, 2).

The patient underwent chiropractic treatment including lumbosacral Cox flexion-distraction technique. Flexion-distraction technique addresses the intervertebral disc, posterior facet elements and osseoligamentous canals with the intent of improving posture and movement while relieving pain. This is done by increasing the intervertebral disc height, decreasing intradiscal pressure and removing subluxation of the facet articulations. [15] Her treatments were twice weekly for seven weeks. The patient reported that her low back pain responded well and with consistent care she experienced periods without low back pain. The pain which was described as radiating down the patient's right thigh and leg did not respond to chiropractic care or to physical therapy.

Discussion

Obesity was the only apparent explanation for the patient's epidural lipomatosis. There was no indication of any additional underlying pathology and the patient denied any use of corticosteroids. Epidural lipomatosis as related to obesity is diagnosed when an individual with a body mass index of greater than 27.5 kg/m^2 [8] and no other

underlying pathologies or history of corticosteroid use has excessive epidural fat in the epidural space. In our case the patient exhibited a BMI of 37.8 kg/m^2. The placement of the body fat appears to be more important than simply obesity in general. There is a suggestion, in one study, of a relationship between epidural lipomatosis and body fat distribution that is centrally located within the abdomen. [6] This is disputed in another study as not having an effect on epidural fat.[4] Reduction in clinical symptoms of low back pain and radiculopathy has been reported when patients undergo a restricted caloric intake. [2, 3, 6, 8, 9]

Epidural lipomatosis is an overgrowth of unencapsulated epidural fat described in the thoracic and lumbar spine and has been reported as being infrequently associated with spinal cord or nerve root compression. [16] Clinical symptoms have been described as radiculopathy or neurogenic claudication when the epidural lipomatosis involves the lumbosacral spine. [7] Symptomatic epidural lipomatosis, as first described in 1975 [13] was reported to rarely result in thecal impingement and neurological deficits.[10] Literature reports epidural lipomatosis as being most frequently found in the thoracic spine. [2, 3, 9, 17] Cauda equina syndrome has been reported to be caused by epidural lipomatosis in the sacral region.

Histologically, there is no difference between the excessive fat associated with epidural lipomatosis and normal epidural fat that appears within the spinal canal. [11, 14, 18] On MRI T1 and T2 weighted images, epidural lipomatosis presents as hyperintense. The thickness of the epidural fat on axial slices is commonly greater than 7 mm. [8] The mean sagittal epidural fat thickness was reported to be 8 millimeters in six patients with spinal epidural lipomatosis as opposed to the average dimension on sagittal views of 4.6

mm for normal epidural fat.[11] The axial MRI image at the level of compression, L5/S1, measured 8.59 millimeters in this case report with a sagittal dimension of 7.75 millimeters. The measurement of the epidural fat, determined using the dimension of the anterior to posterior amount of epidural fat on sagittal MRI imaging in relationship to the anterior to posterior dimension of the spinal canal, has been quantified and graded as 0 when there is a normal level of fat within the spinal canal that occupies less than 40%. Grade I is defined as occupying between 41% and 50% (mild overgrowth), grade II as occupation of fat between 51% and 74% and grade III when 75% or more (severe overgrowth) of the spinal canal is occupied by epidural fat. [14] The current case demonstrates 67% of the spinal canal occupied by epidural fat, denoting a grade II (moderate overgrowth of epidural fat).

Eight different morphological variants (circular, sagittal ovoid, square stellate, palm leaf, coronal linear, cross, sagittal linear, a morphologic variation of the sagittal ovoid and a Y shape) in deformation of the dural sac has been reported, with the Y shape appearance being the most prevalent.[14] The deformation has been explained by the configuration of meningovertebral ligaments that anchor the outer surface of the dura mater to the osteofibrous walls of the lumbar canal. The ligaments are present in the anterior and posterior epidural spaces and are visible when epidural fat is excessive and causes an indentation of the dural sac .[14] The current case report demonstrated a morphological variation to the Y shape deformation of the dural sac.

The patient in this case report presented with symptoms of urinary frequency and incontinence. The literature reports one incidence of urinary disturbance associated with cauda equina syndrome caused by idiopathic sacral epidural lipomatosis. [19] The patient

did not present with obesity as does the patient in this current case report, yet there are similar reports of urinary disturbances. Urinary frequency, urgency and nocturia have also been reported as symptoms in a patient with spinal epidural lipomatosis secondary to hypothyroidism. [20]

The differential diagnosis for this case must include spinal extradural angiolipomas. Angiolipomas are rare and account for 0.14-1.2% of all spinal tumors and most often occur in the thoracic region of the spine.[21] These benign tumors may be either infiltrating or non-infiltrating and demonstrate a vascular component as opposed to the composition of solely fat in spinal epidural lipomatosis. Angiolipomas may cause spinal cord compression and may occur during pregnancy. [21]

Management for spinal epidural lipomatosis includes weight reduction and surgical decompression including laminectomy, interlaminar fenestration and lateral recess decompression. [3] In spinal epidural lipomatosis suspected to be caused by obesity, weight reduction reduced the back pain and radicular symptoms. [2, 3, 6] In several reported incidents weight reduction eliminated the patient's symptoms completely. [2] The patient in this case report received chiropractic care and physical therapy for low back pain and radicular symptoms. The patient reported resolution of the low back pain but continued to have the pain down her right leg.

Further research is needed to determine whether centrally located body fat plays more of a role in epidural lipomatosis than a more general distribution of body fat and to determine a clear correlation between epidural fat and obesity. [4] An important question needs to be answered in order to determine a clear correlation between obesity and spinal

epidural lipomatosis. Why don't more obese patients suffer from symptoms of spinal epidural lipomatosis? [3]

In conclusion, this report presented a patient with symptomatic lumbosacral epidural lipomatosis not associated with corticosteroid use or an underlying pathology, but instead related to excessive body mass index. The excessive epidural fat was identified using MRI. This diagnosis is important for clinicians to recognize in order to treat the patient appropriately with effective treatment procedures.

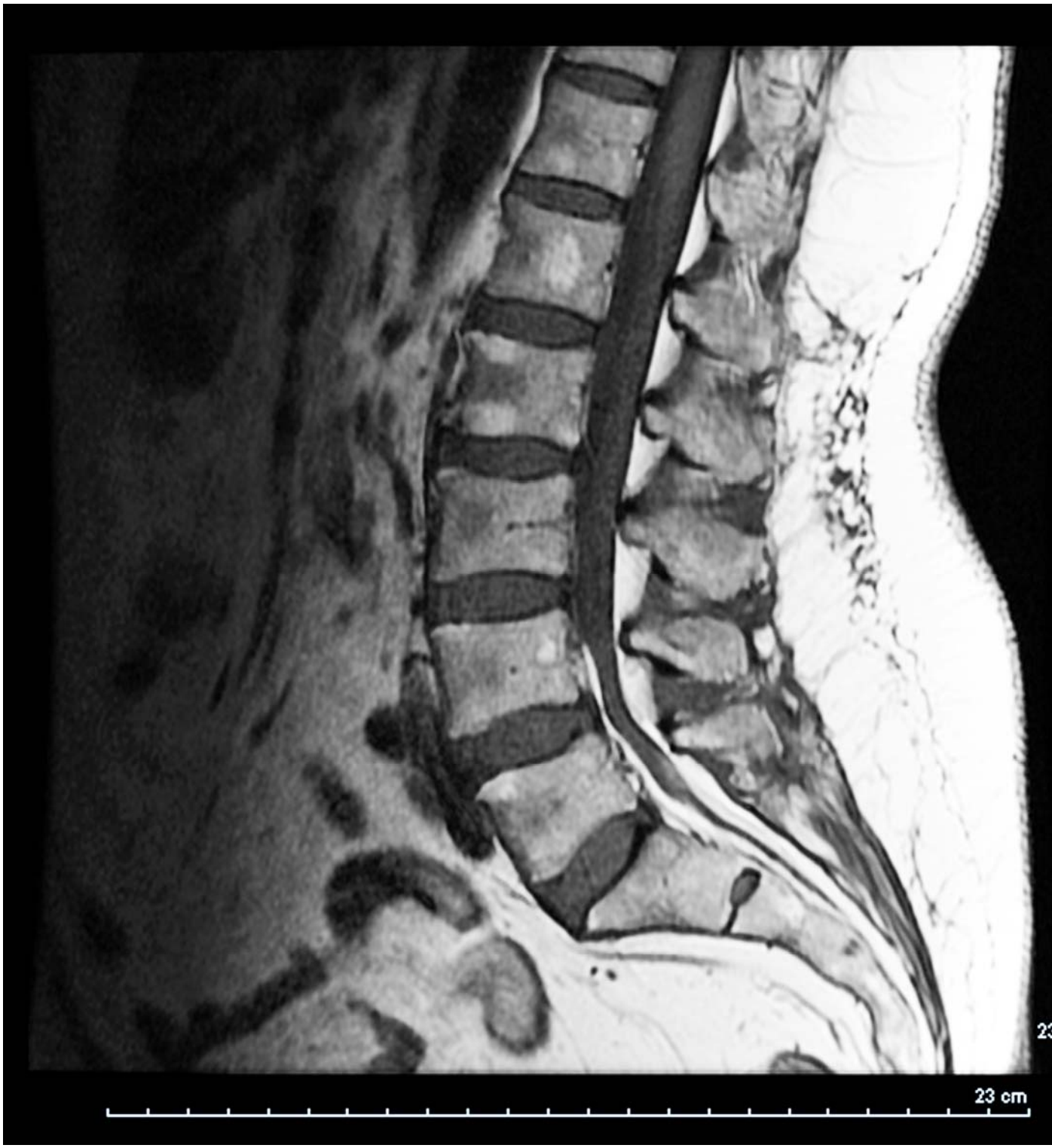

Figure 1
T1-weighted sagittal image of the lumbar spine demonstrating an epidural lipomatosis extending from L4 caudally to S1.

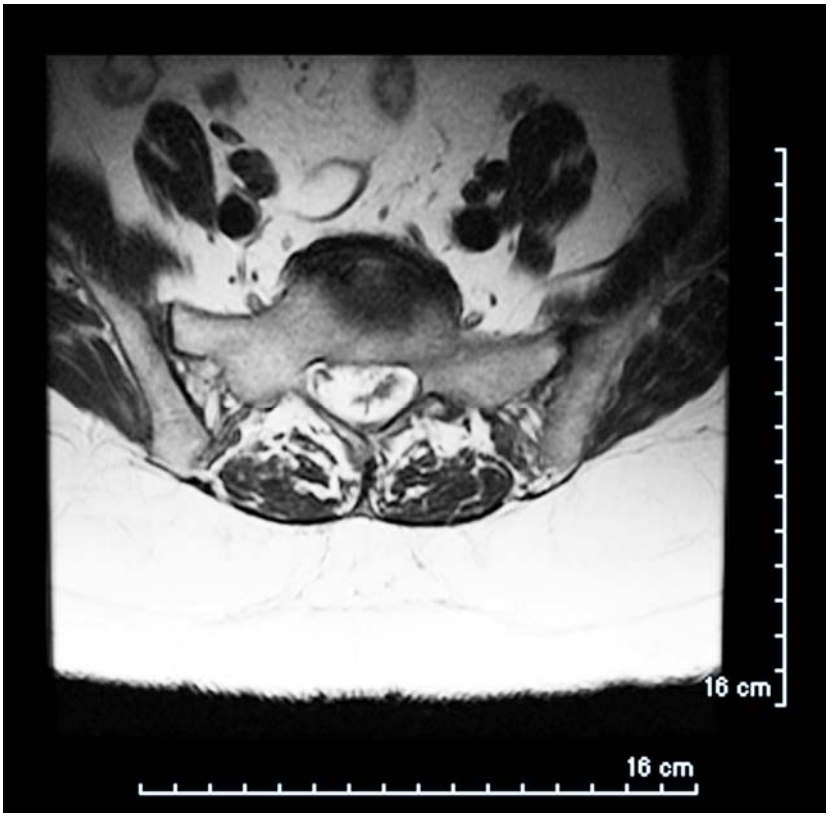

Figure 2
T1-weighted axial image of S1 demonstrating thecal sac deformation from compression by epidural lipomatosis.

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Review Corner:

Neuropathic pain

The objectives of this discussion is to describe the physiologic and clinical features of neuropathic pain and to understand the conditions that predispose patients to neuropathic pain. Additionally, we will consider various treatments for this type of pain, and how these treatments might differ from the treatment of acute pain problems.

Nociceptive v. Acute Pain

Nociceptive pain differs from neuropathic pain. Nociceptive pain results from direct activation of pain nerve fibers, either due to chemical, inflammatory or mechanical mediation. This is the more common type of pain that is well understood in acute injury. Neuropathic pain, on the other hand, consists of pain that is generated or sustained by the nervous system. This can either relate to changes in the peripheral or central nervous systems. For example, peripheral nerve fibers can be altered in terms of sensitivity or response due to many factors. In the central nervous system, there may be reorganization of the pathways that transmit the signal or the functions of systems that normally filter or suppress pain. Of course, central and peripheral factors may combine to contribute to the genesis of neuropathic pain syndromes. Regardless of whether peripheral or central processes predominate, neuropathic pain responds poorly to normal pain treatments, and in fact may be complicated by normal acute pain treatments for it.

Neuropathic pain is, by definition, chronic and may escalate with time. This is as opposed to most acute pain problems that lesson with time and particularly with the healing. Neuropathic pain may augment associated with some structural or physiological changes in pain pathways. Most things that cause pain are capable of triggering a neuropathic pain in susceptible individuals. There are some conditions that are more likely to do this than others, such as diabetic neuropathy, postherpetic neuralgia, phantom limb pain and trigeminal neuralgia. These are relatively well understood, while other conditions, such as mechanical and orthopedic problems, are less commonly appreciated triggers. Nonetheless, these represent significant contributions to neuropathic pain.

There are several different mechanisms for generation of neuropathic pain. These include several conditions that affect the peripheral nervous system and some that affect central nervous system function. Of the pathophysiologic processes that affect the peripheral nervous system, there are four main mechanisms to consider: 1. conditions that directly stimulate pain nerve fibers; 2. those that result in spontaneous firing of damaged nerve fibers; 3. processes that result in oversensitivity of afferent pathways due to denervation; 4. sympathetically maintained pain. Of the processes in the central nervous system, there may be sensitization at the synaptic level or through reorganization of higher processing mechanisms.

Direct stimulation of pain sensitive neurons

The smallest diameter, sensory nerve fibers, C-fibers, represent the nociceptive fibers that transmit intensity of pain. These can be damaged by mechanical injury, and this damage can be augmented by many factors including edema, adhesions or other internal factors. Various chemical mediators can also activate these small diameter afferent fibers. Many of these chemicals are mediators of inflammation.

Sensitization of peripheral nerve fibers

Oversensitivity and even spontaneous activity of damaged fibers can often occur in concert with directly injured nociceptive nerve fibers. At the site of injury there can be changes in neuronal membranes that include augmentation of numbers of numbers of ion channels and insertion of

additional types of receptors. These can result in sensitization of the nerve fiber. Several well-known clinical conditions, such as trigeminal neuralgia, radiculopathy, plexopathies and certain compression injuries in nerves, can result in this type of sensitization. These conditions often result in projected pain, with pain being felt very specifically along the distribution of the peripheral sensory nerve fibers. Initial treatment for this type of neuropathic pain should be directed at the mechanical and chemical factors at the site of damage, though some of these changes may be chronic and resistant to improvement. For example, there may be ectopic foci of firing along and damaged nerve fiber. This type of pain is often described as shooting or stabbing and, when many nerve fibers are firing asynchronously, the pain may be described as a continuous burning pain. This is a process that may produce pain in an anesthetic part of the body (anesthesia dolorosa) and can be extremely bothersome. In many of these conditions, larger fibers may be completely absent, resulting in overall decrease in sensitivity. Persistence of small nerve fibers (pain fibers) can result in magnification of pain due to spontaneous firing.

Deafferentation pain

Deafferentation results from the interruption of sensory conduction, particularly due to damage to large fibers. This process can increase sensitivity and irritability further along the sensory pathway. Some of this occurs as the direct result of loss of competition between the large-diameter “normal” sensory input and input from small diameter fibers. This can magnify the transmission and perception of pain in much the same way as a sound being heard most acutely in an otherwise silent room. Additionally, a chronic lack of normal sensory input has been shown to be capable of decreasing the number of inhibitory neurons in second and third order nuclei of the central nervous system. This, and other factors, can result in spontaneous firing of second and third order in neurons. Therefore, there may be pain in the area of diminished or even completely lost sensation. This results in an extremely distressing kind of "central pain".

Sympathetically maintained pain/Complex regional pain syndrome.

This group of conditions has gone by several names in the past (including complex regional pain, reflex sympathetic dystrophy, causalgia or sympathetically maintained pain), reflecting some variability in triggers and in presentation. These are all categorized by some irregularities in autonomic nervous system function, including changes in circulation and temperature as well as changes in sweating patterns. There may also be elements of neurogenic inflammation. Sympathetic nerve fibers not only secrete norepinephrine, but also certain inflammatory mediators such as prostaglandins and certain nerve growth factors. These may stimulate small diameter nociceptive fibers directly and may sensitize them. This is particularly true when these nociceptive fibers have been damaged.

The sympathetic nervous system does appear to be involved in more general inflammatory reactions. This occurs with release of inflammatory mediators along with sympathetic neurotransmitters. These factors interact with tissue elements, and have been shown to contribute to inflammation in various experimental conditions (such as experimental models of inflammatory arthritis). Therefore, inflammation must be considered a complex interaction between tissue components and the nervous system.

It is known that neurotransmitters released from sensory nerve fibers in the periphery can contribute to the inflammatory reaction as well. Many of these neurotransmitters sensitize other pain fibers and also can result in vasodilation, edema, infiltration of white blood cells and activation of other inflammatory cells. Therefore, the nervous system cannot be considered a passive participant in inflammation, but rather a central factor. This may be why conditions that activate sensory nerve fibers and the sympathetic nervous system can aggravate local inflammation. It also may be why certain types of nerve blocks may be effective in abolishing chronic pain. This is clearly been shown in certain animal models of chronic pain. It is interesting to note that the chronic application of capsaicin may diminish this component of the inflammatory reaction.

Therefore, the family of conditions ranging from true causalgia to various other types of sympathetically maintained pain may simply reflect differences in the specific tissues that neurogenic inflammation is acting upon. It is quite likely associated with many of the same central nervous system changes that were described previously in sensory nerve pathways.

Treatment of complex regional pain syndrome is quite difficult and sometimes frustrating. Early in the condition it's known that aggressive mobilization, and physiotherapy is helpful. Usually active exercises are better tolerated than passive, since there's often cutaneous hyperesthesia. Various methods of pain control from using physiotherapeutic modalities to anti-inflammatory medicines are probably best considered ways to enhance the patient's tolerance to movement-related therapies. The mechanism whereby mobilization helps the condition is not clearly understood, but may involve the kind of low-frequency activation of normal sensory fibers that are known to promote long-term depression of synaptic transmission in overactive pain pathways (see below).

Central factors in neuropathic pain

The central nervous system can participate in the generation of neuropathic pain. Central to this is the fact that neurons are plastic and change both structurally and functionally in response to injury. In many ways it can be stated that the nervous system can create a “memory” of pain. Many of the normal neural factors that are associated with memory in the cerebral cortex can take place in sensory nuclei as well. Convergence of high frequency, high-intensity stimulation appears to be necessary for this kind of memory and the most studied specific mechanism involves glutamate channels. High-intensity stimulation of these pathways results in "long-term potentiation." This may be enhanced by co-activation of various neurotransmitters associated with pain (such as substance P, neurokinin A, calcitonin gene-related peptide, and brain derived nerve growth factor). The resulting sensitized pain transmission neurons are activated much more readily and can even become spontaneously active within the pain pathway. Something that must be kept in mind is that these changes are quite robust and may even result in production of additional receptors and activation of various parts of the neuronal genome. Therefore, the effects of stimulation may be quite difficult to reverse.

Interestingly, low-frequency stimulation of the same pathways may result in long-term depression. Therefore, changes in the sensitivity of neurons are not necessarily immutable.

Although we've come to know many of the important receptors for this kind of pain memory at the neuronal level, it is becoming increasingly obvious that glial cells, may participate in this activation, producing cytokines and growth factors that are capable of enhancing and stabilizing some of these pathologic changes.

Changes in pain transmission pathways explain various clinical observations such as hyperalgesia. Neurons in sensory nuclei become excessively sensitive and may result in a painful experience to even light touch. Local reactions in the spinal cord are likely to be enhanced as well. This may explain local muscle spasm and overactivity of motor function. If the sensitization has been dramatic enough, it may result in spontaneous pain.

These changes have been demonstrated in various experimental models such as nerve root injury. There is also a strong suggestion that changes in the nervous system are occurring at many levels, including the spinal cord, areas of the brain stem that are involved in regulation of muscle tone and autonomic function as well as a levels of the thalamus and cerebral cortex that are involved in pain transmission and sensory and cognitive processing. I would hasten to add that this does not occur with all pain stimuli, and it is believed that some individuals are more likely to develop such chronic pain "Memory". It is also clear that certain stimuli are more likely to produce changes, as well.

The implications of this understanding of pain memory are several. These include the fact that it

is important to recognize the particular types of problems that are likely to become chronic and to treat them early and aggressively in order to attempt to interrupt conditioning at the beginning. Once problems become chronic, however, they are likely to require substantially more treatment, and treatment that takes into account this reorganization of sensory pathways.

Diagnosing neuropathic pain.

The first step in managing neuropathic pain is identifying its development. Usually this is by recognizing the proper clinical setting, and accompanying physical signs. This is much more likely to occur associated with conditions that damage the nervous system, including various direct nerve injuries and diseases such as diabetes, alcohol abuse, zoster, HIV, Lyme disease or conditions involving the central nervous system such as multiple sclerosis.

This kind of pain is much more likely to be described a shooting stabbing burning or searing, and it's often worse at night (a potential distinction from muscular pain). Inflammatory pain tends to be worse first thing in the morning, and during activity. This is also distinct from neuropathic pain. The reason that neuropathic pain is often worse at night may relate to the lack of normal input to the nervous system as well as circadian rhythms in pain thresholds.

The distribution of pain may help identify neuropathic pain. When the cause is peripheral, since symptoms often follow the peripheral nerve or nerve root distribution. When the cause is central, symptoms often involve large areas of the limb or body region. There may be changes in skin color, temperature or texture in the area. There may also be other evidence of damage to the nervous system (which may help to define a cause). Finally, there is likely to be a greater variety of pain related phenomena, including allodynia (pain from an innocuous stimulus) and there is also more likely to be autonomic changes.

Treatment

Ideally, when a specific trigger is identified, treatment of the underlying disease should be initiated. Symptom control through local or regional measures is somewhat preferable to systemic treatment due to potential side effects of systemic treatment. Topical anesthetics with or without ionto- or phonophoresis may be helpful, and gradually escalating doses of capsaicin cream may be helpful as well (if tolerated). Medically, regional anesthetic blocks, including sympathetic blocks, may be helpful. These interventions are more likely to be effective if accompanied by aggressive physical therapy interventions. Electrical stimulation, including transcutaneous electrical nerve stimulation, acupuncture-like stimulation, spinal stimulation or activation of local receptors by mobilization and massage may be quite useful. However, direct stimulation of painful areas may be poorly tolerated. In these cases, stimulation of adjacent areas, or even treatment of the opposite side of the body may be quite useful. These measures may be more useful in helping the person tolerate local therapy in the area of pain.

Unfortunately, nerve destructive procedures, which may be helpful on occasion, may worsen or even contribute to neuropathic pain. Various systemic treatments have been helpful, including certain anticonvulsants that calm abnormal nerve activity, as well as antidepressants that enhance serotonin and norepinephrine in the nervous system. In that regard, 5-hydroxytryptophan, which has similar effects on serotonin transmission, has not been specifically studied in neuropathic pain. There are various experimental treatments that have been attempted to interfere with some of the sensitization that occurs within the nervous system. However, these still remain on an experimental level.

Of interest, many of the acute pain medicines, especially those that act on opiate receptors, may actually result in further sensitization of neural pathways. It is not clear what individuals are most susceptible to this. That's an active area of research interest since it may help determine for whom these medications are appropriate in treatment of chronic pain.

Behavioral therapy is very important in chronic pain since stress amplifies pain and relaxation can reduce excitability of the autonomic nervous system. Additionally, sleep is quite abnormal in neuropathic pain patients, who have particularly disrupted slow-wave sleep. Various sleep interventions may be useful in patients including melatonin and 5-hydroxytryptophan.

The most direct access to sensory neurons is through activation of sensory fibers. It's clear that certain types of sensory stimulation may be capable of suppressing abnormal pain transmission. This is particularly likely to involve stimulation of large, proprioceptive nerve fibers and provides a rational basis for activation of these fibers in the treatment of patients with neuropathic pain.

In summary, neuropathic pain is an important challenge in the management of many chronic pain patients. The cause or trigger may not be evident and pain may outlast the usual duration of recovery. The clinician should be should have heightened awareness to this possibility when there are certain accompanying conditions such as diabetes, zoster, multiple sclerosis, Lyme disease, HIV or radicular involvements. Pain that is worse at night, pain that is described as shooting, stabbing, burning or searing, and pain along non-anatomic distributions may represent early clues to neuropathic pain. This is also true when hyperalgia is prominent, when autonomic signs are apparent or when there is pain in hypoesthetic areas. The recognition of these features should lead to consideration of neuropathic pain.

Standard therapies are often less effective in neuropathic pain patients. Disease modification, local or regional measures, systemic therapies and behavioral interventions should be strongly considered in this group of patients.

Rand Swensen, MD, DC, PhD

Neck Disability Index: State of the Art, 1991-2009

J Manipulative Physiol Ther 2008;31:491-502

Howard Vernon, DC, PhD evaluated the Neck Disability index vs the Oswestry Low Back Pain Index and Roland-Morris Low back Pain for the quantification of a patient's neck pain. He found that there were subtle differences the LBP indices v. the purely cervical spine index. Garnering information from the OI and extrapolating it to the NDI. It was found that the wording of certain pain expressions were not appropriate for the neck pain patient. An example of this would be the that Dr. Vernon found that "sex life" was not as appropriate for neck pain patients and substituted it for "recreation". He also re-worded the verbage, "use of tablets" to "intensity of pain or duration of sleep". This was further helped in clarification by adding the word "neck" with regard to the index. This allowed the reader to know that the index was relevant to neck pain, specifically.

The measurement of the OI is graded on a numerical scale: 0-4= none, 5-14= mild, 15-24= moderate, 25-34= severe and >35 is complicated

Ultimately, for now, the NDI is a good predictor of WAD injury. It is better at predicting symptoms/disability status than 'pain level' as reported by the patient. It is a good predictor of self-rated pain and disability ie., initial NDI is a good predictor of near full recovery and high initial NDI is a good predictor of chronicity. However, muscular

dysfunction and central sensitization must also be taken into account to completely rate the patient and their disability. The oldest of all outcome measures is and remains the NDI. It has a repeatability and reliability that makes it easy to use and efficient in predicting the future outcome of NDI.