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Superior Cluneal Nerve Schwannoma as a Cause of Intractable Ipsilateral Low Back Pain: A Case Report

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Background: Superior cluneal nerve (SCN) entrapment is an infrequent, yet commonly missed etiology of low back pain (LBP). The SCN is prone to entrapment as it travels through the iliocostalis muscle and exits thoracolumbar fascia via an osseofibrous tunnel as it courses over the iliac crest.

Case Report: A patient with chronic LBP exhibited new provocative sacroiliac joint testing not responsive to injections. Subsequent pelvis magnetic resonance imaging revealed a peripheral nerve sheath tumor of the right SCN. Although she exhibited an excellent response to therapeutic nerve blocks, her silicone allergy excluded her from peripheral nerve stimulation as a treatment option. She underwent SCN neurolysis and tumor resection by the neurosurgery department resulting in symptom resolution.

Conclusion: Interventional pain physicians should consider SCN entrapment when diagnosing intractable LBP. In the event of rare etiologies of SCN entrapment, such as neurogenic tumors, a multidisciplinary patient-centered care approach should be sought for symptom management.

Key words: Superior cluneal nerve, pain management, chronic pain, nerve block, schwannoma

BACKGROUND

Low back pain (LBP) is an extremely common problem and is a leading contributor to disease burden worldwide. The point prevalence of LBP has been estimated to be about 7.5% of the global population, or 577 million people in 2017 (1).

Superior cluneal nerve (SCN) compression or entrapment is an infrequent cause of LBP. LBP secondary to SCN etiology tends to be along the medial portion of the iliac crest in the gluteal or lumbosacral area and is worsened by lumbar movement such as extension, flexion, rotating, and prolonged standing or sitting (2-4). Although the medial branch of the SCN is the most common culprit, pain can originate from any branch. The etiology associated with the medial branch can be clinically misleading as sacroiliac (SI) joint-related pathology. Limited literature exists on the incidence of SCN entrapment, however few studies have cited anywhere from 1.6% to 14% of patients with LBP (5). No publications have reported nerve sheath tumors to be associated with the superior cluneal nerve as a causative factor for LBP. We present a rare case of superior cluneal nerve schwannoma presenting as intractable low back pain in the posterior superior iliac spine (PSIS) region that mimicked SI joint pain.

CASE PRESENTATION

A 57-year-old woman with a past medical history of fibromyalgia and trochanteric bursitis presented to the outpatient neurosurgery clinic with significant lumbar

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and bilateral lower extremity pain (more predominant on the right) that was severely exacerbated in the past 4 months. She reported no previous history of traumatic injury or lumbar spine surgery.

Prior to this initial consultation, she was followed by the Physical Medicine & Rehabilitation (PM&R) and Rheumatology departments. Initially, throughout her visits with PM&R, she primarily reported axial LBP at and above the level of the SI joint. Although she reported pain in her SI joints, the most debilitating pain was in the mid-low back. An x-ray of the lumbar spine revealed disc-related degenerative changes involving the entire lumbar spine and T12-L1 without spondylolisthesis, mild bilateral SI joint and hip joint degenerative changes, and no evidence of sacroiliitis. Magnetic resonance imaging (MRI) revealed multilevel degenerative disc disease throughout the lumbar spine without significant canal stenosis and mild to moderate neural foraminal narrowing at a few levels, including bilateral L3-L4 and L4-L5 levels. She had numerous unsuccessful medication trials with meloxicam, modafinil, amitriptyline, nortriptyline, gabapentin, and most recently with methocarbamol and duloxetine.

She subsequently underwent bilateral greater trochanteric bursae steroid injections with PM&R and reported a preprocedural pain score of 5/10 and a 3-week follow-up pain score of 5/10, but also subjective relief. That therapy was followed by 2 medial branch blocks to the lumbar spine and one subsequent radiofrequency ablation (RFA) to the left L3 medial branch, L4 medial branch, and L5 dorsal ramus. However, prior to the right-sided RFA, she reported worsening pain in the region of the PSIS. At this point, she had no therapeutic analgesic relief with any medications or adjunct therapies. A physical exam revealed positive FABER (flexion abduction external rotation) and Gaenslen tests bilaterally, which were new findings from her previous exam. Shortly after, she underwent bilateral intraarticular SI joint injections with minimal benefit.

Given the intractable pain was not amenable to multiple interventions, an MRI of the pelvis was performed which revealed no evidence of sacroiliitis, but incidentally, a T2 hyperintense one centimeter soft tissue nodule in the posterior right subcutaneous tissues associated with a small nerve, as shown in Fig. 1. A repeat MRI with and without contrast medium reidentified the lesion as a possible small tumor on the peripheral nerve sheath of a cluneal nerve branch due to the presence of a target sign.

After identification of the cluneal nerve lesion by PM&R, the patient was referred to the neurosurgery department for further evaluation. Neurosurgery recommended maximizing nonoperative management and referred the patient to interventional pain. An ultrasound-guided right superior cluneal nerve block was performed using 5 mL of 2% lidocaine and 40 mg triamcinolone, as shown in Fig. 2. The patient reported complete relief of her symptoms with this block along with improvement in spinal range of motion and ambulation.

A repeat injection was performed 2 weeks later with a similar improvement in symptoms. The benefit was transient for only a few days, which suggested inflammation was not the primary cause of the patient's pain. Peripheral nerve stimulator placement was considered but the patient's silicone allergy precluded her from this option.

Due to the transient pain relief from steroid injections and exclusion from nerve stimulator placement, neurosurgery was re-consulted to consider resecting the suspected peripheral nerve sheath tumor. The patient was amenable to resection and underwent exploration and neurolysis of the superior cluneal nerve with en bloc resection of the nerve sheath tumor. Final pathology revealed a schwannoma.

At the one-week postoperative follow-up, she reported resolution of her acute symptoms. At one year follow-up, she reported baseline chronic LBP but no further pain in the region of her PSIS. Per institutional guidelines, written Health Insurance Portability and Accountability Act (HIPAA) authorization was obtained from the patient, but institutional review board approval was not required for the generation of this case report.

DISCUSSION

The cluneal nerves are often missed as potential sources of LBP, even by experienced practitioners. There are 3 types of cluneal nerves: superior, middle, and inferior. The SCN may have a variable course and root origin. The SCN's primary function is to provide sensory innervation over the iliac crest and upper outer quadrant of the buttock. Most often the SCN originates from the dorsal rami (L1-L3) (5). At the point of origin these dorsal rami branches unite to form the SCN.

The SCN courses through a variety of back musculature (psoas major, Iliocostalis lumborum, longissimus, spinalis thoracis, paraspinal muscles, latissimus dorsi).

It runs posterior to the quadratus lumborum to pierce the thoracolumbar fascia as it crosses the iliac crest. As it crosses the iliac crest, there is an osseofibrous tunnel within the iliac crest which forms the posterior wall and the thoracolumbar fascia forms the roof. This could be a potential site for injury (5).

The SCN divides into 3 branches: the medial branch of the SCN, the intermediate branch of the SCN, and the lateral branch of the SCN. Cadaveric studies have detailed variable descriptions of the osseofibrous tunnel and a number of branches have been observed passing through the tunnel (6,7). SCN entrapment at the osseofibrous tunnel can result in LBP via referred pain to the corresponding dorsal rami (2,8). The nerve entrapment may lead to perineural edema and result in LBP from compressive neuropathy. Given the small size of the SCN, perineural edema that is often associated with symptomatic compression of peripheral nerves may be difficult to detect.

The proposed criteria for diagnosis of SCN entrapment involves the following items: unilateral or bilateral low back pain at or above the PSIS, worsening reproducible pain with lumbar spine movement, numbness and radiating pain in the SCN area (Tinel sign) when the trigger point is compressed, and symptom relief by SCN block at the trigger point (8-10). Due to the thin nature of the SCN, computed tomography and MRI tend not to be diagnostically informative; however, high-resolution computed tomography can be useful in identifying the thoracolumbar osseofibrous tunnel.

Treatment modalities for SCN entrapment consist of diagnostic and therapeutic peripheral nerve blocks, radiofrequency ablation, peripheral nerve stimulation, and surgical decompression. Cryotherapy is one of the newer modalities of treatment for LBP of cluneal nerve etiology. Block therapy consists of nerve blockage above the trigger point. The literature reveals mixed results regarding nerve block as a treatment for SCN entrapment. Many studies and case reports have reported successful results with Kuniya et al (2) reporting up to 68% of patients experiencing greater than 50% relief after one to 3 SCN nerve blocks alone; Maigne and Doursounian (5) reported less promising results with 28% of patients benefitting from one to 3 SCN nerve blocks. One common technique involves injecting local anesthetic over the iliac crest, 7 cm – 8 cm lateral of the midline (11). We believe an ultrasound-guided SCN nerve block should be considered in patients with intractable LBP as a diagnostic and treatment modality.

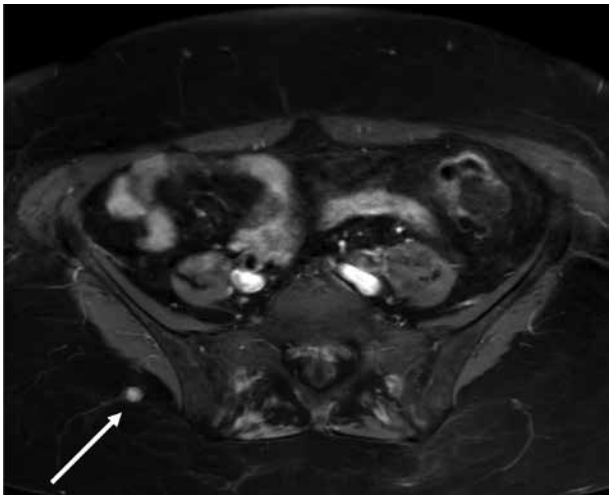

Fig. 1. Magnetic resonance imaging of the pelvis highlighting a rounded soft tissue lesion centered in the deep subcutaneous fat adjacent to the proximal right gluteus medius muscle.

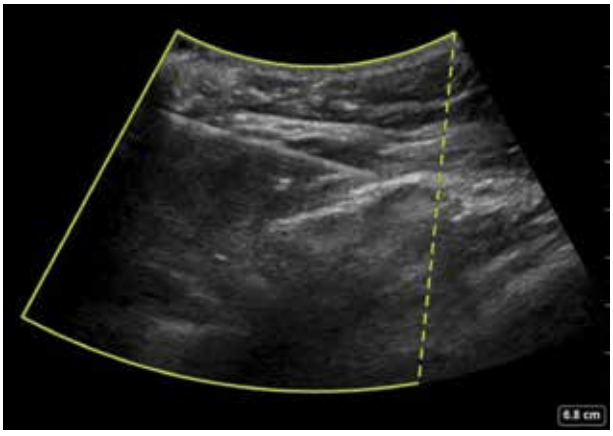

Fig. 2. Ultrasound-guided superior cluneal nerve (SCN) block. Left side of image is lateral; right side is medial. The SCN is located deep in the hyperechoic fascial plane.

Surgical treatment of SCN entrapment is a consideration for patients with pain not relieved by a nerve block or with transient relief from a nerve block. Surgical decompression of the SCN was described in 1997 by Maigne and Doursounian (5). The authors performed a 6 cm skin incision in 19 patients with SCN entrapment. The incisions were made under general anesthesia. In all cases, the site of entrapment was in line with the trigger point. Of the 19 patients who underwent surgical decompression, 13 had greater than 75% relief on the operated side; 7 of these 13 were discovered during surgery to have severe nerve compression (5). Less invasive surgery has been described by Morimoto et al

(12) in which 34 patients underwent a 5 cm incision at the thoracolumbar fascia under a surgical microscope. Incisions were made using local anesthesia only. All patients showed statistically significant improvement according to the Roland-Morris Disability Questionnaire when comparing preoperative scores to postoperative scores (mean postoperative follow-up period was 10 months) (12). Surgical decompression should be considered for patients with LBP secondary to SCN etiology who do not respond to conservative treatment such as SCN nerve blocks.

CONCLUSION

Patients presenting with pain focused over the PSIS, medial iliac crest, and in the lumbosacral region tend to be diagnosed as having a facet syndrome, an iliolumbar syndrome, SI joint pain and/or a lower lumbar disc problem (4). However, of all patients with a chief concern of LBP and/or leg symptoms, 10% have pain originating from SCN etiology (2). These data suggest SCN entrapment to be a significant causative factor for LBP and one that should be considered in the differential diagnosis for LBP. Moreover, LBP of SCN origin can significantly resemble SI joint pain, especially the medial branch of the SCN entrapment. In these cases, we encourage the use of ultrasound for SCN block therapy to rule in or rule out SCN as the cause of LBP. If the etiology is not amenable to interventional pain team intervention, a multidisciplinary patient-centered approach to care should be sought for surgical intervention. This report presents a novel case of exacerbation of preexisting LBP secondary to a SCN schwannoma with successful alleviation of pain after surgical resection.

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