Surgical treatment of superior cluneal nerve entrapment neuropathy

Technical note

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Object. Superior cluneal nerve (SCN) entrapment neuropathy is a poorly understood clinical entity that can produce low-back pain. The authors report a less-invasive surgical treatment for SCN entrapment neuropathy that can be performed with local anesthesia.

Methods. From November 2010 through November 2011, the authors performed surgery in 34 patients (age range 18–83 years; mean 64 years) with SCN entrapment neuropathy. The entrapment was unilateral in 13 patients and bilateral in 21. The mean postoperative follow-up period was 10 months (range 6–18 months). After the site was blocked with local anesthesia, the thoracolumbar fascia of the orifice was dissected with microscissors in a distal-to-rostral direction along the SCN to release the entrapped nerve. Results were evaluated according to Japanese Orthopaedic Association (JOA) and Roland-Morris Disability Questionnaire (RMDQ) scores.

Results. In all 34 patients, the SCN penetrated the orifice of the thoracolumbar fascia and could be released by dissection of the fascia. There were no intraoperative surgery-related complications. For all patients, surgery was effective; JOA and RMDQ scores indicated significant improvement (p < 0.05).

Conclusions. For patients with low-back pain, SCN entrapment neuropathy must be considered as a causative factor. Treatment by less-invasive surgery, with local anesthesia, yielded excellent clinical outcomes.

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Key Words • superior cluneal nerve • entrapment neuropathy • low-back pain • surgery • technique

Superior cluneal nerve neuropathy has been reported as a causative factor in low-back pain; it is a complication of harvesting bone from the posterior iliac crest for grafting (spinal fusion).1,5 Since Strong and Davila10 first reported cluneal nerve syndrome in 1957, SCN entrapment neuropathy has been documented as a cause of low-back pain; however, it tends to be misdiagnosed as a lumbar spine disorder and remains poorly understood clinically. Few reports on surgical procedures address this entity.1,2,6,7,9,11 We present the preliminary outcomes of a less-invasive microsurgical release procedure, performed with local anesthesia, to address SCN entrapment neuropathy.

Methods

Diagnostic Criteria

The proposed criteria for a diagnosis of SCN entrapment neuropathy are unilateral low-back pain involving the iliac crest and buttock, a trigger point over the posterior iliac crest 7 cm from the midline (corresponding to the nerve compression zone), and numbness and radiating pain in the SCN area when the trigger point is compressed. For this study, we included patients for whom low-back pain was bilateral. For diagnostic purposes, we blocked the SCN with a small amount of lidocaine. Symptom re-
relief of more than 75% has been obtained within 2 hours after inducing nerve block,6,7,12 and a 75% pain reduction confirmed diagnosis. Specifically, we injected 2 ml of 1% lidocaine at the trigger points in the buttock; this drug has no effect on pain from other causes, such as sacroiliac joint pain. We chose to inject the trigger point rather than the osseoaponeurotic orifice because this orifice is very difficult to find on the surface of the skin. Among the patients who experienced pain reduction after this test injection, some were judged to not need surgery and were excluded from this study; others, however, experienced pain recurrence after the analgesic effect of the test injection wore off and were considered surgical candidates.

On the basis of their symptoms, for some patients we attempted conservative treatment by performing several SCN blocks. The number of blocks depended on the patients’ condition and the effect of the block (average 6.1 blocks, range 3–10 blocks). Although the blocks relieved their pain by more than 75%, the pain recurred, so these patients were included in the study. We cannot rule out the possibility that some of the patients included in this study had coexisting lumbar spine disease. However, the site of entrapment is far enough away from the lumbar spine to render information on lumbar spine disease unnecessary. We based our decision to treat on the patients’ symptoms.

Before the study, all 34 patients gave written consent for participation. All had received unsuccessful conservative treatment, including peroral medication and SCN block. We excluded patients who obtained pain relief from conservative treatment, patients who refused surgical treatment, patients ineligible for surgery because of their general condition, and patients with dementia. None of the included patients had undergone iliac crest harvest for grafting, had suffered trauma to the affected area, or had reported rapid weight gain associated with the onset of SCN entrapment neuropathy.

We cannot comment on the utility of electromyography and sonography for the diagnosis of SCN entrapment because the nerve is thin and peripheral and difficult to identify on the skin surface. Additional studies to determine whether these modalities are useful for the diagnosis of SCN entrapment are being conducted.

Patients

From November 2010 through November 2011, we surgically treated SCN entrapment neuropathy in 34 patients: 13 men and 21 women, 18–83 years of age (mean age 64 years). The entrapment was unilateral in 13 patients and bilateral in 21. The duration of symptoms from onset to treatment averaged 62.8 months (range 1 month to 19 years). The mean postoperative follow-up period was 10 months (range 6–18 months). We carefully assessed our patients to ascertain that their low-back pain was attributable to SCN entrapment. For some patients, the pretreatment observation period was short and our decision to intervene surgically was based on their pain intolerance and/or their demand for treatment.

Surgical Technique

With the patient in the prone position and the site blocked with local anesthesia, we microsurgically released the SCN entrapment. We began by making a 5-cm skin incision across the trigger point located 7–8 cm from the midline on the iliac crest (Fig. 1). Anatomic studies have shown that the SCN crosses the iliac crest through the thoracolumbar fascia from a rigid osseoaponeurotic orifice located 7–8 cm from the midline.6,8 We carefully dissected the subcutaneous soft tissue and identified the SCN by placing a nerve stimulator on the fat layer of the subcutaneous space. The nerve stimulator system consisted of an electrical stimulator (Neuropack MEB2306, Nihon Kohden), bipolar forceps, and a connective wire. The stimulation rate was 1 Hz, duration was 0.2 msec, and intensity was 2.0–5.0 mA. Stimulation of the affected nerve triggers radiating pain in the SCN area, similar to that resulting from manual compression of the trigger point. The SCN slants from caudolateral to rostromedial and penetrates the thoracolumbar fascia through the orifice just before crossing over the iliac crest (Fig. 2A). After sharply cutting the thoracolumbar fascia of the orifice with microscissors in a distal-to-rostral direction along the SCN to release the entrapped SCN (Fig. 2B), we confirmed SCN decompression by seeing posterior bulging of the SCN. We then cut the thoracolumbar fascia until reaching a point where the SCN was free of kinks. We considered the procedure complete when the patient reported complete symptom relief and disappearance of the radiating pain when we manually compressed the SCN (trigger point) (Fig. 2C). The average surgical time was 45 minutes.
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Immediately after the operation, all patients were able to walk freely. The next day they were discharged with no restrictions or external fixation; they were able to resume their activities of daily living.

Evaluation of Outcomes

Clinical outcomes were assessed by using JOA and RMDQ scores before and at the latest follow-up visit after surgery. For statistical analyses, we performed the paired t-test by using StatMate III (ATMS Co. Ltd.) software. Differences of \( p < 0.05 \) were considered statistically significant.

Results

In all 34 patients, the SCN penetrated the thoracolumbar fascia through the orifice just before crossing over the iliac crest. Superior cluneal nerve entrapment by the fascia was severe, and the SCN was decompressed by dissection of the fascia from the orifice. There were no local or systemic complications during or after the operation. All patients reported symptom improvement during the surgery; none reported symptom worsening after surgery. According to JOA and RMDQ scores, all patients showed significant improvement at the last follow-up visit \( (p < 0.05) \). Before surgery and at the last follow-up visit, the average JOA scores were 13.9 ± 4.2 and 21.1 ± 5.4 and the average RMDQ scores were 14.1 ± 5.0 and 7.3 ± 6.7, respectively.

Illustrative Case

A 70-year-old man with a 3-year history of low-back pain had been conservatively treated elsewhere with medications. However, his pain gradually increased and interfered with his daily activities. He sought care at the Kushiro Rosai Hospital, Hokkaido, Japan, for low-back pain and difficulty standing up, sitting down, and sitting for prolonged periods. His visual analog scale score was 6 of 10 because of low-back pain. He had no obvious neurological deficits, and imaging studies (lumbar radiography, CT, and MRI) revealed no abnormalities. Palpation outside the bilateral posterior superior iliac crest, 8 cm from the midline over the iliac crest, produced severe tenderness. The patient also reported radiating pain in a caudolateral direction in response to compression of the trigger point on each buttock. An SCN block with 2 ml of 1% lidocaine injected at the trigger point in each buttock completely abated the pain, including the radiating pain. However, because the pain recurred 1 week later and subsequent treatments failed to relieve the pain, we performed surgery.

We first addressed the SCN entrapment neuropathy on the left side because that side was the most painful. The SCN penetrated the thoracolumbar fascia through the orifice just before crossing over the iliac crest \( (\text{Fig. 3A}) \). We opened the orifice with microscissors in a distal-to-rostral direction along the SCN \( (\text{Fig. 3B}) \) and released the entrapped nerve \( (\text{Fig. 3C}) \). Intraoperatively, the radi-
ating pain in response to manual direct compression of the SCN disappeared completely (Fig. 3D). The exposed SCN in the operative field was constricted at the site where it penetrated the orifice (Fig. 3E).

One week after releasing the entrapment on the left side, we released the entrapment on the right side. The patient’s low-back pain improved immediately after the operation and had not recurred 12 months later. At the final follow-up visit, the patient’s JOA score had improved from 17 of 29 to 28 of 29 and his RMDQ score had improved from 11 of 24 to 0 of 24.

**Discussion**

The SCN provides sensory innervation to the areas of the posterior iliac crest and upper buttocks. It originates from the upper 3 lumbar spinal nerves (L1–3), passes through the thoracolumbar fascia, and can be entrapped at the osteofibrous orifice where it penetrates the thoracolumbar fascia. The anatomic and functional bases for the development of SCN entrapment neuropathy are a rigid fascial edge and stretching of the gluteus maximus muscle and skin over a large area during flexion of the hip joint. If the nerve is chronically subjected to stretching, the resulting tissue irritation, edema, inflammatory cell infiltration, and scarring can lead to entrapment. Low-back pain caused by SCN entrapment is induced and exacerbated by movements such as rising, sitting, and rolling over, and by prolonged sitting, standing, or walking. According to Maigne and Doursounian, SCN entrapment neuropathy accounted for 1.6% of all cases of low-back pain without sciatica encountered at their institute. Kuniya et al. reported that 12% of all low-back pain was caused by SCN entrapment. We cannot comment on the actual percentage of low-back pain caused by SCN entrapment in this study.

For patients with SCN entrapment neuropathy whose pain is not relieved by conservative treatment with drugs or SCN block, surgical release of the entrapment might be effective. However, only a few reports describe surgical procedures for SCN entrapment neuropathy. Maigne and Doursounian obtained good outcomes for patients with this condition by performing SCN release at the thoracolumbar orifice, with patients under general anesthesia; however, they did not provide details on their surgical procedures. They looked for the SCN at the point where it exited through the osseofibrous orifice just before crossing over the posterior iliac crest. This point was located at the lateral border of the erector spinae muscles. Speed et al. also reported good results for a patient with SCN entrapment neuropathy who, under general anesthesia, underwent microsurgical SCN release. In some patients, it is difficult to identify the SCN because it is thin, and after penetrating through the orifice it is located subcutaneously in an area with much fat tissue. Surgical results tend to be unsatisfactory for patients with no visible nerve compression, and the entrapped nerve might not be addressed because it is obscured by branches and thus overlooked. In all 34 patients reported here, the SCN penetrated the orifice, and all patients reported immediate pain relief as soon as the orifice was opened. On the basis of our experience, we recommend identifying the SCN by first making an incision at the trigger point. Subsequent
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nerve stimulation under a microscope is very useful for identifying the SCN because it is a sensory nerve that can be easily monitored.

Using our less-invasive surgical technique and local anesthesia, we can visually confirm adequate SCN decompression and intraoperatively obtain patient reports of their sensations. We can also check for the disappearance of the radiating pain by directly compressing the SCN at the trigger point during the procedure. Sufficient decompression of the SCN is better determined by the patient’s intraoperative confirmation of the disappearance of the radiating pain than by the surgeon’s visual confirmation. Local rather than general anesthesia enables patients to return sooner to their activities of daily living.

We posit that the radiating pain is attributable to kinking of the SCN at the orifice and that this kinking can lead to nerve irritation caused by tissue irritation, edema, inflammatory cell infiltration, and scarring.

Although the etiology of SCN entrapment neuropathy remains unclear, the symptoms are low-back pain (buttock pain) and paresthesia in the area of SCN innervations. For a confirmed diagnosis of SCN entrapment neuropathy, the test SCN block results must be positive. Although most previously reported patients were affected unilaterally, two-thirds of the patients reported here were affected bilaterally.

Our study has some limitations. The number of patients was small and the postoperative follow-up period was relatively short (mean 10 months). Nonetheless, none of the patients experienced recurrence of pain after surgery. To assess the eventual rate of recurrence resulting from scar formation or adhesions, long-term follow-up studies are needed.

Conclusions

Superior cluneal nerve entrapment neuropathy must be considered as a clinical entity for patients with low-back pain. For patients who do not respond to conservative treatment, including peroral medications and SCN block, SCN entrapment neuropathy can be successfully addressed by less-invasive surgery performed with the use of local anesthesia.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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