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# Superior Cluneal Nerve Entrapment: An Overlooked Cause of Low Back Pain and the Role of Nerve Blocks

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**Background:** Low back pain is a leading cause of disability; superior cluneal nerve entrapment (SCN-E) is an underdiagnosed etiology. SCN-E mimics common pain generators such as lumbar radiculopathy, facet joint dysfunction, and sacroiliac joint pain, thus leading to diagnostic challenges and delayed treatment.

**Case Reports:** We describe 2 cases of SCN-E with distinct predisposing factors, including spinal scoliosis. A 74-year-old woman with a history of polymyalgia rheumatica presented with persistent low back pain and buttock pain despite standard treatments. A 77-year-old man with prior lumbar laminectomies reported chronic low back pain and right hip pain refractory to previous interventions. Both patients underwent ultrasound-guided superior cluneal nerve blocks, resulting in significant pain relief, thereby confirming SCN-E as the pain generator.

**Conclusion:** These cases highlight the importance of recognizing SCN-E in patients with refractory low back pain, particularly in those with altered spinal biomechanics. Ultrasound-guided SCN blocks serve as both a diagnostic tool and a therapeutic intervention, facilitating targeted pain management.

**Key words:** Superior cluneal nerve entrapment, superior cluneal nerve block, spinal scoliosis, altered spinal biomechanics, case series

## BACKGROUND

Low back pain (LBP) is a leading cause of disability worldwide, affecting an estimated 619 million people in 2020 (1). Superior cluneal nerve entrapment (SCN-E) is an underdiagnosed cause of LBP, often mimicking more common conditions like facet joint disorders, sacroiliac joint dysfunction, and lumbar radiculopathy (2). The incidence of LBP attributed to SCN-E is estimated to range from 1.6% to 14% (3).

The SCN consists of purely sensory fibers which originate from the posterior rami of T12-L5. These fibers pass through the psoas major and paraspinal muscles, run posterior to the quadratus lumborum, and continue through the inferior portion of the latissimus dorsi (2,4,5). The nerve then penetrates the thoracolumbar fascia and traverses the iliac crest through an osteofibrous tunnel to provide cutaneous innervation of the lumbar and superior gluteal regions. It is this fibrous tunnel formed by the fascia and the iliac crest where the SCN is prone to entrapment. There are various entrapment etiologies, including stretching of the erector spinae muscles, thoracolumbar fascia hypertrophy, or iatrogenic causes (i.e., injections and surgeries) (6).

Patients with SCN-E can experience LBP radiating to

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the upper gluteal regions of the buttock and posterior thigh. Symptoms can be exacerbated with lumbar rotation, extension, or flexion, as well as standing or walking. Additionally, given the nerve roots involved, SCN-E often presents similarly to sciatic nerve compression. Therefore, it is frequently overlooked due to its nonspecific symptoms and its absence from traditional diagnostic algorithms (5). A diagnostic SCN block is effective in both confirming an SCN-E diagnosis and providing therapeutic relief.

Our case series highlights 2 unique presentations of SCN-E in patients with thoracolumbar scoliosis: one with a history of polymyalgia rheumatica and the other with a history of multiple spine surgeries. These cases contribute to the limited body of literature exploring the interplay between postural abnormalities, surgical history, and SCN-E.

## CASE PRESENTATIONS

### Patient #1

A 74-year-old woman with a history of polymyalgia rheumatica, osteoporosis, and left knee arthroplasty presented to our pain clinic with right lower extremity pain and new-onset bilateral LBP and upper buttock pain. Physical therapy and a right trochanteric bursa injection resolved her leg pain; however, her buttock pain persisted. She described the pain as dull, achy, and mixed with sharp pain radiating down her right posterior thigh. A physical examination revealed mild thoracolumbar scoliosis and a loss of lumbar lordosis. She was also noted to have focal tenderness over the area medial to the right posterior superior iliac spine and iliac crest. An ultrasound-guided right SCN injection provided > 70% pain relief for 2 weeks, as measured by the Visual Analog Scale (VAS). Upon recurrence, conservative management with therapy, lifestyle modifications, and multimodal pain medications was recommended.

### Patient #2

A 77-year-old man with a history of multiple lumbar laminectomies presented to our pain clinic with chronic LBP and right hip pain. Despite previous treatments, including hip joint injections, transforaminal epidural steroid injections, and lumbar radiofrequency ablations, his pain persisted. He described sharp, burning pain in the right buttock, radiating to the hip, and worsened with movement. A physical examination revealed tenderness to palpation over the lumbar paraspinals and the medial aspect of the posterior iliac crest. He also had reduced lumbar lordosis, as well as moderate scoliosis. An ultrasound-guided right SCN injection was performed, which resulted in a VAS score reduction from 9/10 to 2/10 and 3–4 months of significant relief. He also reported regaining functional independence and re-engaging in regular activities and hobbies.

### Procedure

We identified the posterior inferior iliac spine through palpation and confirmed it using a linear ultrasound probe. With the patient prone, a linear transducer was placed in the transverse plane at the posterior inferior iliac spine and progressively shifted cephalad and laterally until a small fat pad, located between the posterior iliac crest, thoracolumbar fascia, and the lateral edge of the erector spinae muscle, was optimally visualized. At this point, the gluteus maximus muscle disappeared, and the gluteus medius became visible, as seen in Fig. 1.

A 22G, 3.5-inch spinal needle was then advanced into this space using an "in-plane" approach. The needle was inserted through the thoracolumbar fascia, close to the lateral edge of the iliocostalis muscle, where the posterior and anterior layers of the thoracolumbar fascia fuse. The transducer was redirected along the long axis of the osteofibrous tunnel in order to ensure in-plane needle insertion and to allow visualization of the entire tunnel along the thoracolumbar fascia. Once the needle tip was visualized, a 5 mL solution consisting of 1% lidocaine (2 mL), 0.25% bupivacaine (2 mL), and 40 mg methylprednisolone (1 mL) was slowly injected under continuous ultrasound guidance. During the injection, separation of the erector spinae muscle from the posterior layer of the thoracolumbar fascia was observed. After the injection was completed, the needle was safely removed.

## DISCUSSION

These 2 cases emphasize the importance of recognizing SCN-E as an overlooked, yet significant, contributor to chronic LBP, particularly in patients whose pain is refractory. SCN-E is often misdiagnosed due to its symptomatology overlapping with more commonly recognized pain generators, such as lumbar radiculopathy, facet joint dysfunction, and sacroiliac joint pain. As a result, many patients with SCN-E undergo prolonged, ineffective treatments, delaying appropriate management and relief.

Both of our patients had previously undergone unsuccessful conservative and pharmacological treatments, including physical therapy, pharmacologic intervention, and various minimally invasive musculoskeletal injections. Notably, both reported significant pain relief following SCN blocks, demonstrated by reductions in VAS pain scores and functional improvements. This further supports the role of SCN-E as a distinct pain generator and highlights the diagnostic and therapeutic utility of targeted nerve blocks. However, relief duration differed between the 2 cases: one experienced 2 weeks of relief, and the other achieved sustained improvement for 3 months.

Although the literature on pain relief duration post SCN nerve blocks is limited, some studies report significant relief lasting from 3 months to 4 years (7,8). The differences in duration and extent of pain relief in our patients could reflect their underlying comorbid conditions, which led to varying degrees of abnormal posturing and muscular asymmetry (2). For example, Patient One's history of left knee arthroplasty likely contributed to postoperative compensatory biomechanics, such as asymmetric loading and altered gait mechanics. Additionally, underlying polymyalgia rheumatica, characterized by systemic inflammation and stiffness, may have further exacerbated postural asymmetries and blunted the analgesic response from the nerve block.

The SCN's anatomical course makes it particularly vulnerable to entrapment at the osteofibrous tunnel along the iliac crest. This entrapment may arise due to postural instability, thoracolumbar fascia hypertrophy, overstretching of erector spinae muscles, or postoperative changes, such as scar tissue and fascial tightening. Our clinical cases highlight spinal scoliosis as a distinct and potentially underrecognized risk factor for SCN-E. The altered spinal biomechanics inherent to scoliosis may lead to increased tension within the posterior myofascial chain, thereby increasing susceptibility to SCN irritation through mechanisms such as friction, traction, or direct compression (9). Additionally, asymmetric loading patterns associated with scoliotic deformities may contribute to fascial plane tightening, which can not only worsen the degree of scoliotic curvature but also predispose to initial SCN-E onset and subsequent recurrence (10). Similarly, postoperative changes in patients who have undergone spinal surgery, including scar tissue formation and fascial contractures, may contribute to nerve entrapment. This is due to key thoracolumbar musculature involvement, such as the erector spinae and latissimus dorsi, which contribute to the osteofibrous tunnel (11). Postural abnormalities in these patients, such as hyper- or hypolordosis and pelvic obliquity, can exacerbate mechanical compression of the SCN through iliac crest distortion and thoracolumbar osteofibrous tunnel narrowing (11) .

Our case emphasizes the importance of considering SCN-E in patients with persistent LBP. Early recognition and use of diagnostic nerve blocks can both establish the diagnosis and provide symptom relief, while helping to avoid unnecessary interventions.

## CONCLUSION

These cases emphasize the importance of recognizing SCN-E as a potential cause of refractory LBP. Given its nonspecific symptoms and frequent misdiagnosis, SCN-E should be considered in patients with persistent LBP, particularly those with a history of altered biomechanics or prior spine surgeries. Diagnostic nerve blocks of the SCN serve a dual role in confirming the diagnosis and providing symptomatic relief. Increased awareness and timely intervention can improve patient outcomes and prevent unnecessary treatments targeting more commonly suspected sources of LBP.

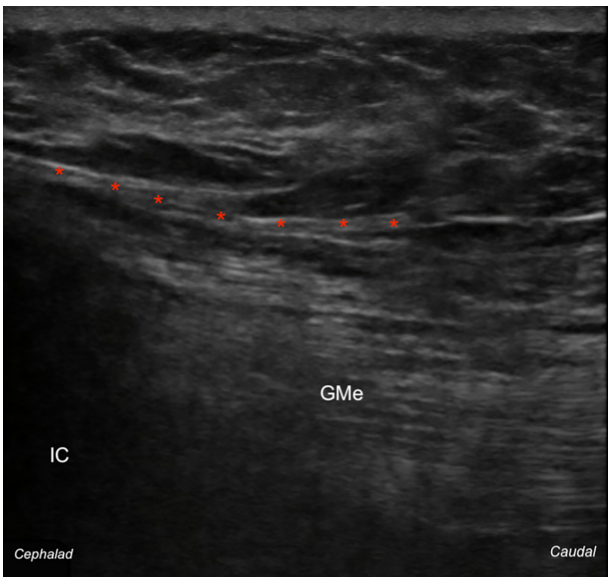


Fig. 1. Transverse image at the iliac crest under ultrasound imaging. The red stars indicate the intermediate and lateral branches of the superior cluneal nerve.
Note: IC = Iliac crest ; GMe = Gluteus medius

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