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Case Report

Easy to treat when the diagnosis is made: Three cases of clunealgia and the advantage of ultrasonography

Damla Yürük, Ömer Taylan Akkaya, Özgür Emre Polat, Hüseyin Alp Alptekin, Selin Köse Güven

Department of Algology, Health Sciences University, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey

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ABSTRACT

In this article, we present three cases of clunealgia admitted with low back pain. Their pain relieved with superior cluneal nerve block. The posterior side of the iliac crest, which is the location where the superior cluneal nerve passes, was identified using a high-frequency linear transducer. The drug injected separates the erector spinae muscle and thoracolumbar fascia and accumulates between these two structures. All patients were discharged with a complete pain relief. This report highlights the fact that superior cluneal nerve entrapment should be kept in mind in patients with low back pain and that ultrasound guidance can correctly identify the infiltration and eliminate anesthetization of other surrounding structures.

Keywords: Buttock pain, entrapment neuropathy, superior cluneal nerve, ultrasound.

In clinical practice, diagnosis of entrapment neuropathies (EN) is easy to recognize in the upper limb than the lower limb; therefore, it is believed that nerve compression in the lower limb is more frequent than thought.[1]

The cluneal nerves provide sensory innervation of the buttock skin. They are defined as the superior, medial, and inferior cluneal nerves according to the area they innervate on the buttock. The medial cluneal nerves arise from the posterior rami of the sacral spinal nerves and innervate the skin of the posteromedial area of the buttock. These nerves pass beneath the long posterior sacroiliac ligament and frequently entrapment can be seen.[2] The inferior cluneal nerves are gluteal branches that originate from the posterior femoral cutaneous nerves and provide the cutaneous sensation of the inferior part of the buttock and the perineum. Entrapment can be seen at the level of the sacrotuberous ligament, while passing below the ischium.[3]

The superior cluneal nerves (SCNs) arise from the lateral branches of the dorsal rami of spinal nerves Th12-L3 and consist of medial, intermediate and lateral branches.[4] These branches penetrate the thoracolumbar fascia and cross the iliac crest to supply the skin of the lower back, the upper buttock, and the proximal lateral thigh.[5] The SCN-EN can be seen at the level of the originating spinal nerves or osteofibrous orifice, where it passes through the thoracolumbar fascia and produces low back pain (LBP).[6] The incidence of SCN-EN is about 1.6 to 14%.[7] Treatment contains avoidance of forward flexure or acute bending of the low back, non-steroidal anti-inflammatory drug (NSAID) therapy, and local steroid injection. Nerve decompression is the last resort for these patients.[8]

In this article, we report three cases presenting with severe LBP radiating to the ipsilateral upper buttock. In two cases, SCN-EN was caused by failed back surgery syndrome or trauma, while one case had no

Corresponding author: Damla Yürük, MD. SBÜ Dışkapı Yıldırım Beyazıt Eğitim ve Araştırma Hastanesi Algoloji Bölümü, 06110 Altındağ, Ankara, Türkiye.
e-mail: damlayuruk@hotmail.com

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known cause. All were treated with ultrasound (US)-guided SCN block.

CASE REPORT

Three patients were admitted to our algology outpatient clinic due to pain in the lower back and upper buttock resistant to conventional pharmacotherapy and physiotherapy (Table 1).

Case 1- A 39-year-old woman underwent back surgery and developed pain in her lower back with radiation to the right leg two years later. A diagnosis of radicular pain was made. After L4-5 transforaminal epidural steroid injection (TFESI) was performed, her right leg pain decreased, but her low back and upper buttock pain on the right side remained.

Case 2- A 30-year-old woman had a five-month history of idiopathic upper buttock pain on the left side.

Case 3- A 37-year-old man presented with a history of falling to the ground while playing football. He developed pain immediately lasting for five months in the posterior iliac crest and upper buttock on the left side.

In all cases, pain severity was evaluated using the Visual Analog Scale (VAS). The scores of Cases 1, 2, and 3 were 8, 6, and 6, respectively before the procedure. All of the patients received combined drug therapy consisting of NSAIDs and opioids for pain relief. Cases 1 and 3 received additional physiotherapy; however, no pain relief was achieved.

On physical examination, the muscles of the thigh, hip, and hamstrings were normal, and there was no motor weakness in any patient. Trigger points were detected over the affected side of the posterior iliac crest (4 to 8-cm from the midline). Pain was exacerbated during extension movements of the lumbar spine. Sensory assessment revealed disturbance around the painful part of the low back, posterior iliac crest, and upper buttock. Pressure applied to the sacroiliac joints, and facet joints was painless, and the straight leg raise test was negative. Hip joint movements were normal and painless.

Other causes such as lumbar spinal canal stenosis, lumbar disc herniation, lumbar facet degeneration, scoliosis, and hip and sacroiliac joint disease were considered in the differential diagnosis. Lumbar magnetic resonance imaging (MRI) of Case 1 showed paramedian disc protrusion at L4-5 on the right side. After L4-5 TFESI was performed, her right leg pain decreased, while pain in the lumbar area remained. Some other pathologies other than lumbar nerve root compression which contributed to pain were considered. Lumbar and sacroiliac joint MRI of Case 2 revealed no pathological findings. Although lumbar MRI of Case 3 showed disc degeneration at L4-5 on the left side, it was considered incidental.

All cases were diagnosed with SCN-EN according to their medical history and neurological and radiological examination findings. A detailed information about the SCN block was provided to each case, and a written informed consent was obtained.

Patients were positioned in the prone position and the skin was cleaned under sterile conditions in the operating room. A high-frequency linear array probe of US (MyLab 30; Esaote SpA, Genoa, Italy) was utilized. The US transducer was positioned at the posterior edge of the iliac crest, which is the point where the SCN passes orientated in the transverse plane. The lateral border of the lumbar erector spinae muscle, which locates between the anterior and posterior layers of the thoracolumbar fascia and attaches to the posterior edge of the iliac crest medially, was defined. A 25-gauge, 90-mm Quincke point spinal needle was advanced utilizing an in-plane approach and, then, inserted and transmitted laterally. During injection, the spinal muscle of the spine and the back layer of the thoracolumbar fascia were separated, where the SCNs

TABLE 1
Characteristics of cases

Case	Age/Sex	Pain location	Baseline VAS score	Treatments	Etiology
1	39/F	Low back and upper buttock pain on the right	8	NSAIDs, tramadol, physiotherapy	Failed back surgery
2	30/F	Upper buttock pain on the left	6	NSAIDs	İdiopathic
3	37/M	Posterior iliac crest and upper buttock on the left	6	NSAIDs, physiotherapy	Trauma

VAS: Visual Analog Scale; NSAID: Non-steroidal anti-inflammatory drug.

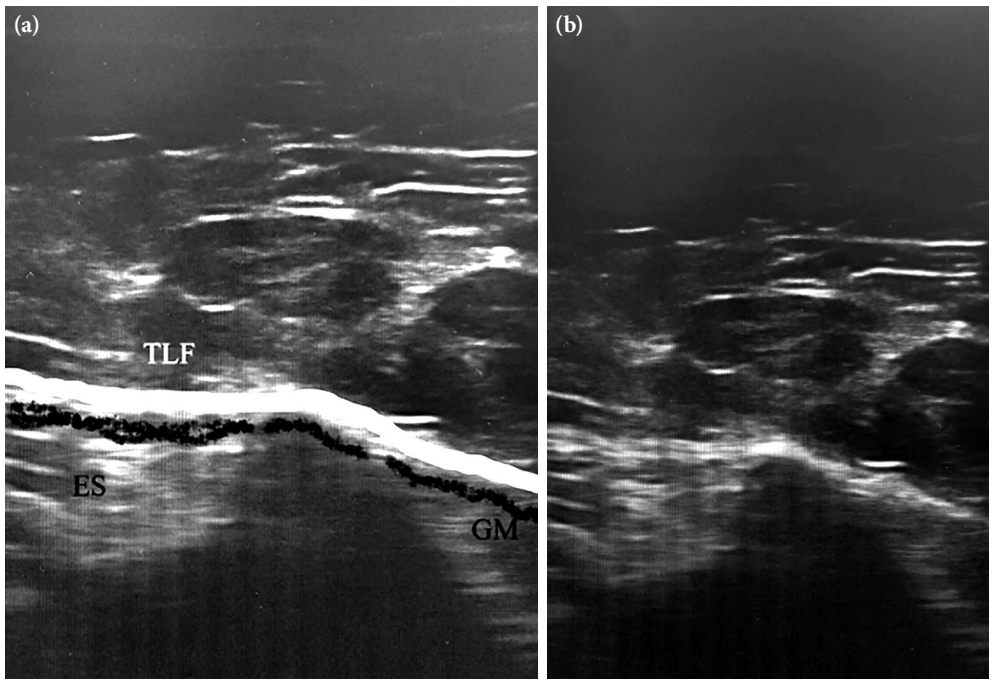

Figure 1. (a) Views of ultrasound-guided superior cluneal nerve block. The erector spinae muscle and posterior layer of thoracolumbar fascia must be separated, where the superior cluneal nerves pass. ES: erector spinae muscle. **(b)** Views of ultrasound-guided superior cluneal nerve block showing without labeling on anatomical structures.

GM: Gluteus medius muscle, white line; TLF: Thoracolumbar fascia, black line showing spread of local anesthetic and steroid combination.

pass. For diagnosis, 8 mL of 0.5% bupivacaine was injected, and the space between the muscle and fascia was seen to open. After one month, all cases were injected with 2 mL of dexamethasone and 8 mL of 0.5% bupivacaine using the same method (Figure 1a, b).

One hour after the SCN block with 8 mL of 0.5% bupivacaine, the VAS score was 0 in all three cases. All were discharged with recommendations to prevent the development of SCN-EN. One month later, the VAS scores of Cases 1, 2, and 3 were 5, 2, and 3, respectively. After one month, the SCN block with 2 mL of dexamethasone and 8 mL of 0.5% bupivacaine was applied to Cases 1 and 3. Case 2 was followed with observation only, as she refused a repeat injection. The VAS scores of Cases 1, 2, and 3 were 2, 2, and 1, respectively at two months and 2, 1, and 1, respectively at three months (Table 2).

DISCUSSION

Nerve compression lesions are usually caused by various types of injuries to the nerve such as traction, friction, and repetitive compression, leading to nerve ischemia. As a result, tissue edema, irritation, inflammatory cell infiltration, and scarring in the surrounding tissue which prevent regular sliding motion of the nerve can occur. Recently, facial structures close to the nerve have become prominent among the potential reasons of nerve compression.

TABLE 2
Visual Analog Scale scores of patients before and after superior cluneal nerve block

Case	Before	After 1 hour	After 1 month	After 2 month	After 3 month
1	8	0	5	2	2
2	6	0	2	2	1
3	6	0	3	1	1

Pain can be started by actuation of nociceptors inside of the connective tissue contacting peripheral nerves.[9] The possibility of SCN traumatic injury exists as in other superficial nerves.

Trescot et al.[10] reported that cluneal nerve damage developed spontaneously rather than iatrogenic causes. Current reports have also shown that the pathophysiological basis of SCN-EN is a rigid fascial edge and stretching of the dorsal muscles that attach to the dorsal edge of the posterior iliac crest, such as the thoracolumbar and latissimus dorsi muscles.[11] Stretching of the fascia due to activity increases symptoms. It can be seen in soldiers, athletes, and bodybuilders. Excessive trunk rotation may be the reason for SCN-EN.[12] Another report suggested that repetitive movements during bowling might be the cause of irritation of the SCN on the side contralateral to the bowling arm.[13] Iatrogenic lesions of the SCN can be observed in bone harvesting operations at the posterior iliac crest[14,15] and due to intragluteal injections.[16]

The diagnosis of SCN-EN can be only made clinically, since it is undetectable by radiological or electromyographical studies and, therefore, it is an underdiagnosed condition. Physical examination and medical history are helpful in the diagnosis. Posture causing compression in the nerve increases symptoms in EN.[17] Patients attempt to reduce pain and numbness in the SCN area which increases during lumbar extension by walking in the flexion posture.[18] Furthermore, due to SCN traction, the pressure applied to the trigger point produces pain.[19] The clinical features for SCN-EN judgment are as follows: unilateral pain and a trigger point on the iliac crest of the posterior ilium and upper buttocks region that matches to the nerve compression zone, numbness and a shock-like sensation in the SCN area, and symptom relief from injection of local anesthetic.[20]

In all three cases, significant symptomatic improvement elicited by SCN block confirmed that the pain was due to SC-EN. Loss of sensory function in the expected dermatome indicates the success of the SCN block. Complete pain relief 1 h after the SCN block depends on the membrane stabilizing and the analgesic effects of local anesthetic.[21] As the efficacy of the local anesthetic diminished over time, the VAS scores one month after the SCN block increased, compared to 1 h after.

Corticosteroids may be an effective therapy for pain by inhibiting proinflammatory cytokines, prostaglandin synthesis, neural firing, and input to central neurons. They may also decrease neurogenic extravasation and perineural edema formation by reducing substance P at the site of nerve entrapment.[22] In the light of these data, we performed SCN block utilizing a combination of a long-acting amide local anesthetic (bupivacaine) and methylprednisolone. This combination was preferred, as it was previously reported to provide an effective and long-lasting pain relief.[23] Due to the anti-inflammatory effects of corticosteroids, the VAS scores at two and three months after the SCN block were lower than the VAS scores before the SCN block.

The SCN block applied in SCN-EN reduces pain by 28 to 100%.[24] Many injection techniques for SCN have been described in the past several decades and, indeed, all are landmark-based (blind) techniques. These methods have a limitation in exactly locating a target nerve, as only bony structure is used as a marker for the SCN block. Currently, clinicians are able to reliably visualize nerves under the US guidance. Several scanning techniques have been proposed to recognize the SCN which mainly target the segments at the iliac crest level.[25,26] The SCN is visualized as a hyperechoic ovoid structure inside the hypoechoic subcutaneous layer.[27]

The utilization of US during the interventional procedures in the treatment of chronic pain provides many benefits to clinicians by placing the needle in the correct target and injection. It also improves the visualization of the nerve and surrounding structures, the spread of the medication, and the advancement of the needle to the target. Thus, accidental damage to the neurovascular structures near the target is prevented.[28]

Although there is no study comparing the clinical efficacy of SCN blocking landmark-based (blind) techniques and US guidance, cadaveric studies provide valuable data on this subject. The precision of US guidance was confirmed in a cadaveric study with Nielsen et al.[29] This study reported that US-guided nerve block reliably anesthetized the SCN with a success rate of 90%. The authors also reported that US-guided SCN block with 10 mL of methylene blue injectate had a successful spread rate of coloring and, to provide the injectate spread to the lateral edge of the erector spinae muscle, the needle tip should be placed laterally during the nerve block procedure. It should be noted that the amount of local anesthetic given is critical to avoid misdiagnosis of pain, since the presence of pain in close proximity to structures may complicate the diagnosis. In order to not spread the drug out of the target tissue, a volume

of 10 mL was used as the optimal dose. The SCN block provided both diagnosis and treatment, and no complications were observed. The misguided needle or the use of high volumes of medication may result in anesthetization of other sites, such as the sacroiliac joint. Real-time US-guided visualization of needle advancement and medication delivery around the cluneal nerve can effectively eliminate this.

Landmark-based (blind) techniques recommend a needle entry above the trigger point on the posterior iliac crest corresponding to the nerve compression zone, where the location of these nerves is in large variable. Lu et al.[30] reported that the SCN medial branch was localized 70 to 80 mm laterally top of the iliac crest in their cadaveric studies. Kuniya et al.[31] also reported the mean distance between the posterior superior iliac spine and the medial branch as 45 mm and explained this shorter distance by the smaller body size of Japanese cadavers. Loubser et al.[32] showed that sex differences in the posterior superior iliac spine to SCN length of distance were due to the sexual dimorphism of the bony pelvis. According to these studies, the targeted distance can be variable. Blind infiltration cannot ensure correct localization and, therefore, US guidance is superior in achieving the target anatomical structure for both diagnosis and treatment in SCN-EN. Considering the aforementioned anatomic variations, instead of blind injection made approximately 70 to 80 mm lateral to the midline on the iliac crest, we applied the SCN block with US guidance. The opening of the area where the nerve is located (between the muscle and fascia) after the injection can be only seen under the US guidance.

In conclusion, SCN-EN should be kept in mind in patients with LBP. Ultrasound-guided SCN block can identify the correct infiltration site and preclude the anesthetization of other surrounding structures, thereby, preventing complications and avoiding an inaccurate diagnosis.

Declaration of conflicting interests

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