CASE REPORT

Superior Cluneal Nerve Entrapment—Possible Pain Generator in Low Back Pain

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Abstract

High number of patients coming to pain clinic complain of low back pain (LBP) and this puts the pain physician in lot of diagnostic predicament. Consideration of common reasons from myofascial pain generator to rarer like cluneal nerve entrapment requires updating of knowledge regarding various rare pain generators in the back region. The cutaneous branches of cluneal nerve, namely, superior and middle are purely sensory. They provide sensation in the lumbar region and the buttocks, and their impingement around the iliac crest can cause LBP radiating to lower limb. Diagnosing uncommon cause for LBP owing to superior cluneal nerve impingement becomes a challenging task due to the absence of any diagnostic criteria apart from the high-index suspicion based on clinical findings and ruling out other common causes of LBP. It is usually misdiagnosed as a lumbar spine disorder. Here we present a case of chronic low backache with vague symptoms, which was successfully treated with the cluneal nerve block and the diagnosis of cluneal nerve entrapment was confirmed.

Keywords: Low backache, Superior cluneal nerve, Superior cluneal nerve entrapment neuropathy.

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Introduction

The lifetime prevalence of low back pain (LBP) in the general adult population is estimated to be 85–90%.[1] High prevalence rates of LBP have been reported among working-age population.[2] There are multiple causes for LBP such as ligament sprain, paravertebral muscle and fascia sprain, facet joint arthropathy, the vertebral periosteum, and spinal nerve root and spinal canal stenosis. The clinical features and causes of LBP remain poorly understood.[3] However, in the vast majority of cases no particular etiology can be identified and are labeled as nonspecific back pain and are usually attributed to muscle or ligament sprain.

One cause, presumed to be rare, is medial superior cluneal nerve entrapment (MSCNE), which is possibly underestimated and an infrequent cause of unilateral LBP. There is no diagnostic gold standard established for MSCNE. The literature contains limited information about MSCNE.[4] To date the diagnosis of MSCNE is clinical with high-index suspicion and by ruling out other etiology. Here we present a case of SCNE and draw attention to pain physician in SCNE.

Case Description

A 53-year-old male driver by profession, without a known case for any comorbidities, presented to our outpatient department with complaints of LBP and discomfort over left buttock, gradually developed over 2 years. Pain was also felt at the posterior thigh region up to the knee joint. Pain was characterized as continuous and dull aching, with a visual analog scale of 4–5/10. The pain intensity increased on walking or running and relieved on sitting down for short duration but increased on sitting for longer duration. The patient reported that pain got relieved when involved in prolonged activity. The patient took analgesics for temporary relief. Sometimes long hours of driving increase the pain. Pain was associated with tingling on the same side but without a history of numbness and weakness of lower limb. No history of morning stiffness was reported. No traumatic incident or injury was reported prior to the onset of symptoms. These problems had been very disruptive to his professional career and he had to remain absent from his duties.

On examination, a range of spinal movement was preserved. During flexion of hip joint, the patient complained of pain in the buttock region. The patient had normal gait and no obvious muscle atrophy, swelling, or scars were seen on inspection. A neurologic examination revealed no sensory or motor disturbance in his legs. There was mild tenderness over left posterior superior iliac spine on palpation, with no tenderness over midline. Sacroiliac (SI) joint tenderness was present. The flexion, abduction, and external rotation (FABER) test was positive on the left side. Compression distraction test was negative. The straight leg raise (SLR) test and Ely test were negative. Examination of the piriformis muscle revealed no significant findings. Patient routine investigation showed no abnormal findings. Imaging of spine and MRI lumbosacral region was grossly normal.

Considering the positive FABER test, local tenderness, and prolonged sitting increases pain, involvement of the SI joint was our probable diagnosis. Patient underwent pharmacological treatment and physiotherapy, but he did not respond to the anticipated level. Increase in pain on running and walking with no morning stiffness was going against SI joint pain. We decided to go ahead with diagnostic SI joint block along with L4–L5 facet medial branch

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block using sensorcaine and triamcinolone. After diagnostic block, patient symptoms persisted.

After the failure of diagnostic block, we again had a relook toward the pain generator. Local tenderness over iliac crest of around 7–8 cm from the midline, typical pain after muscle fatigue, and replicated posterolateral aspect of thigh pain made us think of cluneal nerve entrapment as a cause of LBP and buttock pain. Based on the literature search, the most likely involvement of the medial branch of superior cluneal nerve (MSCN) was suspected as it represented the area supplied over the iliac crest.

We conducted SCN diagnostic block with ultrasound (US) machine on high-resolution mode. Initial detection was performed at the point where the MSCN crosses over the posterior iliac crest in transverse view before the MSCN entered the thoracolumbar fascia. The US-guided fine-needle, in-plane technique of selective block was performed with 3–5 mL of 1% lidocaine along with 40 mg triamcinolone. Patient experienced numbness within the presumed MSCN territory and blockade; thus, this procedure was rated as successful. When patient had numbness, patient did not have pain, which confirmed the diagnosis of MSCN neuropathy. Six months' follow-up showed no recurrence of the symptoms.

Discussion

Factors concerned with the manifestation of LBP are ligaments sprain, paravertebral muscles and fascia sprain, facet joint arthropathy, the vertebral periosteum, and spinal nerve root and spinal canal stenosis. As many as 80–85% of patients with isolated LBP cannot be given the exact pathoanatomical diagnosis. It is difficult to base a diagnosis on radioimage findings alone. To understand its origin, clinical observations and an understanding of its specific attributes are necessary.[5]

Intermittent claudication, LBP related to spinal stenosis on imaging studies, and relief of symptoms with spine flexion are suggestive of lumbar spinal stenosis (LSS). Low back pain due to superior cluneal nerve entrapment neuropathy (SCNEN) is exacerbated by extension of the lumbar region, which improved with flexion posture, and both forward and backward bending are painful and restricted in range.[5–7] Low back pain tends to be misdiagnosed as lumbar spine disorder, and its clinical features and etiology remain poorly understood.[8] In our review of the literature, few studies were found on the connection between intermittent LBP and SCNEN.

Our patient suffered from LBP that was triggered by prolonged sitting and standing. The trigger point pain localized to the iliac crest with radiation on to the ipsilateral buttock and thigh; this is often a clinical finding for SCNEN.[9,10]

The SCN block resulted in dramatic pain reduction, and that we diagnosed SCNEN supported clinical criteria and confirmed with diagnostic block.

Chiba et al.,[3] in their study, found that patients were incapable of prolonged standing or walking, and their body posture affected their LBP, that is, it worsened upon backward bending (lumbar extension) and was mitigated by forward bending. They concluded that SCNEN induces intermittent LBP in the diagnosing of SCNEN and that etiology could involve SCN traction due to changes in the orifice around the SCN. Also, manual pressure on the trigger point could cause pain because of SCN traction (Fig. 1).

The SCN arises from the rami laterales of the posterior branch of the lower thoracic and lumbar spinal nerve (T12-L4). It invaginates the thoracolumbar fascia and occupies the upper buttock region over

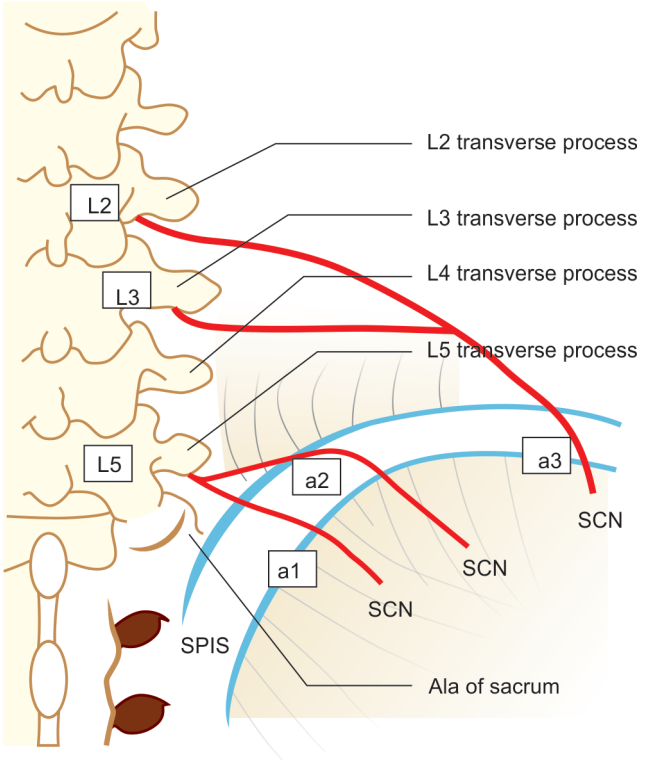

Fig. 1: Branches of the superior cluneal nerve

the iliac crest. Branches of SCN extend from a site of discontinuity of the fascia and innervates the buttocks cutaneously.[11–13] The SCN entrapment at the osteofibrous orifice, where it penetrates the thoracolumbar fascia, produces LBP.[8,12,13] Low back pain because of SCN entrapment is induced and exacerbated by movements such as rising, sitting, rolling over, crouching, lateral bending, and rotating, and by prolonged sitting, standing, or walking. However, its etiology remains poorly understood.[10,11,14,15]

Ermis et al.[4] stated that the SCNs supply the skin of the upper part of the buttocks. The tunnel where entrapment of the medial branch of the SCN usually occurs is found on the iliac crest approximately 7–7.5 cm away from the midline. Complaints of pain over the medial portion of the iliac crest and in the gluteal or lumbosacral area are often diagnosed as a facet syndrome, lower lumbar disc problem, or an iliolumbar syndrome (involvement of the iliolumbar ligament). The MSCNE have same clinical features but can be misdiagnosed.

Trescot[16] described that cluneal neuralgia was more commonly the result of a spontaneous entrapment of the nerve rather than of a nerve injury. This clinical entity may be underdiagnosed and should be considered as a potential cause for chronic LBP or leg pain.

Kuniya and Aota,[12,17] in their prospective study to analyze the prevalence of SCN disorder and to characterize its clinical manifestations, performed nerve block in suspected and selected patients. If meager decrease in pain or recurrence was observed, nerve blocks were repeated weekly up to three times. In certain patients who had intractable symptoms, surgery was performed, with almost complete relief of symptoms in few of these patients. They concluded that SCN disorder is not a rare clinical entity and may be thought about as a explanation for chronic LBP or leg pain.

As stated by Strong and Davila,[18] the patient is rarely aware of the presence of a trigger area in the back. The SCN blocks are not only useful for obtaining pain relief but form a diagnostic basis too.

Conclusion

We report a case of low backache with unusual and rare etiology often misdiagnosed and treated. Based on the literature, this is an underdiagnosed cause of LBP that has good outcome if recognized and treated.

The SCNEN was thought about as a determining factor in patients with LBP.

One ought to be aware of the spinal nerve origins of SCNs ranging from T12 to L5, though predominantly the L4, and the L5 lateral branches run through the osteofibrous tunnel in the fascia overlying the iliac crest. This evidence can explain how SCN entrapment disorder may lead to varied leg symptoms mimicking sciatica.

Pain physicians might confuse it with sacroiliitis, piriformis syndrome, etc. because of the similar clinical mimicries. So accurate diagnosis of SCN is priceless here, otherwise unwanted spine interventions may pose patients to lifelong sufferings.

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