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EDITORIAL

Superior Cluneal Nerve Disorder: How often do We Consider?

The superior cluneal nerve (SCN) is formed by the cutaneous branches of the dorsal rami of T11-L4 and supplies skin over the buttocks.[1] The first detailed description focusing SCN and low back pain (LBP) was made by Strong and Davila in 1957.[2] Though the chief complaint was limited to LBP in their series, patients also had referred pain in leg. Maigne and Sato described referring LBP preferably in and around iliac crest may develop following dermatomes of corresponding cutaneous dorsal rami; pressure on the iliac crest reproduces pain corresponding to the exit of the SCN.[1] According to researchers, medial branch of the SCN passing through an osteofibrous tunnel between thoracolumbar fascia and iliac crest might get entrapped and generated symptoms following its course.[1,3] Moreover, 39% of the SCN medial branches travel through the tunnel and only 5% of them exhibited macroscopically apparent entrapment.[4] Strong and Davila reported that the prevalence of SCN disorder was 9.8% of LBP sufferers.

Sometimes, SCN disorder patients are significantly older and have more vertebral fractures in the thoracolumbar or lumbar spine, eliciting pre-existing either asymptomatic or subclinical SCN entrapment over the iliac crest by irritation of the SCN at its origin from unstable facet joints due to increased kyphosis of the spine. Strong and Davila used diagnostic criteria for SCN disorder; a constant tender point no larger than 2 cm in diameter was situated in the low lumbar or episacral area, an injection with 2 ml of 1% procaine eliminated the trigger point and LBP preferably, relief should have been obtained by injection at least twice.[2] However, their criteria lacked a clear definition of pain relief and did not limit the tender point to the iliac crest where the medial branch of the SCN passed through an osteofibrous tunnel. According to Kuniya et al, the diagnosis of SCN disorder was made solely based on two clinical criteria: (1) maximal tenderness existing over the iliac crest, about 7 cm from midline and 4.5 cm from posterior superior iliac spine, and (2) the chief complaint being reproduced by palpation of the iliac crest.[1] It is likely that SCN disorder is not a rare clinical entity and is more commonly the result of spontaneous entrapment of the nerve than a nerve injury during bone harvest, as Trescot stated.[5] Trescot narrated that SCN entrapment may cause pain referral down the leg, producing 'pseudo-sciatica' features confusing with lumbar disk herniation or lumbar spinal canal stenosis conditions.[5] Similar result found in Strong and Davila's study, as enumerating 53.8% had referred leg pain. Superior cluneal nerve with leg symptoms was 50% in Kuniya et al and 42% out of them had unnecessary lumbar surgeries because of similar clinical mimicries of lumbar spinal stenosis. Sometimes, patients complain symptoms in a variety of areas from groin to sole. Since the SCN is thought to be composed of sensory branches of the dorsal rami of T11-L4 and supplies skin over the gluteal area, it is yet to know why the condition presents with sciatica features.[1] To resolve this fact, Konno et al performed an anatomical study using cadavers to identify the origin of the nerve passing through the osteofibrous tunnel between thoraco-lumbar fascia and iliac crest.[6] Of the ten specimens, the medial branch of the SCN that passed under the tunnel, getting traced medial to the L3, L4, and L5 foramina in one, five and four cases respectively. Besides, the most superior branch of the medial cluneal nerve (MCN) that constitutes of sensory branches of the dorsal rami of S1 to S3 often anastomoses with medial branches of the SCN proximally and distally.[7,8] So, L4 and L5 lateral branches pass through the tunnel and later anastomose with the S1 and S2 lateral branches explaining leg symptoms in association with SCN disorder mimicking sciatica.

It is not unlikely that the lumbar motion limitation and leg complaints could lead to misdiagnosis and unnecessary spine surgeries. However, characteristic painful limping and restricted lumbar motion differ from those seen in spine conditions. Tightening buttocks often aggravates pain while ambulation, suggesting that constriction of the gluteus muscles squeezes the SCN at the fascial orifice. Patients often realize that compressing above the iliac crest with hands reduced symptoms.[1] Symptoms often aggravates during flexion. Coupling rotation to contralateral side and flexion further aggravates symptoms. Sometimes, flexion and contralateral rotation strain the SCN. Besides, patients with SCN disorder often reports aggravated symptoms on lumbar spine extension. These characteristic signs are useful as a provocative examination maneuver to screen and differentiate SCN disorders from lumbar disorders.[1] Patients having true sciatica due to spinal canal or foraminal lesion generally have gluteal regions (Valleix's points) tenderness. Nevertheless, iliac crest tenderness can also be seen in sciatica, but it would not reproduce leg symptoms as like as SCN. So, how to approach it? Superior cluneal nerve blocks have some diagnostic and therapeutic value. In

spite of pain provocation while injecting, SCN blocks to be mandatory for accurate diagnosis and control of intractable symptoms; effective in curbing short-term symptoms. In some patients, surgery requires releasing SCN constriction under the thoracolumbar fascia tightly attached over the bony groove on the ilium and selecting these patients as surgical candidate would improve overall surgical outcome.

In fine, SCN disorder, is an example of extraspinal sciatica and sometimes pain physicians might get confused it with lumbar spinal stenosis, piriformis syndrome, etc. because of similar clinical mimicries. So, accurate diagnosis of SCN is priceless here, otherwise unwanted spine interventions may pose patients in life-long sufferings.

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