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Reviews

A Comprehensive Review of Cluneal Neuralgia as a Cause of Lower Back Pain

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Lower back pain (LBP) is one of the most common presenting complaints in clinical adult medical patients. While most often diagnosed as "nonspecific mechanical" in etiology, several lesser known, rarer causes of LBP exist, some of which can even cause neuropathic pain. One of these infrequent causes, cluneal neuralgia (CN), is associated most often with damage or entrapment of the cluneal nerves, particularly the superior cluneal nerve (SCN) and/or the middle cluneal nerve (MCN). These nerves supply sensation to the posterior lumbar and buttock area. However, the LBP caused by CN is often difficult to recognize because it can mimic radiculopathy or sacroiliac joint (SIJ) pain or lead to symptoms in the legs. This makes CN significantly important for clinicians and surgeons to include in their differential. A thorough history proves beneficial in the diagnostic workup, as many risk factors for CN have been reported in the literature. If a CN diagnosis is made, several effective conservative measures can alleviate patients' pain, such as nerve blocks, peripheral nerve stimulation, or high frequency thermal coagulation. Additionally, surgical treatments, such as CN release or endoscopic decompression, have resulted in fantastic patient outcomes. The purpose of the present investigation is to investigate the existing literature about CN as a cause for LBP, consider its epidemiology, discuss its pathophysiology and risk factors, elucidate its clinical presentation and diagnosis, and examine the various treatment modalities that have been reported across the world.

INTRODUCTION

Lower back pain (LBP) is a frequently encountered clinical complaint and is nonspecific in most cases.[1] A relatively rare but overlooked cause of LBP is cluneal neuralgia (CN), or neuropathic pain caused by damage to the cluneal nerves.[2] The cluneal nerves function as purely sensory nerves and the superior cluneal nerve (SCN) and middle cluneal nerve (MCN) both provide cutaneous innervation to the buttock and posterior parasacral region. Both nerves may cause acute and chronic LBP, in addition to leg symptoms, in the cases of superior cluneal nerve-entrapment (SCN-E) and middle cluneal nerve (MCN-E).[2] Generally, CN is thought of as a diagnosis of exclusion, but clinicians familiarizing themselves with and recognizing the presence of SCN-E and MCN-E will prove beneficial in diagnosing and treating CN. The present investigation summarizes treatment modalities of CN, which are comprised of various conservative and interventional pain measures and surgical management.

EPIDEMIOLOGY

The incidence and prevalence of CN need stronger evidence and further investigation. However, the literature review suggests that it is a relatively rare etiology of LBP. In pa-

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tients with LBP, the incidence of SCN-E is somewhere between 1.6%-14%.[2,3] Another prospective study reported that SCN disorders comprised 12% of all patients presenting with LBP and/or leg symptoms and approximately 50% of SCN disorder patients had leg pain and/or tingling. The average duration of these SCN symptoms was found in that study to be 27.3 months +/- 56.5 months (range 0.1-444 months).[4] Epidemiological data on MCN-E is lacking, but the incidence in LBP patients was found to be 13%.[5] A distinct population that had much more transient cluneal neuralgia was postpartum mothers. The incidence in over 13000 reported obstetric cases was found to be 0.73%.[6]

PATHOPHYSIOLOGY/RISK FACTORS

The pathophysiology of SCN-E and MCN-E involves mechanical irritation when the peripheral nerve is locally compressed. This trauma can result from traction, friction, or repetitive compression, which all result in edema around the nerve and interference with its normal sliding movement.[7] Though histopathologic evidence on CN is lacking, one pathological study on 2 patients s/p SCN-neurectomy revealed thickened perineurium, subperineurial edema, Renaut bodies, and nerve enlargement related to an increase in thinly myelinated fibers, which paralleled findings in other studies as well.[8–13] This sheds some light on the microanatomical considerations on cluneal neuralgia, but more literature exists on SCN-e and MCN-e on a macroscopic level. In the following paragraphs, we investigate the anatomy of the SCN and its described pathophysiologies, followed by that of the MCN, and conclude with a brief investigation of risk factors and possible associations relating to, and causing, cluneal neuralgia.

The SCN is a purely sensory peripheral nerve, derived from the cutaneous branches of the dorsal rami of T11-L4/L5 which passes through the psoas major and paraspinal muscles and travels posterior to the quadratus lumborum. It is relatively thin, with a mean diameter of 1.1 mm.[14–16] The SCN has been found to exhibit various branching patterns, and a variable number of branches among the population (more than 3 branches in one-third of subjects).[3,4,15–20] The SCN's medial branch in particular has been shown to penetrate the gluteal fascia and may become entrapped in the fascia attached to the iliac crest, termed the "osteofibrous tunnel," (OT) or a bony groove in the iliac crest itself.[3,4,15,16,20,21] Other investigations have corroborated that in a variable percentage of cases (16-95%) SCNs pass through the OT, many of which reveal entrapment.[16,17,22] A recent study proposed that the SCN's proximity to a rigid fascial edge over which it runs can subject the nerve to mechanical forces upon flexion of the hip joint and stretching of the gluteus maximus, leading to edema, irritation, inflammatory cell infiltration, scarring, and subsequent entrapment.[7] The superficial thoracolumbar fascia and gluteal fascia attaching to the iliac crest were incriminated as entrapping branches of the SCN in all 19 patients requiring SCN release in this recent study.[4] Although Maigne et al initially reported that the SCNs passing through the OT most commonly arose from L1 & L2 nerve roots (60% and 27%, respectively), another anatomical study on 37 dissections showed that SCN branches passing through the OT originated from L3-L5. Most of these SCNs at risk of entrapment originate from the lower lumbar nerve; this evidence may explain why SCN-E disorder can cause leg symptoms mimicking sciatica.[2,16]

The MCN provides sensation to the posteromedial area of the buttock and is most often composed of sensory branches of the dorsal rami of S1-S3, but its origin and pathways can vary.[23–25] In addition, it may anastomose with the SCN in the subcutaneous tissues of the buttock.[16] Overall, the MCN is less likely to be entrapped than the SCN because it travels superficially to the long posterior sacroiliac ligament (LPSL), and is shorter and thinner than the SCN (at an average of 0.8mm), and courses through paraspinal muscles attaching onto the dorsal sacrum.[23] However, being within or under the LPSL is a possible source of entrapment.[16,26–28] The proposed etiology of pain is either from mechanical stress in the ligament or nerve compression under the ligament.[16] Anatomically, the LPSL sustains tensile stresses during physiological loading of the SIJ, which helps describe the clinical picture of MCN-E (Dumas).[29] Similarly to SCN-E, MCN-E can also cause leg symptoms. However, the authors advised thorough workup before sending these patients to surgery, as half of their subjects with intractable LBP with MCN-E had concomitant sacroiliac joint pain (SIJ) or contributed by other diseases like SCN-E and radiculopathy.[5] Another important cause of MCN-E that needs more evidence is lumbar disc herniation. This case report suggests that surgery for this cause of LBP could be avoided with the treatment of the MCN-E.[30]

Recent scientific literature has shed light on many possible risk factors associated with cluneal nerve entrapment/injury. Importantly, a recent prospective study of LBP reported more women and older patients included in the SCN disorder group than in the non-SCN disorder group, which included those suffering from LBP. The average age in the SCN disorder group was 64.[4] It has been shown that as populations age, the incidence of LBP increases. Thus, it can be inferred that SCN-E and MCN-E prevalence (diagnoses of exclusion) is higher in the older population, although more studies are certainly warranted on this topic.[31] Also, vertebral fractures have been described as predisposing to SCN disorders. Kuniya et al reported that the prevalence of SCN disorders was significantly higher in patients with vertebral fractures (26/96, 27%) than in the remaining patient population (87/738, 12%) without vertebral fractures ($p<0.01$). The authors proposed a mechanism for this injury: entrapment over the iliac crest by irritation of the SCN at its origin from unstable facet joints and/or stretching of the SCN stemming from spinal kyphosis.[4] Similarly, another study also proposed that SCN-E should be considered after vertebral compression fracture patients presented with LBP (Kim et al 2015).

Though stronger studies are indicated, it seems in the current literature that past surgical history in the posterior sacral and lumbar spine area could predispose patients to LBP caused by SCN-E and MCN-E.[4,32,33] Delawi et al reported that the most common complication of autologous

bone graft harvest at the iliac crest is postoperative pain in the donor.[34] Similarly, lumbar disc surgery with fusion using the iliac crest resulted in persistent graft site pain and could cause injury to the SCN.[32,33] The L1-L3 nerves, and consequently the SCN, seem to be the most likely sensory nerves to be encountered during posterior iliac crest harvesting for spinal fusion.[35] However, a newer publication expressed that nerve injury following iliac crest harvest could be considered a risk factor for cluneal neuralgia, although, on the whole, nerve entrapment was a much more common cause.[36] Other surgeries may also lead to SCN injuries, such as spinal fusion procedures, sacroiliac screw placements, decubitus ulcer debridement, or muscle flap surgeries.[37–41] Any patients with lumbar or pelvic procedures are at risk of SCN-E.[42] Another anatomic study suggested that most SCNs were found to pierce the iliocostalis muscle, which could compress the SCN and lead to LBP, or conversely, a surgical approach to either the iliocostalis muscle or erector spinae mm could injure/compress the SCN, although a further clinical investigation is warranted.[21]

While more investigation needs to be done on the causes of cluneal neuralgia, two more studies regarding pathophysiology are worth mentioning. First, transient cluneal neuralgia can be observed in some postpartum mothers.[6] Physiological and anatomical changes during pregnancy, but also parturition itself or operative delivery can lead to this nerve injury. In the studied obstetric cases, postpartum cluneal nerve injury caused loss of sensation over the buttocks.[6] In another prospective observational study, the overall incidence of early postpartum neurological deficits was found as 2%, with about 13% of those presenting with cluneal nerve injury. Risk factors of injury in these mothers that were statistically significant included a past history of neurological conditions or history of a prior back injury. However, these postpartum deficits were all resolved in a few weeks' time.[43] Lastly, Ermis et al did a diagnostic prospective study on military personnel that showed the medial branch of the SCN as suffering entrapment in "muscle disorganization" and a taut, thickened peri-iliac band. However, limitations to the study's clinical application were acknowledged, including that those results were unclear as to whether the "muscle disorganization" they found was related to nerve pathology or if it gave rise to nerve pathology. Also, given the military personnel subject population, the authors acknowledged that truly generalizable results should be seen more often in athletes and other physically trained people, but this is not the case.[44]

The authors feel that other causes of CN should be investigated in future studies, including soft tissue pathology, such as obesity and Parkinson's disease. The latter particularly has been hypothesized to lead to abnormal muscle tone and posturing, which could theoretically compress the cluneal nerves.[2] However, the current literature indicates that SCN-E and MCN-E are far and away from the most common causes of LBP due to CN.

CLINICAL PRESENTATION/DIAGNOSIS

Cluneal neuralgia, due to superior and middle cluneal nerve (SCN and MCN) entrapment, most often presents in the form of low back pain (LBP). LBP is commonly defined as pain, tension, or stiffness found between the costal margin and inferior gluteal folds. With cluneal nerve entrapment, however, there may be tenderness at the rim of the iliac crest, decreased sensation of the buttocks below the iliac crest, and leg pain usually radiating to the ipsilateral leg.[45,46] The pain is often exacerbated by moving the lumbar region, movements such as rotating, bending, extending, prolonged sitting, or walking.[47] Leg symptoms are seen in 47-84% of patients, a symptom that mimics lumbar radiculopathy.[47–49] In a study by Strong and Davila in 1957, twenty-one out of the thirty patients complained of leg pain in various areas.[49]

Although the features of SCN and MCN entrapment remain to be fully elucidated, the correct identification of symptoms is required in order to prevent misdiagnoses. Cluneal nerve entrapment needs to be suspected when the following criteria are met- LBP is present with iliac crest and buttocks involvement. The pain is exacerbated upon movement; a trigger point, corresponding to the pressure zone of the nerve, is present over the posterior iliac crest. Upon palpitation of the trigger point, there is tenderness, numbness, or radiating pain, which signifies a positive Tinel sign. Finally, there should be relief of symptoms upon nerve block.[46,47] The SCN and MCN are very thin, so imaging studies such as computed tomography (CT) and magnetic resonance imaging (MRI) studies are not diagnostically helpful.[47]

Multiple studies help to illustrate the criteria that are first met before diagnosing SCN and MCN entrapment. In a 2013 case report, a 48-year-old woman presented with LBP and radiating buttock pain; mid-posterior thigh pain was provoked by palpitation of the MCN trigger points.[46] This pain was relieved by infiltration of a local anesthetic, Lidocaine. In a similar prospective study, the diagnostic criteria consisted of 1) a tender point on the posterior iliac crest where the medical branch of SCN runs and 2) palpitation triggered the complaint of LBP and/or leg symptoms.[49] The LBP and leg symptoms were assessed using a visual analog scale (VAS) score. The VAS score was recorded before the injection (68.6 ± 19.2 mm), fifteen minutes (31.6 ± 27.0 mm), and again one week after (45.2 ± 28.8 mm). Although there was a marked decrease in the VAS score both fifteen minutes and one week after, the biggest decrease was seen fifteen minutes after the injection; however, both demonstrated a significant decrease compared to the mean VAS score before injection ($p < 0.05$).[49] Another significant finding was more women and older subjects ($p < 0.05$) were in the suspected SCN disorder group compared to the non-SCN disorder group. Although nerve block injections had to be repeated up to three times, symptom relief was achieved in 85% of SCN disorder patients. Nerve block proved to be an effective treatment modality in this patient population.[49] One study sought to investigate MCN entrapment and ascertain the relationship between the MCN

and long posterior sacroiliac ligament (LPSL), using cadavers. The anatomical study identified 64 MCN branches in thirty hemipelves, ten of which penetrated the LPSL. Four of those ten branches had evident constriction under the ligament. Being that this was the first anatomical study performed to elucidate MCN entrapment in cadavers, it is highly likely that this clinical phenomenon is not rare and may be underdiagnosed.[50] A separate anatomical study considered the relationship between the SCN to the posterior iliac crest and thoracolumbar fascia in fifteen cadavers. While the intermediate and lateral branches of the SCN pierced or passed through a fissure in the fascia, the medial branches of the SCN appeared to be trapped between the taut fibers of the thoracolumbar fascia and superior rim of the iliac crest.[51,52] Anatomical knowledge of the cluneal nerves may help to highlight future decompressive procedures when entrapment is suspected.

DIFFERENTIAL DIAGNOSIS

Multiple pathologies must be considered when clinically presented with a patient suffering from low back pain (LBP) and/or leg involvement. There are mechanical causes (97%), nonmechanical spinal conditions (1%), and visceral diseases (2%) which lead to LBP. Some mechanical causes of low back pain include internal disk disruption, facet joint pain, sacroiliac joint pain, sacroiliac ligament pain, torsion injuries, vertebral insufficiency fractures, scoliosis, interspinous tissue injury, lamina impaction, and spondylolysis in a sportsperson.[53–59] A retrospective study found that the younger the patient, the higher the likelihood that the LBP was discogenic in nature, whereas in older patients, sacroiliac joint pain or an acetogenic cause was more likely.[60] Statistically, 40% of adults suffer from disc-related pain, 30% suffer from facet joint pain, and 20% suffer from sacroiliac joint-related pain.[61] It is clinically important to note that MCN entrapment mimics sacroiliac joint pain: sacroiliac joint pain is also associated with LBP and buttock pain; however, the SIJ score helps to differentiate between SIJ-related pain and pain caused by other factors such as lumbar spinal canal stenosis and lumbar disc herniation. If the SIJ block is ineffective, MCN entrapment should be considered.[62] A retrospective analysis investigated LBP due to SCN entrapment versus lumbar spinal canal stenosis using the Roland Morris Disability Questionnaire (RMDQ).[63] The study found that SNC entrapment heavily affects physical and psychological function; RMDQ scores were significantly higher, and an increased level of disability was seen in the patients with SCN entrapment compared to patients with lumbar spinal canal stenosis.[63] On the other hand, nonmechanical spine conditions such as neoplasia, spondylarthritis, Scheuermann disease (especially Type II), Paget disease, and diffuse idiopathic skeletal hyperostosis, can also cause LBP. LBP can also signify other visceral diseases: prostatitis, endometriosis, pancreatitis, cholecystitis, ulcer, chronic pelvic inflammatory disease, nephrolithiasis, pyelonephritis, and pyelonephritic abscess, to name a few.

In a clinical review on the diagnosis and treatment of LBP, 90% of patients with LBP have "non-specific low back pain," a diagnosis solely based on the exclusion of other specific pathologies.[64] Some "red flags" which seem to indicate an underlying pathology include an onset of <20 or >55 years of age, non-mechanical pain, thoracic pain, weight loss, feeling unwell, widespread neurological symptoms, a structural spinal deformity, or previous history of carcinoma, steroids, or HIV. The following indicators are more suggestive of a nerve root problem: unilateral leg pain that is greater than the low back pain, pain that radiates to the foot or toes, numbness and paresthesia, localized neurology, and when a straight leg test incites more leg pain.[64] Diagnostic imaging studies should be considered when patients exhibit little to no improvement after 6 weeks of medical management and/or physical therapy. Imaging should also be considered for patients with a suspected underlying condition or patients with severe or progressive neurologic deficits.[65]

TREATMENT

Cluneal Neuralgia has a wide array of treatment options for those suffering from the condition. The treatments range from a simple nerve block all the way to peripheral nerve stimulation and surgical procedures. After an individual has been diagnosed with CN, the options for treatment are divided into broad categories: conservative management, peripheral nerve stimulation, or surgical intervention.

CONSERVATIVE TREATMENT

If an individual chooses conservative management, one of the first and simplest treatments is a nerve block. In a nerve block, a local anesthetic with or without a steroid is injected directly into the area causing the pain. This will desensitize the nerve and the steroid can provide an anti-inflammatory effect, leading to a temporary resolution of pain. This procedure can be diagnostic and therapeutic. It can be used as a diagnostic tool in that if it causes a resolution of pain then the individual is likely to have a form of CN. However, if the procedure does not help resolve the pain, then it is unlikely that the individual has a diagnosis of CN. It is therapeutic in that if the individual has CN then the procedure is likely to help resolve the pain, producing a therapeutic effect.[66] Related to the entrapped middle cluneal nerve usually being found in the Long Posterior Sacroiliac Ligament (LPSL), researchers have found that ultrasound-guided nerve block into the LPSL is able to reliably anesthetize the superior cluneal nerve with a success rate of 90%.[67,68] This treatment is also beneficial for individuals who are planning to undergo surgery in that it anesthetizes the skin, which can control postoperative pain at the incision site.[67] A downfall of local anesthetic alone is that it is temporary and will wear off over time, causing individuals to have to receive more injections if they continue to choose conservative management.

Another conservative management option is high-frequency thermal coagulation. This treatment uses a needle,

placed in the LPSL, that transmits high-frequency radio waves and heat, which are used to degenerate the cluneal nerve, rendering it unable to transmit pain signals. The needle is heated to a temperature of 90°C and is left in place for 90 seconds.[68] This process is done in three different areas along the LPSL. In a recent study, all individuals who received high-frequency thermal coagulation reported pain alleviation with no reports of complications.[68] In addition, thermal coagulation extended the duration of pain relief to 145.7 ± 38.7 days compared to 7.7 ± 6.6 days of pain relief from a simple nerve block.[68] Although the duration of pain relief is significantly extended with high-frequency thermal coagulation, the pain will still eventually return and will require further treatment. Related to this, high-frequency thermal coagulation is an attractive option for individuals wanting to avoid the operating room. On the other hand, it may not be the best long-term option for those wanting to permanently eliminate the pain.

Another conservative management option for cluneal neuralgia is the use of a Capsaicin patch. Capsaicin is a highly selective TRPV1 vanilloid receptor ligand. The TRPV1 vanilloid receptor is largely involved in the transmission of pain signals.[69] By prolonged exposure of these receptors to Capsaicin, they essentially become desensitized to the pain signals. In addition to desensitizing the fibers to pain, the presence of the Capsaicin patch also decreases the density of these fibers in the skin, as well as decreasing the expression of the TRPV1 receptor.[69] Researchers have been able to use Capsaicin patches to significantly reduce pain in 24% of patients making Capsaicin patches a potential first-line treatment for individuals with chronic CN.

Another conservative management option for CN is wireless peripheral nerve stimulation. In this treatment, after a diagnosis of CN is made, a Tuohy needle is advanced into the area of the nerve using ultrasound guidance. Leads are inserted through the needle and the needle is removed, and a receiver is then inserted. This treatment allows for wireless nerve stimulation of the CN, eliminating the need for surgery.[70] Researchers were able to use peripheral nerve stimulation to provide satisfactory pain control to all individuals in the study.[70] Using this technique, researchers were able to lower the pain score from 6.4 before treatment, to 1 after treatment.[70] Although peripheral nerve stimulation, using this technique, has been shown to significantly reduce the pain caused by CN, it is likely to not be used as the initial, non-surgical treatment. It is most useful in individuals whose pain is resistant to other non-surgical medical treatments.[3]

SURGICAL TREATMENT

In many instances, individuals who have refractory CN consider a surgical treatment when more conservative measures are unsuccessful. Individuals who undergo surgical procedures are usually those whose non-surgical medical treatment has been ineffective or those who choose to forgo non-surgical treatment and instead elect surgery. Fortunately, there are several highly effective surgical methods that can provide a significant reduction of pain for these individuals. The benefit of surgical procedures is that there is direct visualization of the nerve and its surroundings, making it safer for the patient and limiting the possibility of potentially harmful effects. The potential negative effect of surgical treatment is that anytime there is a surgical procedure, there are risks of general anesthesia, unintentional nerve damage, or damage to surrounding tissue.

First, individuals can undergo an open surgical procedure to release the cluneal nerve from its entrapment. This procedure is done by making a small incision in the gluteal area where the pain is localized. The surgeon then dissects the subcutaneous soft tissue and exposes and identifies the nerve by placing a nerve stimulator in the area. Related to the anatomical path of the nerve, the thoracolumbar fascia is cut along the path of the superior cluneal nerve until there are no kinks in the nerve.[71] This will free the nerve from its entrapment. This technique was found to be highly effective, resulting in all 34 individuals who underwent the procedure reporting symptom improvement, while none of the individuals reported worsening of their symptoms.[71] This procedure is relatively quick and lacks in serious potential complications with the average duration of surgery being 45 minutes. The fallback of this procedure, when compared to other surgical treatments, is that it requires a larger incision site, which potentially exposes the individual to more surgical site trauma.[71,72]

The second surgical approach is a minimally invasive surgery. In the minimally invasive technique, a transgluteal endoscopic approach is performed, in which a surgeon uses a camera and endoscopic instruments, each inserted into a port surrounding the path of the cluneal nerve.[72] The surgeon then uses the camera and instruments to decompress the nerve, similarly to the open approach. This procedure has been shown to provide excellent relief of pain caused by Cluneal neuralgia. At one year, 73% of individuals reported having a good treatment response, while 40% claimed that the procedure provided optimal results. This is increased from 57% and 31% at six months, respectively.[72] A major benefit of the minimally invasive procedure is that individuals are left with two small incision sites, both less than 5 mm, which decreases the chance of incision site complications.[72] A downfall of this procedure is that the tissue is not as clearly visualized, which can lead to unintended damage to the nerve or surrounding tissue.

A problem that some surgeons run into during surgery is identifying the cluneal nerve surgery, due to its small size, and confirming that it has been sufficiently decompressed. A unique tool that surgeons can utilize to help solve this problem is intraoperative indocyanine green video angiography.[73] Indocyanine green video angiography provides real-time patency information and flow dynamics of the vessels supplying the cluneal nerve.[73] This allows surgeons to more accurately identify the nerve and confirm that adequate decompression is achieved. This technology has been shown to be effective in identifying not only the cluneal nerve but also other peripheral nerves, leading to a more precise and effective decompressive surgery.

Overall, individuals who undergo surgery for CN report excellent long-term treatment regarding their pain. The reoccurrence rate is 13%.[74] This is likely due to a lack of full decompression of the nerve, an entrapment of other branches of the nerve, or entrapment of a different nerve altogether.[74] This can be avoided by decompressing as many of the cluneal branches as possible during the original surgery. This would prevent the need for another operation, which would expose the individual to the risks associated with doing so. All individuals who experience a reoccurrence experienced it in the first 25 months after surgery, suggesting the need for reevaluation for a minimum of 25 months post-op.[74]

CONCLUSION

The cluneal nerves, particularly the SCN and MCN, are cutaneous sensory nerves that innervate the posterior lumbar and buttock region and infrequently cause LBP.[2,16,18] A thorough review of SCN and MCN anatomy reveals that CN is most often caused by entrapment from various etiologies, and can lead to varied symptoms.[75] The SCN is most likely to become entrapped or constricted in the OT, while the MCN can be entrapped within or under the LPSL.[3,4,15,16,20,21,26–28] Risk factors leading to the development of SCN-E or MCN-E most notably include age, sex, vertebral fractures, and past surgical history.[4,32,33,42] CN most commonly presents as LBP, although it can often have leg symptoms or mimic radiculopathy or SIJ pain.[62] The following specific criteria should be met when diagnosing cluneal nerve entrapment: pain exacerbated by movement, trigger point eliciting tenderness, numbness, or radiating pain on palpation, and relief of symptoms with a nerve block.[4,76] Once cluneal nerve entrapment is diagnosed, it can be managed conservatively, with nerve blocks, high-frequency thermal coagulation, capsaicin patches to "defunctionalize" nociceptor fibers, and wireless peripheral nerve stimulation.[77] These options can help avoid surgical treatment. However, many forms of efficacious surgical treatment possibilities exist, and they provide patients with excellent long-term outcomes. These surgical approaches include cluneal nerve release or minimally invasive endoscopic decompression and can be augmented by indocyanine green video angiography.

In summary, the etiology of LBP should be investigated for possible CN. Spinal surgeons and clinicians evaluating LBP should consider SCN-E and MCN-E before treatment of these patients to prevent misdiagnoses. Subsequently, conservative and surgical options should be evaluated on an individual level and based on symptom severity to best treat patients suffering from LBP related to CN.

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