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CASE REPORT

Low back pain due to middle cluneal nerve entrapment neuropathy

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Abstract
Purpose The etiology of low back pain (LBP) is complicated and the diagnosis can be difficult. Superior cluneal nerve entrapment neuropathy (SCN-EN) is a known cause of LBP, although the middle cluneal nerve (MCN) can be implicated in the elicitation of LBP.
Methods A 76-year-old woman with a 4-year history of severe LBP was admitted to our department in a wheelchair. She complained of bilateral LBP that was exacerbated by lumbar movement. Her pain was severe on the right side and she also suffered right leg pain and numbness. Based on palpation and nerve blocking findings we diagnosed SCN-EN and MCN entrapment neuropathy (MCN-EN).
Results Her symptoms improved with repeated SCN and MCN blocking; the MCN block was the more effective and her symptoms improved. As her right-side pain around the MCN -EN with severe trigger pain recurred we performed microscopic right MCN neurolysis under local anesthesia. This led to dramatic improvement of her LBP and leg pain and the numbness improved. At the last follow-up, 7 months after surgery, she did not require pain medication.
Conclusions The MCN consists of sensory branches from the dorsal rami of S1–S4. It sandwiches the sacral ligament between the posterior superior and inferior iliac spine as it courses over the iliac crest. Its entrapment at this hard orifice can lead to severe LBP with leg symptoms. An MCN block effect is diagnostically useful. Less invasive MCN neurolysis under local anesthesia is effective in patients who fail to respond to observation therapy.

Keywords Low back pain · Middle cluneal nerve · Entrapment neuropathy · Neurolysis

Introduction

Low back pain (LBP) is elicited by heterogeneous factors. Its etiology can be complex and its diagnosis difficult [1]. Superior cluneal nerve entrapment neuropathy (SCN-EN) is a known cause of LBP and some patients suffer severe symptoms [2–9]. It can be less invasively treated by SCN blocking and neurolysis. In rare cases, the middle cluneal nerve (MCN) [9, 10] is implicated in the elicitation of LBP [8, 11] and its clinical course and etiology remain unclear. We report the successful treatment of a patient with LBP due to MCN-EN.

Case report

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

A 76-year-old woman with Parkinson's disease (PD) reported suffering LBP for 4 years. Her symptoms gradually worsened and 18 months ago she became unable to walk more than 10 m because of LBP. Despite observation and treatment at a local hospital her symptoms failed to improve.

She was admitted to our hospital 10 months ago. She manifested no motor weakness or urinary problems. She

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complained of bilateral LBP that required her to use a wheelchair. Her pain was exacerbated by lumbar movement; it was severe on the right side and she complained of right leg pain and numbness [numerical rating scale (NRS) 8, Roland-Morris Disability Questionnaire (RDQ) score 19, Japanese Orthopedic Association (JOA) score 11, Hoehn-Yahr scale 4]. Lumbar radiograms showed L4 slippage without instability. Magnetic resonance imaging (MRI) scans revealed multilevel lumbar spinal canal stenosis (Figs. 1, 2).

Palpation identified two tender sites; one was located 70 mm lateral to the midline on the iliac crest and the other 35 mm caudal to the posterior superior iliac spine (PSIS) at a slightly lateral point at the edge of the iliac crest (Fig. 3). Tenderness at the first site led us to suspect SCN-EN. Based on tenderness at the second site, we suspected MCN-EN because it is there where the MCN penetrates the long posterior sacroiliac ligament (LPSL) between the PSIS and the posterior inferior iliac spine (PIIS). Her pain transiently but dramatically improved with SCN and MCN block with 0.75% ropivacaine (2 ml each). Our diagnosis was SCN-EN and MCN-EN. MCN blockage was the more effective. Her symptoms recurred and we performed three more SCN- and four MCN blocks. Her pain improved and she was discharged with an NRS of 1, an RDQ score of 0, and a JOA score of 25. Although her trigger point pain due to SCN-EN disappeared, some pain due to MCN-EN persisted.

She subsequently reported gradually recurring right-side pain around the MCN-EN and severe trigger pain (NRS 8, RDQ score 11, JOA score 7). Her SCN-EN pain and trigger point pain at the SCN had disappeared. With the patient in the prone position, the senior author (T.I.) performed microscopic right MCN neurolysis under local anesthesia. Using the gluteal maximus muscle (GMaM)-splitting approach with a 7-cm linear incision (Fig. 3), the GMaM fascia was opened to explore the distal portion of the MCN. After confirming that it penetrated the LPSL to the proximal site (Fig. 4) we decompressed the MCN to that site by excising the LPSL.

Subsequently, her LBP, leg pain, and numbness dramatically improved. At the last follow-up, 7 months after surgery, her LBP had not recurred and she required no pain medication (NRS 0, RDQ score 0, JOA score 21, Hoehn–Yahr scale 3).

Discussion

LBP due to MCN-EN

The SCN harbors sensory branches from the dorsal rami of the lower thoracic and lumbar roots. SCN entrapment around the iliac crest elicits pain [2–9]. Between 1.6 and 14% of all LBP is due to SCN-EN and it is unexpectedly rare [5, 6]. The MCN contains sensory branches from the dorsal rami of S1–S4. It sandwiches the LPSL between the PSIS and the PIIS where it courses over the iliac crest and runs to the buttocks [9, 10]. Based on their anatomical study, Tubbs et al. [9] reported that the MCN cannot be entrapped because it runs superficial to the PSL. McGrath and Zhang [10], on the other hand, reported that the lateral branches of the dorsal sacral rami penetrate the LPSL and that pain elicitation is possible due to MCN-EN. As did we, others [8, 11] obtained a good outcome by addressing the MCN that elicited LBP, however, details regarding MCN decompression surgery were not provided.

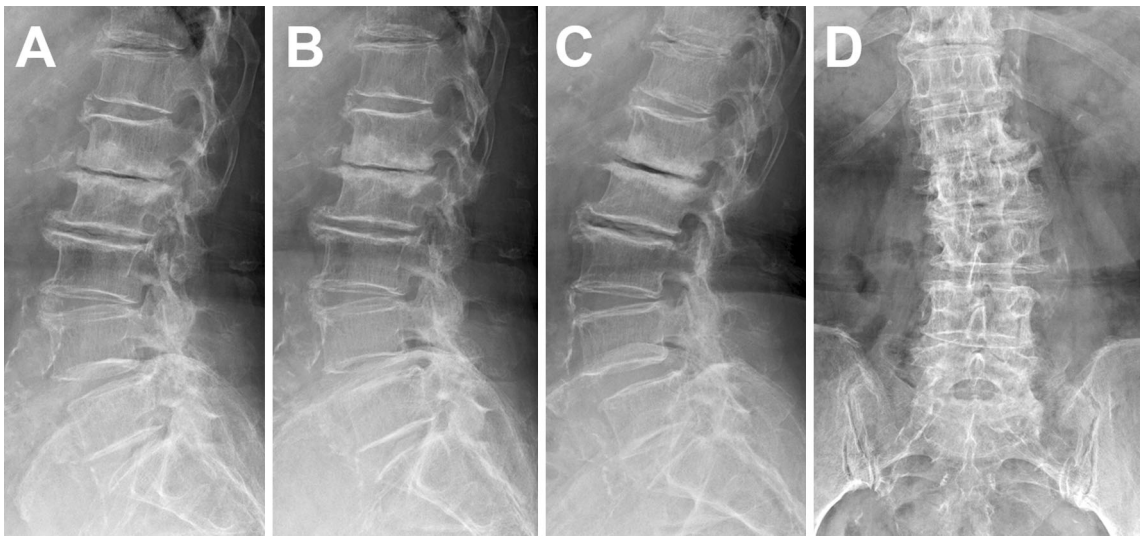

Fig. 1 Lumbar radiograms showed slight scoliosis and L4 slippage without instability. **a–c** Lateral views (**a** neutral, **b** flexion, **c** extension). **d** Antero-posterior view

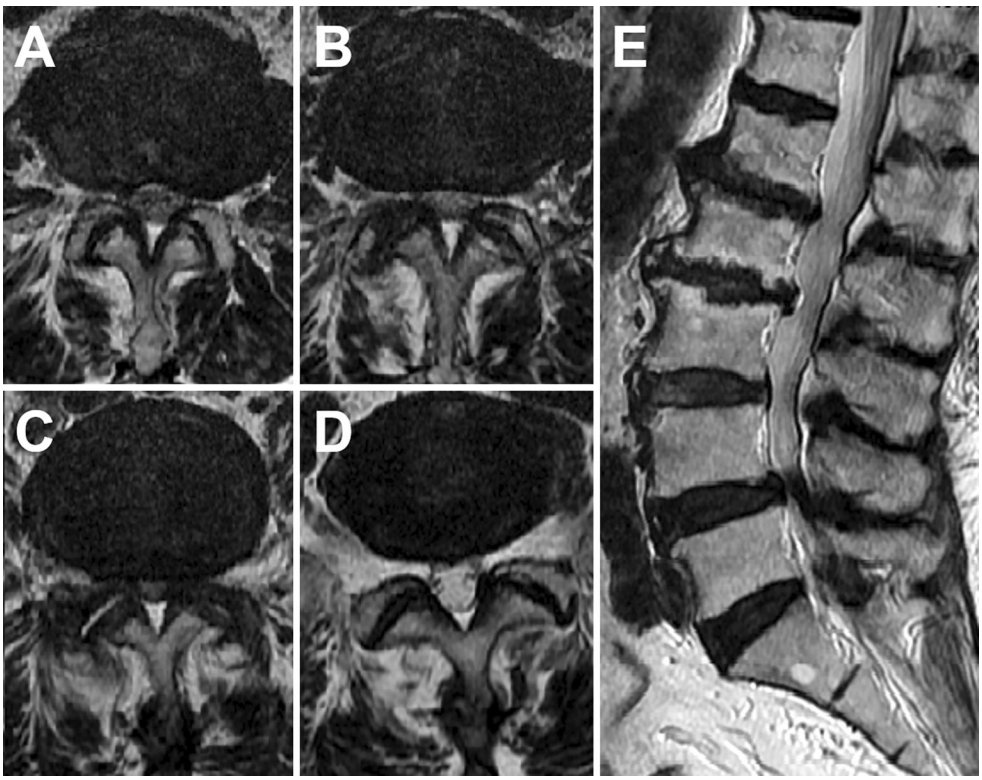

Fig. 2 MRI (T2-weighted image) revealed lumbar spinal canal stenosis from L2/3 to L4/5. **a** L2/3 axial image. **b** L3/4 axial image. **c** L4/5 axial image. **d** L5/S1 axial image. **e** Sagittal image

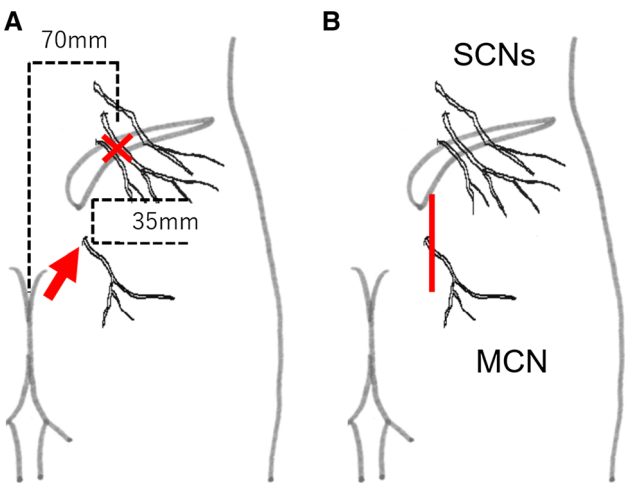

Fig. 3 Schematic of the right lumbar region and hip. **a** Palpation confirmed two trigger points. Superior cluneal nerve entrapment neuropathy was identified 70 mm lateral to the midline on the iliac crest (*X*). Middle cluneal nerve (MCN) entrapment neuropathy was located 35 mm caudal to the posterior superior iliac spine (PSIS) and at a slightly lateral point at the edge of the iliac crest. The MCN passed through the long posterior sacroiliac ligament between the PSIS and the posterior inferior iliac spine (*arrow*). **b** For MCN neurolysis we made a 70-mm-long skin incision across the trigger point on the buttock

Symptoms and diagnosis of MCN-EN

Patients with LBP due to SCN-EN report that several lumbar postures and motions exacerbate their symptoms. Leg symptoms associated with LBP were reported by 47–84% of patients with SCN-EN [5–8]. The condition mimics radiculopathy due to lumbar disease [12]. In our patient, LBP due to MCN-EN was increased by lumbar movement and she suffered leg symptoms on the affected side. Our and earlier observations [8, 11] indicate that, like patients with SCN-EN, some patients with MCN-EN may experience leg symptoms.

The diagnosis of SCN-EN is based on the symptomatology and on pain reduction by SCN blockage [2–8]. In our patient the confirmed trigger point was located 35 mm caudal to the PSIS and at a slightly lateral point at the edge of the iliac crest where the MCN passes and runs through the LPSL between the PSIS and PIIS. As the local injection of ropivacaine resulted in dramatic pain alleviation we diagnosed MCN-EN. MRI study revealed severe lumbar stenosis. However, as elicitation of her LBP was attributable to the area of the MCN, and as we detected an MCN-EN trigger point, we suspected that at least some of her LBP involved MCN-EN. In fact, MCN block alone

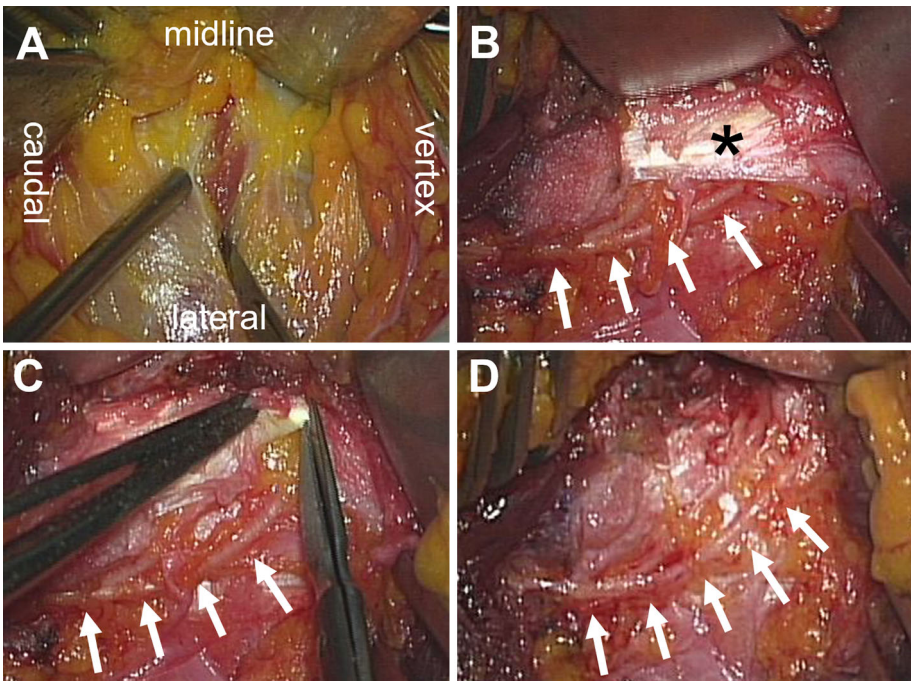

Fig. 4 Intraoperative photographs. At the *top* of the *photo* is the midline. The vertex side is on the *right*. **a** To approach the middle cluneal nerve (MCN) we split the gluteus maximus muscle. **b** Exploration of the distal portion of the MCN (*arrows*) showed that it penetrated the long posterior sacroiliac ligament (LPSL) (*asterisk*) to the proximal portion. **c**, **d** We decompressed the MCN (*arrows*) proximally by excising the LPSL

dramatically alleviated her LBP and we did not think that treatment of her severe lumbar stenosis was necessary.

Etiology of MCN-EN

The etiology of MCN-EN and of SCN-EN remains unclear. The pathology of SCN-EN may be associated with not only compression at the orifice of the thoracolumbar fascia, but also with an increased paravertebral muscle tonus and SCN stretching due to changes in posture and motion [7, 8]. Increased traction and entrapment of the SCN in the paravertebral muscle may exacerbate LBP in patients with SCN-EN [7]. The MCN can also be compressed at the hard orifice between the iliac bone and the LPSL. After passing through the orifice, it passes the GMaM and reaches the skin. MCN-EN may be associated with increased GMaM tonus and MCN stretching due to posture and motion changes.

Dysfunction of the sacroiliac joint (SIJ) may result in SIJ pain. Repetitive loading of the SIJ may affect structures around this joint although it is reinforced with hard ligaments and can move only slightly [13]. The MCN is sandwiched by a strong LPSL between the PSIS and the PIIS around the SIJ and slight but repetitive SIJ movement, loading, and minor subluxation may lead to MCN-EN.

The incidence of LBP is particularly high in PD patients and its treatment is often difficult and insufficient [14–17]. Elsewhere [3, 7] we reported that LBP in these patients was improved by SCN-EN treatment and that their Hoehn-Yahr scale decreased. Many PD patients suffer musculoskeletal pain related to rigidity and akinesia [14–17] and their increased muscle tonus and abnormal posture due to overloading of the paravertebral muscles may elicit SCN-EN. As our patient had PD, MCN-EN may have contributed to her severe LBP.

Conclusion

We report the successful treatment of a patient with LBP due to MCN-EN. The diagnosis is based on the symptomatology and on pain reduction by MCN blockage. Less invasive MCN neurolysis under local anesthesia is effective in patients who fail to respond to observation therapy. We suggest that MCN-EN treatment should be considered when symptoms and physiological findings are suggestive of its presence.

Compliance with ethical standards

Conflict of interest The authors have no personal, financial, or institutional interests, in any of the drugs, materials, or devices described article.

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