Surgical treatment of middle cluneal nerve entrapment neuropathy: technical note

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OBJECTIVE The etiology of low-back pain (LBP) is heterogeneous and is unknown in some patients with chronic pain. Superior cluneal nerve entrapment has been proposed as a causative factor, and some patients suffer severe symptoms. The middle cluneal nerve (MCN) is also implicated in the elicitation of LBP, and its clinical course and etiology remain unclear. The authors report the preliminary outcomes of a less invasive microsurgical release procedure to address MCN entrapment (MCN-E).

METHODS The authors enrolled 11 patients (13 sites) with intractable LBP judged to be due to MCN-E. The group included 3 men and 8 women ranging in age from 52 to 86 years. Microscopic MCN neurolysis was performed under local anesthesia with the patient in the prone position. Postoperatively, all patients were allowed to walk freely with no restrictions. The mean follow-up period was 10.5 months. LBP severity was evaluated on the numerical rating scale (NRS) and by the Japanese Orthopaedic Association (JOA) and the Roland-Morris Disability Questionnaire (RDQ) scores.

RESULTS All patients suffered buttock pain, and 9 also had leg symptoms. The symptoms were aggravated by standing, lumbar flexion, rolling over, prolonged sitting, and especially by walking. The numbers of nerve branches addressed during MCN neurolysis were 1 in 9 patients, 2 in 1 patient, and 3 in 1 patient. One patient required reoperation due to insufficient decompression originally. There were no local or systemic complications during or after surgery. Postoperatively, the symptoms of all patients improved statistically significantly; the mean NRS score fell from 7.0 to 1.4, the mean RDQ from 10.8 to 1.4, and the mean JOA score rose from 13.7 to 23.6.

CONCLUSIONS Less invasive MCN neurolysis performed under local anesthesia is useful for LBP caused by MCN-E. In patients with intractable LBP, MCN-E should be considered.

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KEYWORDS low-back pain; middle cluneal nerve; neuropathy; neurolysis; surgical results; surgical technique

THE etiology of low-back pain (LBP) is heterogeneous and is unknown in some patients with chronic pain.⁶ Superior cluneal nerve entrapment (SCN-E) has been proposed as a causative factor; in some patients the symptoms are severe.^{1,2,4,5,7,10–12,15,17,20–23,25,26} The reported incidence of SCN-E ranges from 1.6% to 14%.^{15,17} It can be less invasively treated by SCN blocking and neurolysis. The middle cluneal nerve (MCN) can also be involved in the elicitation of LBP in which the clinical course and etiology remain unclear.^{3,13,18,22,25} We report the preliminary

outcomes of a less invasive microsurgical release procedure used to address MCN entrapment (MCN-E).

Methods

Diagnostic Criteria

We modified previously reported diagnostic criteria for SCN-E to diagnose MCN-E.^{3,17,20,25} LBP due to MCN-E elicits pain on the affected side; it involves the buttock and worsens with lumbar movements. The trigger point is 35

ABBREVIATIONS GMaM, GMeM = gluteus maximus muscle, gluteus medius muscle; JOA = Japanese Orthopaedic Association; LBP = low-back pain; LPSL = long posterior sacroiliac ligament; MCN, MCN-E = middle cluneal nerve, MCN entrapment; NRS = numerical rating scale; PIIS = posterior inferior iliac spine; PSIS = posterior superior iliac spine; RDQ = Roland-Morris Disability Questionnaire; SCN, SCN-E = superior cluneal nerve, SCN entrapment; SIJ = sacroiliac joint.

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FIG. 1. Schematic of the right lumbar area and hip. **A:** The trigger point (arrow) at the site of MCN-E is located 35 mm (D, dashed line) caudal to the PSIS and at a slightly lateral point at the edge of the iliac crest. **B:** For MCN neurolysis we made a 7-cm skin incision (red line) across the trigger point on the buttock. Copyright Juntaro Matsumoto. Published with permission. Figure is available in color online only.

mm caudal to the posterior superior iliac spine (PSIS) at a slightly lateral point at the edge of the iliac crest (corresponding to the nerve compression zone). Patients report numbness and radiating pain in the MCN area on trigger-point compression (Fig. 1A).

To diagnose MCN-E, we block the nerve at the trigger point in the buttock with 2 ml 1% lidocaine. We apply the block carefully to avoid its reaching the sacroiliac joint (SIJ). Symptom abatement by more than 50% should oc-

cur within 2 hours after nerve blockage. Patients whose pain was sufficiently controlled by several MCN blocks were excluded from this study; those who reported pain recurrence after the analgesic effect had worn off were considered surgical candidates.

Patients and LBP Assessment

Between May 2016 and January 2017, 11 patients with intractable LBP due to MCN-E underwent MCN neurolysis (Fig. 2, Table 1). The group included 3 men and 8 women ranging in age from 52 to 86 years; the affected site was unilateral in 9 and bilateral in 2 patients (13 sites). The interval between symptom onset and treatment ranged from 2 months to 16 years; follow-up ranged from 7 to 15 months. None of the 11 patients had undergone iliac crest harvest or suffered trauma to the affected area. Excluded were patients whose LBP improved under observation therapy, including the administration of oral medications such as nonsteroidal antiinflammatory drugs, pregabalin, and tramadol; patients who responded to repeat MCN blockage lasting at least 3 months; those who refused surgical treatment; and patients whose general condition or dementia rendered surgery inadvisable. Our study was approved by the institutional ethics committee of Kushiro Rosai Hospital; prior written informed consent was obtained from all patients included in this study.

Surgical Technique

With the patient in the prone position and under local anesthesia, the senior author (T.I.) performed microscopic MCN neurolysis. After the gluteus maximus muscle (GMaM) was split with a 7-cm linear incision across the

FIG. 2. Algorithm for patient recruitment and treatment.

TABLE 1. Characteristics of our patients with LBP due to MCN-E

Case No.	Age (yrs)	Sex	Site	Symptom	Treated Branches	Reop	FU (mos)	NRS Score		RDQ Score		JOA Score	
								Preop	Postop	Preop	Postop	Preop	Postop
1	64	F	Bilat	LBP + LP	1 (rt), 1 (lt)	Yes (rt)	15	8	2	4	0	13	24
2	75	F	Lt	LBP + LP	1		13	4	2	5	1	21	26
3	86	F	Rt	LBP + LP	1		11	8	1	9	0	11	21
4	75	F	Lt	LBP + LP	1		11	5	1	15	0	12	23
5	76	F	Rt	LBP + LP	1		11	8	0	11	0	7	21
6	73	M	Rt	LBP + LP	1		11	9	2	19	2	11	27
7	67	M	Rt	LBP + LP	1		10	5	1	10	2	15	26
8	52	M	Bilat	LBP + LP	1 (rt), 2 (lt)		10	8	1	8	2	24	27
9	72	F	Rt	LBP	1		8	8	2	9	4	13	20
10	80	F	Rt	LBP + LP	3		8	8	2	14	3	9	19
11	61	F	Lt	LBP	1		7	6	1	15	1	15	26

FU = follow-up; LP = leg pain.

trigger point from the PSIS to the caudal side, the GMaM fascia was opened to explore the distal portion of the MCN (Figs. 1B, 3A–C, and 4A). To identify the MCN, an electrical nerve stimulator (Neuropack MEB2306, Nihon Kohden) featuring bipolar forceps and a connective wire was used. The rate, duration, and intensity of stimulation were 1 Hz, 0.2 msec, and 1.0–3.0 mA, respectively. Stimulation of the affected nerve triggers radiating pain in the MCN area that is similar to the radiating pain elicited by manual compression of the trigger point.

Usually, the MCN penetrates the long posterior sacro-

iliac ligament (LPSL) between the PSIS and the posterior inferior iliac spine (PIIS). We confirmed visualization of the PSIS and the PIIS, and of the LPSL between the PSIS and PIIS. The MCN slants from caudolateral to rostromedial and penetrates the LPSL to the proximal site. The LPSL was sharply cut and excised with microscissors in a distal-to-rostral direction along the MCN to release the entrapped portion (Figs. 3D, 3E, 4B, and 4C). The distal portion of the MCN was also released to render the MCN moveable. This decompression procedure reduces MCN tension. When the radiating pain elicited by manual direct

FIG. 3. Illustration of neurolysis for MCN-E. **A:** Exposure of GMaM. **B:** After splitting the GMaM, the ventral fascia of the GMaM is opened to explore the distal portion of the MCN. **C:** The MCN slants from caudolateral to rostromedial and penetrates the LPSL to the proximal site. **D:** For MCN decompression, the LPSL is sharply cut and excised with microscissors in a distal-to-rostral direction to the MCN. **E:** The MCN is released by cutting the LPSL and the nerve is decompressed and mobilized. **F:** After MCN decompression, we confirm disappearance of the radiating pain on direct manual compression of the MCN in the surgical field. Copyright Juntaro Matsumoto. Published with permission. Figure is available in color online only.

compression in the surgical field disappeared, we considered the procedure complete (Fig. 3F).

Immediately after surgery, all patients were allowed to walk freely with no restrictions or external fixation. They were discharged on the next day and they returned to their activities of daily living.

Evaluation of Clinical Outcomes

LBP severity was evaluated on the numerical rating scale (NRS) and by the Japanese Orthopaedic Association (JOA) and the Roland-Morris Disability Questionnaire (RDQ) scores.^{8,9} For statistical analysis we subjected our data to the paired t-test by using Statmate III software (ATMS Co., Ltd.). Differences of $p < 0.05$ were considered statistically significant.

Results

Symptoms of MCN-E

All 11 patients reported severe LBP and were admitted to our institute. All suffered buttock pain, and 9 also had leg symptoms. The symptoms were aggravated by standing ($n = 5$), prolonged standing ($n = 3$), lumbar flexion ($n = 2$), rolling over ($n = 2$), prolonged sitting ($n = 2$), and especially by walking ($n = 9$). The trigger point coincided with the MCN-E site. When blockage of the MCN for diagnostic purposes lessened the pain by more than 50% we made a diagnosis of MCN-E. Patients who received oral medication for MCN-E also underwent MCN blocking. The number of blocks depended on the patient's condition and the effect of the block (average number of blocks 2.5, range 1–6). Patients whose status failed to improve underwent MCN neurolysis.

Of the 11 patients, 5 had undergone lumbar surgery and 1 had Parkinson disease.¹³ Radiological studies revealed lumbar spinal canal stenosis in 4, scoliosis in 2, and an old vertebral compression fracture in 1 patient; 3 had undergone SCN surgery and 4 had been treated by gluteus medius muscle (GMeM) decompression. Preoperatively their mean NRS, RDQ, and JOA scores were 7.0, 10.8, and 13.7, respectively.

Surgical Results

The 11 patients underwent MCN neurolysis; the number of nerve branches addressed were 1 in 9 patients, 2 in 1 patient, and 3 in 1 patient. Even in patients who underwent surgery for 2 or more MCNs, enlargement of the incision was not necessary because all treated MCNs were between the PSIS and PIIS. Because SCN surgery is performed above the PSIS and GMeM decompression is performed outside the buttocks, a previous history of such operations (SCN, $n = 3$; GMeM, $n = 4$) did not affect our MCN operation.

One patient (case 1) required reoperation due to LBP recurrence in the early follow-up period. Although her symptoms improved initially, they reappeared 8 months later on her right side and she underwent MCN redecompression. Although one MCN branch had been decompressed during the first operation, pain recurrence was attributable to another strangulated MCN.

There were no local or systemic complications dur-

FIG. 4. Photographs of our procedure to release MCN-E. **A:** The MCN (arrows) is seen to penetrate the LPSL (asterisk). **B:** The LPSL (asterisk) is opened with microscissors in a distal-to-rostral direction along the MCN (arrows). **C:** The entrapped MCN (arrows) is released from the LPSL. Figure is available in color online only.

ing or after surgery. All symptoms, including leg symptoms associated with MCN-E, improved significantly ($p < 0.05$); the mean posttreatment NRS score was 1.4 (range 0–2), the mean RDQ score was 1.4 (range 0–4), and the mean JOA score was 23.6 (range 19–27).

Discussion

LBP due to MCN-E

The SCN consists of sensory branches from the dorsal rami of the lower thoracic and lumbar posterior roots. Its entrapment around the iliac crest results in LBP.^{5,10,11,15,17,19,22,25} The MCN is composed of sensory branches from the dorsal rami of S-1 to S-4.¹⁴ It passes through the LPSL between the PSIS and the PIIS, slants over the iliac crest, and runs to the buttocks.^{14,18,25} According to Tubbs et al.,²⁵

the nerve cannot be entrapped because it runs superficial to the LPSL. McGrath and Zhang¹⁸ reported that because the MCN penetrates the LPSL, its entrapment can elicit pain. In a cadaveric study, Konno et al.¹⁴ reported that 30% of the MCNs on 30 sides featured a branch that traversed under the LPSL. Their anatomical findings suggested that MCN-E is not a rare clinical entity. Others^{3,13,22} obtained good outcomes by MCN decompression in patients with LBP due to MCN-E. Thus, the etiology and the treatment of LBP due to MCN-E remain controversial.

SCN-E was reported to be implicated in 1.6%–14% of all cases of LBP.^{15,17} Our search of the literature found no documentation of the incidence of MCN-E in patients with LBP.

MCN-E Diagnosis

LBP due to SCN-E is induced and exacerbated by several lumbar postures and movements,²⁰ and 47%–84% of LBP patients with SCN-E present with leg symptoms.^{15,17,19,22} Our 11 patients with LBP due to MCN-E also reported symptom worsening elicited by lumbar movements, especially those involved in walking; 9 suffered leg symptoms.^{3,22} Because the condition may mimic radiculopathy due to lumbar disease,²⁴ a differential diagnosis is important.

In patients with SCN-E, radiological and electrophysiological studies do not provide useful diagnostic information. Therefore, their clinical symptoms must be assessed carefully.^{5,10,11,15,17,19,22} To diagnose MCN-E, we modified SCN-E diagnostic criteria and identified the area of the MCN implicated in the elicitation of LBP and the trigger point at the site of MCN-E. We also evaluated the response to MCN blockage.

We cannot comment on the utility of electromyography for the diagnosis of MCN-E because the nerve is thin, peripheral, and difficult to identify on the skin surface. Additional studies are needed to determine whether electromyography is useful for the diagnosis of MCN-E.

MCN-E Surgery

In patients with MCN-E whose pain is not relieved by conservative treatment with drugs or by MCN block, surgical release of the entrapment may be effective. There are few descriptions of the surgical procedures that address MCN-E neuropathy.^{3,13,22} In a single-case report, Aota³ found MCN decompression under general anesthesia to be useful. Strong and Davila²² also performed MCN release under general anesthesia, but provided no surgical details. To the best of our knowledge, ours is the first documentation of a surgical procedure that successfully addressed MCN-E.

Like the SCN, the MCN is thin and its intraoperative identification can be difficult. On the other hand, unlike the SCN, the MCN passes through a narrow space between the PSIS and the PIIS. Our experience suggests that placing an incision at the trigger point and using a nerve stimulator under microscopic observation is useful for the identification of the MCN because it is a sensory nerve that can be easily monitored.

One of our 11 patients (case 1) required reoperation in

the early follow-up period. During the first operation we performed a single MCN decompression procedure and neglected to explore other MCNs. Konno et al.¹⁴ reported that 11% of all MCNs featured 2 branches. Among the 13 sites addressed in our series, 2 featured more than 1 branch (cases 8 and 10). This observation shows the need for careful inspection during MCN surgery. In another patient (case 1), we also missed an MCN because we intraoperatively confirmed the loss of tenderness in only the decompressed nerve. Therefore, the abatement of pain elicitation must be confirmed in the entire surgical field.

Our less invasive surgery under local anesthesia facilitates the intraoperative visual- and patient-based confirmation of adequate MCN decompression. Patients who suffered radiating pain due to MCN compression reported its disappearance on surgical decrease of MCN tension. To confirm alleviation of MCN tension, we applied traction or moved the MCN under the microscope. However, these procedures require expertise. The disappearance of radiating pain attributable to MCN kinking at the entrapment point can be confirmed by directly compressing the MCN at the trigger point.

Etiology of MCN-E

The etiology of MCN-E and SCN-E remains unclear. Pain due to SCN-E is elicited by various lumbar postures and dynamic motions.^{1,2,4,5,10,15,20,23} This suggests a relationship between the etiology of SCN-E and SCN stretching with posture and motion.¹⁹ Because LBP due to MCN-E is also related to various lumbar postures, we think that the etiology of MCN-E may also be related to MCN stretching with posture and motion.

Although SCN-E has been recognized as entrapment of the SCN at the osteofibrous tunnel resulting in LBP,^{17,22} decompression of SCN branches that do not pass through the osteofibrous tunnel may successfully address SCN-E.^{5,15,20,22} This suggests that the etiology of SCN-E may also involve factors other than nerve compression within the osteofibrous tunnel.¹⁹ It might be associated not only with compression at the orifice of the thoracolumbar fascia, but also with increased paravertebral muscle tonus and SCN stretching with changes in posture and motion.¹⁹

The MCN may be compressed at the narrow gap between the iliac bone and the LPSL. It then passes the GMaM and reaches the skin. An increased GMaM tonus and MCN stretching due to posture and movement¹³ may contribute to MCN-E. Repeat loading of the SIJ may affect structures around this joint and its dysfunction may elicit SIJ pain.¹⁶ Also, because the MCN passes through the narrow gap under the LPSL between the PSIS and the PIIS around the SIJ, slight but repetitive SIJ loading and minor subluxation may result in MCN-E.¹³

Our study has some limitations. The number of patients was small and studies on larger populations are needed. We based our diagnosis on clinical symptoms and evaluation of the treatment outcomes with respect to symptom improvement reported by the patients. Placebo nerve blockage with normal saline may help to distinguish the placebo effect of the block—however, ethical considerations forbid this approach. Therefore, the objective diagnosis of MCN-E remains difficult. Last, the postoperative

follow-up period was relatively short (mean 10.5 months). Long-term follow-up studies are underway to evaluate the therapeutic effect of our procedure and to assess the recurrence rate due to scar formation or adhesion.

Conclusions

MCN-E should be considered in patients with intractable LBP. MCN neurolysis performed under local anesthesia was an effective and less invasive treatment in our series.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Matsumoto, Isu. Acquisition of data: Matsumoto. Analysis and interpretation of data: Kim. Drafting the article: Isu. Critically revising the article: Isu, Kim. Reviewed submitted version of manuscript: Isu, Kim. Approved the final version of the manuscript on behalf of all authors: Matsumoto. Administrative/technical/material support: Isu, Iwamoto, Morimoto, Isobe. Study supervision: Kim.

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