Original Article
Efficacy of continuous lumbar sympathetic nerve block for cold allodynia in patients with lumbar disc herniation

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Abstract: Cold allodynia is a complaint in patients with lumbar disc herniation (LDH), and significantly impairs their quality of life. We treated cold allodynia with continuous lumbar sympathetic nerve block (CLSNB) with patient-controlled analgesia (PCA) and determined the therapeutic outcome in long-term follow-up. Eight patients with a median age of 52.0 years were enrolled between September 2013 and August 2014. These patients had long-term cold allodynia in their affected lower extremity, and did not respond to established treatments. CLSNB was finally performed. The patients were followed up for at least 24 weeks. The average cold allodynia score on the visual analog scale (VAS) declined significantly from 7.1 before treatment to 1.6 at discharge, 1.8 at 1 week after discharge, 3.2 at 4 weeks, 4.0 at 8 weeks, and 4.6 at 24 weeks (P<0.05). All patients obtained immediate continuous cold relief during hospitalization. Seven of 8 patients had satisfactory pain relief at 1 week after discharge, 6 of 8 at 4 weeks, 5 of 8 at 8 weeks, 4 of 8 at 12 weeks, and 4 of 8 at 24 weeks. There were no reported complications. CLSNB is an effective and safe technique in treating cold allodynia in patients with LDH, and can provide long-term benefit.

Keywords: Continuous lumbar sympathetic nerve block, cold allodynia, lumbar disc herniation

Introduction
Cold allodynia is a common symptom in a variety of diseases, and is characterized by pain with normally innocuous cold stimulation and an avoidance response in a cold environment [1-3]. It is a complaint in patients with lumbar disc herniation (LDH), and significantly impairs their quality of life.

Previous studies reported improvement in cold allodynia in patients with discogenic visceral pain or complex regional pain syndrome (CRPS) using continuous lumbar sympathetic nerve block (CLSNB) [4, 5]. However, there are no reports on the efficacy of CLSNB in treating cold allodynia in patients with LDH. The purpose of this study was to evaluate the efficacy of CLSNB in treating cold allodynia in the lower extremity in patients with LDH during 24 weeks of follow-up.

Methods
Patients
Eight patients with LDH who had clinical symptoms and physical findings suggestive or indicative of cold allodynia in their affected lower extremity were selected as participants in this study. There was no history of cold allodynia in the lower extremity prior to development of LDH. Despite a variety of treatments, the cold allodynia remained unimproved. Exclusion criteria were spondylolisthesis, diabetes mellitus, vascular diseases, injuries, inflammatory arthritis in the lower extremity, and cognitive impairment. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of the Xuan Wu Hospital, Capital Medical University. Written informed consent was obtained from all participants.

CLSNB
Patients who agreed to treatment underwent percutaneous CLSNB using a postero-lateral approach with an 18-gauge needle. Under computed tomography (CT) guidance, the needle was inserted into the paravertebral sympathetic trunk at the level of the L4 vertebral body. A 20-gauge catheter was inserted into the paravertebral sympathetic trunk through the needle (Figure 1A). CT was used to reconfirm catheter
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Position by infusion of 4 mL of 1% lidocaine plus 1 mL contrast medium (Omnipaque) (Figure 1B). After 5 min, when the temperature in the affected lower extremity had clearly increased, a subcutaneous tunnel was created to fix the catheter. If no side effects occurred 20 min later, a mixture of 20 mL of 1% lidocaine and 20 mg of methylprednisolone was injected, and the catheter was connected to a patient-controlled analgesia (PCA) pump containing 0.4% lidocaine and 1 mg/mL methylprednisolone. The basal dose of PCA was 5 mL/h. The pump connector was immersed in 95% alcohol for 20 min before a replacement pump was connected to the catheter. The duration of CLSNB was 3 wk [4].

Cold pain assessment

All of the patients were assessed for cold pain severity using a visual analog scale (VAS), ranging from 0, indicating no cold or pain, to 10, indicating worst cold or pain. The degree of cold pain was estimated before treatment, on the 1st, 7th, and 14th day during treatment, at discharge, and at 1, 4, 12, and 24 weeks after discharge. Satisfactory cold pain relief was defined as a 50% or more reduction in the VAS score.

Adverse effects and complications

Postural hypotension, diarrhea, or other complications during the procedure were recorded.

Statistical analysis

Statistical analyses were performed using Statistical Packages for Social Sciences 14.0 version (SPSS Inc., Chicago, IL, USA). The clinical course was analyzed via one-way analysis of variance (ANOVA) followed by a Tukey post-hoc test, and further comparison was esti-

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**Table 1.** Characteristics of patients with cold alodynia in lumbar discherniation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Symptomlocation</th>
<th>Symptom duration (years)</th>
<th>Level of disc herniation (MRI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>46</td>
<td>F</td>
<td>Right leg</td>
<td>2</td>
<td>L3-4/L4-5/L5-S1 /</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>M</td>
<td>Left leg</td>
<td>1</td>
<td>L4-5/L5-S1 /</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>M</td>
<td>Right/Left leg</td>
<td>2</td>
<td>L3-4/L4-5 L4-5</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>M</td>
<td>Right/Left leg</td>
<td>3</td>
<td>L4-5/L5-S1 L4-5</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>F</td>
<td>Right leg</td>
<td>0.5</td>
<td>L4-5</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>F</td>
<td>Right leg</td>
<td>5</td>
<td>L2-3/L3-4/L4-5/L5-S1 L4-5</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>F</td>
<td>Right/Left leg</td>
<td>1</td>
<td>L4-5/L5-S1 L5-S1</td>
</tr>
<tr>
<td>8</td>
<td>65</td>
<td>F</td>
<td>Right/Left leg</td>
<td>10</td>
<td>L4-5/L5-S1 L2-3</td>
</tr>
<tr>
<td>Average</td>
<td>52</td>
<td>/</td>
<td>/</td>
<td>3.1</td>
<td>/</td>
</tr>
</tbody>
</table>

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Figure 1. A: CT guided puncture of right lumbar sympathetic trunk (L4). Catheter with steel wire (the arrow) was inserted through needle; B: Contrast medium spread along L4 to L3 and L2 paravertebral sympathetic ganglion.
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The median age and symptom duration of the 8 patients (three men and five women) were 52 and 3.1 years, respectively. Patient characteristics are shown in Table 1. The average VAS score for cold pain declined significantly from 8.0 before treatment to 3.125 at discharge ($P<0.05$), 3.875 at 1 week after discharge ($P<0.05$), 4.5 at 4 weeks ($P<0.05$), 4.62 at 8 weeks ($P<0.05$), and 4.857 at 24 weeks (Figure 2). All patients obtained immediate continuous cold relief during hospitalization. Seven of 8 patients had satisfactory pain relief at 1 week after discharge, 6 of 8 at 4 weeks, 5 of 8 at 8 weeks, 4 of 8 at 12 weeks, and 4 of 8 at 24 weeks (Figure 3).

There was no report of vascular injury or infection. There were no serious complications such as nerve damage resulting in paralysis. No patients reported postural hypotension, diarrhea, or other complications during the procedure. No patient reported worse pain after the procedure. There were no issues with postoperative instability during follow-up, but surveillance flexion-extension X-rays were not performed.

### Discussion

The symptoms of cold alodynia in the lower extremity were clearly improved with CLSNB in patients with LDH in this study. The transient receptor potential (TRP) protein family plays an important role in cold sensation [6, 7]. In particular, the TRP melastatin 8 (TRPM8) and (TRPA1) ion channels are important in cold alodynia [8-11]. Inflammatory reactions and nerve injury can stimulate ion channels and lead to cold pain [12]. Cold alodynia is associated with lumbar sympathetic nerves. It was reported that the lumbar sympathetic nerves could play a role in cold sensation by changing the expression of TRPA1 [13]. The TRPA1 receptor was involved in cold alodynia in a mouse model of sympathetically maintained neuropathic pain [14]. However, it was unclear how this affected the sympathetic nerves. Tang suspected that anterior herniation of the lumbar disc could induce sympathetic inflammation [4].

In our 8 patients, the symptoms of cold alodynia did not improve despite nerve blocks, radiofrequency, or decompression surgery. However,
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anterior herniation of the lumbar disc was found in 7 of 8 patients in this study.

CLSNB was effective in improving the symptoms of sympathetically maintained pain (SMP) through modulation of the function of sympathetic nerves, and therapeutic value was identified in previous studies [4, 5, 15]. Initially, lumbar sympathetic nerve block was able to provide transient relief in SMP by altering the excitation of the sympathetic nerves [16-18], but the therapeutic efficacy returned when the sympathetic block subsided [19]. Lumbar sympathectomy could improve the symptoms of cold hypersensitivity in neuropathic pain [20, 21].

In our study, up to 24 weeks of relief of cold allodynia was seen in patients with LDH after receiving CLSNB with lidocaine and methylprednisolone.

There are limitations in this study. First, the number of patients was small, but preliminary clinical data was provided for further research. Second, a randomized, controlled clinical trial was not conducted due to ethical issues. Third, whether methylprednisolone played an important role in treating cold allodynia in patients with LDH was uncertain. However, there has been no evidence that methylprednisolone could improve the symptoms of cold allodynia in patients with LDH in previous literature.

In conclusion, CLSNB is an effective and safe technique in treating cold allodynia in patients with LDH, and can provide long-term benefit.

Disclosure of conflict of interest

None.

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References


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