ABSTRACT

Chronic neuropathic pain after spinal cord injury is often refractory to conventional treatments. Spinal cord stimulation (SCS) is used to manage intractable chronic neuropathic pain. A 42-year-old man presented with chronic neuropathic pain in his right lower extremity for 5 years. He had undergone posterior fusion from T11 to L3 for a burst fracture of L1 vertebra and conus medullaris syndrome. Conservative treatment with medications, pain blocks, and physical therapy did not relieve the neuropathic pain in the right lower limb. A paddle-type SCS electrode lead was inserted at the level of T9–11 vertebrae for test stimulation. Postoperatively, the patient immediately complained of decreased proprioception, while the motor and sensory neurologic states did not change. Since his neurological deficit did not recover spontaneously, we had to remove the epidural SCS electrode to resolve the neurological symptoms.

Keywords: Spinal cord stimulation; Spinal cord injuries; Chronic pain; Neurologic deficits

INTRODUCTION

Chronic neuropathic pain after traumatic spinal cord injury (SCI) has a high prevalence rate of 53%. SCI patients with neuropathic pain showed reduced quality of life and are frequently refractory to conservative treatments. Spinal cord stimulation (SCS) could be a good alternative treatment for intractable chronic neuropathic pain in SCI. SCS is a reversible therapy with relatively few complications. However, SCS may lead to some complications, such as wound infection, postoperative hematoma, hardware failures, and neurologic deficits. In this report, we present a case of a patient who showed decreased proprioception, known as posterior cord syndrome, immediately after insertion of paddle-type SCS leads.

CASE REPORT

A 42-year-old man was diagnosed with an unstable L1 burst fracture with incomplete SCI after a fall injury and underwent posterior fusion from T11 to L3 5 years ago at another
hospital. After rehabilitation, his neurological status improved and he was able to self-ambulate without assistance. However, he suffered from neuropathic pain and muscle cramps in the right leg after traumatic injury. Intermittent neuropathic pain occurred approximately 50–100 times per day and persisted for 3–5 minutes. His neuropathic pain consisted of the combination of pin-pricking pain on his right leg and muscle cramp. He had received pharmacological treatment with opioids, anticonvulsant agents (pregabalin), and muscle relaxants (baclofen) for 5 years. Pain blocks, such as lumbar epidural block with corticosteroids and local anesthetic agents, were also administered. Since chronic neuropathic pain in the right leg was not relieved with various conventional treatments, he visited numerous hospitals to find an alternative treatment.

When he visited our hospital, the motor grade of the right ankle dorsiflexion was slightly decreased to grade 4. A slight decrease in touch sensation was also observed in the right leg compared to the left leg, whereas cold sensation showed no difference in both lower extremities. Decreased sensation in the perianal areas and urinary and defecation difficulties were also reported by the patient, which were sequelae of SCI at the L1 level, conus-medullaris syndrome.

Under general anesthesia, subtotal laminectomy T11 was performed, and a paddle-type electrode lead (Sppecify™ 5-6-5 Lead; Medtronic, Dublin, Ireland) was inserted into the posterior epidural space (FIGURE 1). The location of the SCS lead was confirmed by a portable fluoroscope. After implantation of the electrode lead, the extension wire was connected to the SCS lead and externalized for test stimulation. After test stimulation, chronic neuropathic pain subsided by more than 80% compared with preoperative pain. However, the patient was unable to engage in self-ambulation without assistance. He complained of difficulty in “maintaining balance while walking” and had to walk with assistance such as a walker or pole. Since motor and sensory examination showed no changes from the preoperative neurologic status, we excluded the possibility of postoperative epidural hematoma. Thus, no further imaging studies, such as magnetic resonance imaging (MRI), were performed.

FIGURE 1. A paddle-type spinal cord stimulation electrode lead (Sppecify™ 5-6-5 Lead; Medtronic) was placed midline at the level of T9–11.
postoperatively. Since the chronic neuropathic pain improved, we observed a clinical course using corticosteroids and rehabilitation. No evidence of wound complications or infections was observed. Despite rehabilitation physical therapy, the difficulty in ambulation did not improve. Therefore, the SCS lead was removed 3 weeks after the initial insertion.

Motor evoked potential (MEP) and somatosensory evoked potential (SSEP) were measured 3 weeks after SCS lead removal. The MEP showed no abnormal findings. The SSEP of the upper extremities showed no abnormal findings, whereas the SSEP of the lower extremities decreased, indicating dysfunction of the somatosensory pathway of the lower extremities (FIGURE 2).

Based on the clinical manifestation and electrophysiological evaluation, the patient had proprioception problems after SCS lead insertion and was diagnosed with posterior cord syndrome. His proprioception sensation recovered approximately 6 months after SCS lead removal.

**DISCUSSION**

Chronic SCI pain can be categorized as nociceptive or neuropathic. The International Association for the Study of Pain (IASP) defines nociceptive pain as pain arising from nociceptor activation. Musculoskeletal nociceptive pain arises from the preserved sensory receptors within the musculoskeletal system, and its pain is characterized as ‘dull’ or ‘aching,’ which responds to anti-inflammatory or opioid medications. Visceral nociceptive pain refers to pain in the thorax, abdomen, or pelvis, as it can be triggered by food intake or functions of the internal organs, such as defecation.\(^1\)

ISAP defines neuropathic pain as pain caused by a lesion or disease in the somatosensory nervous system. It can be classified into at-level SCI neuropathic pain and below-level SCI neuropathic pain. At-level SCI neuropathic pain is defined by its presence anywhere within

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**FIGURE 2.** SEP was examined from both median nerve of upper extremities (A) and both tibial nerve of lower extremities (B). Stimulation from both median nerves produced SSEP, whereas stimulation from both tibial nerves did not produce any SSEP, indicating the dysfunction of somatosensory pathway in both lower extremities.

SSEP: somatosensory evoked potential.
a region spanning one dermatome above and 3 dermatomes below the neurological level of injury (NLI). Below-level SCI neuropathic pain refers to neuropathic pain perceived by more than 3 dermatomes below the dermatome of the NLI. A necessary condition for classifying pain as below-level SCI pain is that a lesion must affect the spinal cord and that the pain is believed to arise as a result of this damage. The characteristics of at-level SCI neuropathic pain include sensory deficits within the pain distribution accompanied by allodynia or hyperalgesia. SCI neuropathic pain is described as ‘pricking,’ ‘hot burning,’ ‘squeezing,’ ‘tingling,’ and ‘electric shock-like.’\(^{1,11}\)

As the mechanisms of SCI pain are poorly understood, it is often refractory to pharmacological treatment. SCS can be another option for SCI patients suffering from intractable SCI pains. Although conventional SCS only has 30%–40% success rate\(^8\) and the clinical efficacy of burst SCS (bursts of 5 pulses with an internal frequency of 500 Hz) is inconclusive,\(^7\) SCS is considered a reversible and safe treatment for chronic neuropathic SCI. Overall complication incidences related to SCS are 30%–40%.\(^5\) The most common complications are related to hardware-related complications, such as lead migration and failure or battery failure, with a rate of 2.1%–27%, followed by wound infection (superficial and deep) at 4%–10%.\(^5\) Complete removal of SCS hardware may be required to resolve infection, especially for deep infection.

Neurologic complications after SCS occur very rarely, with an incidence rate of 0.03%–0.25%\(^4,9\). Levy et al.\(^9\) reviewed the literature on the incidence of neurological damage following surgical paddle lead implantation and subtyped the neurologic deficits into 4 categories: (1) major motor deficit (paralysis, inability to move muscle); (2) limited motor deficit (motor weakness); (3) sensory deficit (numbness, hypoesthesia, or anesthesia); and (4) autonomic changes (bowel and/or bladder dysfunction or sexual dysfunction). Among 44,587 cases, major motor deficits were reported in 111 (0.25%) cases and limited motor deficits in 61 (0.14%) cases. The overall risk of motor deficits after the insertion of the paddle-type SCS lead was 0.39% (172/44,587). Six (0.013%) cases showed autonomic deficits and 46 (0.10%) cases showed sensory deficits. Among those who showed motor deficits, 68 were accompanied by epidural hematoma: 16 cases (0.036% of the sample) had limited motor deficits and 52 (0.12%) had major motor deficits. Of 46 cases (0.1%) of SCS paddle lead patients with complications consisting of sensory deficit only, 21 (46%) recovered completely, 7 (15%) recovered partially, and 18 (39%) had no reported recovery. Ha et al.\(^6\) reported that 2 of 16 patients complained of loss of proprioception at 36 and 6 months after SCS lead insertion. Both showed spinal stenosis at the site of lead insertion, which was resolved by decompression surgery.

Since postoperative hematoma can lead to neurological deficits, the condition of coagulation disorders should be managed. Medications that may lead to coagulopathy, such as antiplatelet agents (e.g., aspirin) or anticoagulant agents (e.g., warfarin), should be discontinued perioperatively. The choice of electrode can be another factor in neurologic deficits. Levy et al.\(^9\) believe that the volume (thickness × width × length) and stiffness of the electrode are related to the risk of neurological complications. A large volume of electrode lead may cause acute blunt trauma during insertion and contribute to spinal cord ischemia by compressing it. The use of intra-operative evoked potentials, especially under general anesthesia, can provide information regarding the integrity of the spinal cord pathways relative to their preoperative status. The intraoperative evoked potential may verify placement of the electrode and detect intraoperative neurologic injury by evaluating spinal pathway integrity.\(^9\)
The patient in this study showed decreased proprioceptive sensation immediately after paddle-type SCS electrode lead insertion. His motor grade and sense of touch and temperature did not change compared to the preoperative neurologic status. In this case, 3 possible mechanisms that lead to postoperative neurological changes were proposed: (1) direct blunt trauma to the spinal cord, (2) postoperative epidural hematoma, and (3) spinal cord ischemia. We did not perform any imaging study postoperatively since the patient’s motor status showed no difference from the preoperative state. Unfortunately, we could not determine the exact reason for the postoperative proprioception deterioration. Spinal stenosis could lead to proprioception deterioration without any motor or sensory problems in the chronic phase,\(^9\) which implies that external compression of the cord by the hematoma could also lead to proprioception. Thus, decreased proprioception without motor and sensory problems caused by a postoperative hematoma is also a possible mechanism. However, we believe that postoperative hematoma is less likely to cause decreased proprioception since his neurological status did not change immediately after postoperatively and no epidural hematoma was observed when the SCS electrode was removed. The existence of cord ischemia was not evaluated because we did not perform postoperative MRI. However, we believe that blunt trauma during insertion is the most likely cause of the posterior cord syndrome.

**CONCLUSION**

Although SCS is a safe and reversible treatment method for SCI patients who are refractory to pharmacological treatment, neurological complications may occur rarely. The most common neurological complication is motor deficit, mainly due to postoperative hematoma. Here, we present a patient who developed posterior cord syndrome immediately after insertion of a paddle-type SCS electrode lead and review the relevant articles.

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