RESEARCH ARTICLE

Effects of pulsed radiofrequency on spasticity in patients with spinal cord injury: a report of two cases

Min Cheol Chang*, **Yun Woo Cho**
Department of Physical Medicine and Rehabilitation, College of Medicine, Yeungnam University, Daemyungdong, Namku, Daegu, Republic of Korea

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Correspondence to:
Min Cheol Chang, M.D., wheel633@hanmail.net.

orcid:
0000-0002-7629-7213 (Min Cheol Chang)

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Abstract

Spasticity following spinal cord injury (SCI) results in functional deterioration and reduced quality of life. Herein, we report two SCI patients who presented with good response to pulsed radiofrequency (PRF) for the management of spasticity in the lower extremities. Patient 1 (a 47-year-old man) had complete thoracic cord injury and showed a phasic spasticity on the extensor of both knees (3–4 beats clonus per every 30 seconds) and tonic spasticity (Modified Ashworth Scale: 3) on both hip adductors. Patient 2 (a 64-year-old man) had incomplete cervical cord injury and showed a right ankle clonus (approximately 20 beats) when he walked. After the application of PRF to both L_2 and L_3 dorsal root ganglion (DRG) (patient 1) and right S1 DRG (patient 2) with 5 Hz and 5 ms pulsed width for 360 seconds at 45V under the C-arm guide, all spasticity disappeared or was reduced. Moreover, the effects of PRF were sustained for approximately 6 months with no side effects. We believe that PRF treatment can be useful for patients with spasticity after SCI.

Key Words: *nerve regeneration; spinal cord injury; spasticity; pulsed radiofrequency; case report; neural regeneration*

Introduction

Spasticity is a release phenomenon in which there is a loss of inhibitory mechanisms following an upper motor neuron injury (Adams et al., 2005) that permits uncontrolled excessive phasic or tonic reflex activity associated with involuntary movements, clonus, and increased muscle tone. Spasticity following spinal cord injury (SCI) occurs in 65–78% of patients (Nielsen et al., 2007), leading to various medical complications, impaired function, and decreased quality of life. Because of its high incidence and close association with patients' function and quality of life (McKinley et al., 1999), an appropriate management of spasticity is important for SCI patients.

Several management methods, including medication, stretching exercise, cryotherapy, nerve block using alcohol or phenol, electrical stimulation, and antispastic orthosis have been applied to manage spasticity following SCI (Kirshblum et al., 2002). When spasticity is poorly controlled, neurosurgical procedures such as selective dorsal rhizotomy, continuous infusion of intrathecal baclofen, and myotomy or tenotomy can be considered (Kirshblum et al., 2002). However, these surgical procedures are invasive and can often lead to unsuccessful outcomes; therefore, they have rarely been used for the management of spasticity. Alternatively, continuous radiofrequency (CRF) lesion of the dorsal root ganglion (DRG) has been applied to patients with intractable spasticity after SCI (Coleman, 1976; Krieger et al., 1982; Herz et al., 1983, 1990; Kasdon et al., 1984; Kenmore, 1997; Reynolds et al., 2014), brain injury (Kasdon et al., 1984; Herz et al., 1990; Kenmore, 1997), or cerebral palsy (Herz et al., 1983, 1990; Vles et al., 1997, 2010; de Louw et al., 2002, 2005). CRF exposes the target nerves to a continuous electrical stimulation and ablates the structures by increasing the temperature around the RF needle tip. The thermal lesion at the unmyelinated fibers of DRG is known to decrease spinal input through deafferentation (de Louw et al., 2002). However, CRF induces a lesion at the nerve fibers, leading to motor deficit, dysesthesia, or neuralgia (Coleman, 1976; Krieger et al., 1982; Herz et al., 1990). A recently introduced pulsed radiofrequency (PRF) incorporates a brief stimulation period followed by a long resting phase (Sluijter et al., 1998), which exposes the target nerves to an electric field without sufficient heat to cause significant structural damage. Several studies have evaluated the pain-control effect of PRF to date (Choi et al., 2012; Park et al., 2012; Schianchi, 2015); however, its effects on the management of spasticity have not yet been reported.

Herein, we report the successful application of PRF to the DRG for the management of spasticity in patients with SCI.

Case Report

Two SCI patients with spasticity were prospectively recruited for this study. All subjects provided informed consent for participation and the study was approved by the Institutional Review Board of ouruniversity hospital (YUH-16-0430-D7).

Patient 1, a 47-year-old man, visited the department of physical medicine and rehabilitation at our university hospital due to spasticity. Twenty-eight years ago, the patient had a compression bursting fracture on the T_8 vertebral body due to a fall, at which time he received spinal interbody fu-

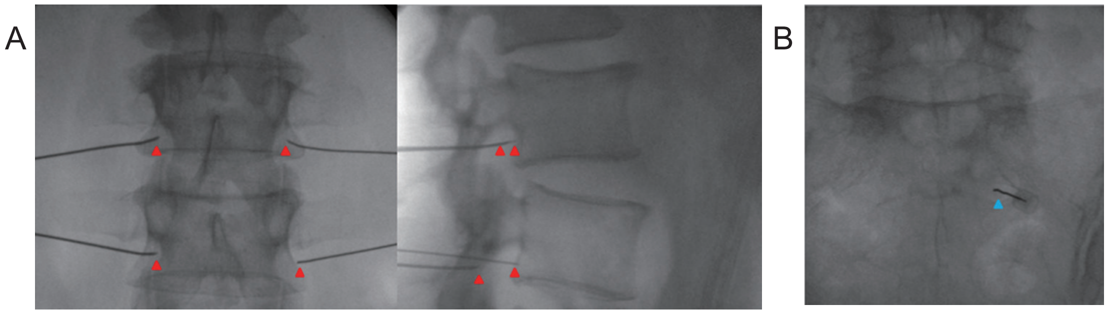

Figure 1 X-ray images of pulsed radiofrequency procedure.
(A) Anterior-posterior and lateral lumbar spine X-ray images showing the placement of needles in the intervertebral foramen $L_{2/3}$ and $L_{3/4}$ (red arrows). (B) Anterior-posterior lumbosacral spine X-ray image showing the placement of needle in the right S_1 foramen (blue arrow).

sion from T_6 to T_{10}. He developed complete paraplegia (the American Spinal Injury Association [ASIA] Impairment Scale: A). From 3 months before the recent visit to our department, the patient had 3 to 4 beats knee extension clonus (phasic spasticity) and spasm (tonic spasticity) of adductor thigh muscles (Modified Ashworth Scale [MAS, Bohannon et al., 1987]: 3) in both of his legs. The clonus was manifested every 30 seconds and initiated without external stimulation. His spasticity was not controlled, although he was taking baclofen three times a day (15–15–15 mg), as well as 2 mg diazepam once a day. Due to the clonus on the knee extensor, he woke up several times a night. We performed the PRF procedure (Cosman G4, Burlington, MA, USA on both L_2 and L_3 DRGs with 5 Hz and 5 ms pulsed width for 360 seconds at 45 V under the C-arm fluoroscopy guide (**Figure 1A**) (Lee et al., 2016; Park et al., 2016). The electrode tip temperature did not exceed 42°C.The patient was laid in a prone position for C-arm fluoroscopy (Siemens, Erlangen, Germany), and an 18-gauge curved tip cannula (SMK pole needle 100 mm with a 10 mm active tip, Cotop International BV, Amsterdam, the Netherlands) was placed around the DRGs. Following PRF, the clonus of both knee extensors disappeared completely, and the spasticity on both hip adductors was reduced from MAS 3 to 1^+. The patient reported that the spasticity on both hip adductors was reduced by approximately 50% and his quality of sleep had dramatically improved. There were no complications after PRF; however, the spasticity returned at nearly the same degree 6 months after the procedure. We repeated the PRF using the same method as for the previous PRF, which led to similar results that were also sustained for 6 months.

Patient 2 was a 64-year-old man who visited our physical medicine and rehabilitation department due to spasticity. Three months before his visit, he had an anterior approach microscopic discectomy on $C_{5–6}$ and $C_{5–6}$ with an interbody fusion and a decompressive total laminectomy on $C_{4–6}$ using aposterior approach and lateral mass screw fixation due to cervical spondylotic myelopathy with spinal stenosis on $C_{4–6}$. For three months before the operation, he had suffered from severe neuropathic pain, hand motor weakness, and gait disturbance. His symptoms occurred spontaneously without any history of trauma. Upon magnetic resonance imaging (MRI) before the operation, spinal stenosis at the level of $C_{4–6}$ and cervical spinal cord compression were manifested. In addition, T2-weighted images revealed high signal intensity in the cervical spinal cord at the $C_{5–6}$ disc space level. After the operation, pain and motor weakness was greatly improved (numeric rating scale [Ferreira-Valente et al., 2011]: 1–2 out of 10, motor power [Jang et al., 2013]: 4^+ out of 5, ASIA Impairment Scale [Bohannon et al., 1987]: D). The patient was able to walk outside independently, and had almost no problems using his upper extremities. However, right ankle clonus manifested 1 month after the operation when he walked, which was triggered when he pushed the ground with his right foot. The clonus was approximately 20 beats. Moreover, the right plantar flexor spasticity showed MAS of 2. His spasticity was not controlled by baclofen three times a day (15–15–15 mg) and 2 mg diazepam once a day. Therefore, under the C-arm guide, we performed the PRF procedure on the right S_1 DRG at 5 Hz and 5 ms pulsed width for 360 seconds at 45 V with the constraint that the electrode tip temperature did not exceed 42°C (**Figure 1B**). The patient was laid in a prone position for C-arm fluoroscopy (Siemens), and an 18-gauge curved tip cannula (SMK pole needle 100 mm with a 10 mm active tip, Cotop International BV) was placed around the right S1 DRG. After the procedure, the ankle clonus or spasticity at his right plantar flexor disappeared and the MAS score was reduced from 2 to 1. In addition, the patient reported no complications after PRF. At 6 months post PRF procedure, the ankle clonus recurred, but the degree of the clonus was not severe (1–2 beats), and it only occurred occasionally. Because the patient only had minor complaints with respect to this occasional clonus, we decided to observe his symptoms without any management.

Discussion

Spasticity following SCI frequently causes joint contractures, pressure sores, and pain, often resulting in functional deterioration and reduced quality of life (McKinley et al., 1999). Therefore, spasticity should be managed appropriately. The most commonly used conventional treatment is oral medication and Botulinum neurotoxin injection; however, the anti-spastic effects of these treatments vary (Kirshblum et al., 2002). Therefore, other non-conventional treatments should be considered for patients who show no positive response to these conventional treatments. Our patient did not show any successful response to medication; however, spasticity was dramatically reduced or disappeared after the PRF procedure on DRG on the lumbar or sacral level ($L_{2/3}$ and S_1).

Because PRF applies a brief electrical stimulation, it does not produce sufficient heat to substantially destroy nerve tissue (Sluijter et al., 1998). The destructive effects of PRF reportedly occur at the microscopic and subcellular levels (Cosman et al., 2005; Erdine et al., 2009). Therefore, we attempted to manage our patient's spasticity using PRF on DRG, and responses to PRF were successful in both cases. In patient 1, the clonus on the knee extensor was phasic spasticity, and spasm on the adductor muscles was tonic spasticity. Patient 2 showed ankle spasticity with a mixed characteristic of phasic and tonic spasticity. Therefore, PRF is thought to be effective atcontrolling both phasic and tonic spasticity.

Although the mechanism by which DRG-PRF manages spasticity has not been elucidated, we suggest the following possible mechanisms. First, DRG-PRF appeared to have enhanced the inhibitory control of spinal reflex anddecreased the excitatory afferent input entering the spinal cord. Several researchers have reported that an electromagnetic field of PRF enhances the various descending inhibitory pathways (Sluijter and Racz, 2002). Moreover, Cosman et al. (2005) reported that PRF using a low-frequency electricalstimulation of the neurons resultedina long-term depression of synaptic transmission.Second, PRF on DRG might have controlled the patients'spasticity by eliminating or reducing the flow of nociceptive information. Noxious stimuli can trigger or aggravate spasticity in patients with SCI (Bang et al., 2015). PRF results in ultrastructural lesions of the sensory nociceptive axons. These lesions are selectively located in the smaller, principal sensory nociceptors (C-fibers, and A-delta fibers), while they are infrequently identified on the larger non-pain related sensory fibers (A-beta fiber) (Erdine et al., 2009).

To date, many studies have investigated the effects of radiofrequency on the management of spasticity in patients with SCI (Coleman, 1976; Krieger et al., 1982; Herz et al., 1983, 1990; Kasdon et al., 1984; Kenmore, 1997; Reynolds et al., 2014), cerebral palsy (Herz et al., 1983, 1990; Vles et al., 1997, 2010; de Louw et al., 2002, 2005), traumatic brain injury (Kasdon et al., 1984; Herz et al., 1990; Kenmore, 1997), and anoxic encephalopathy (Kasdon et al., 1984). However, all of these applied CRF, which uses constant high-frequency electric current, often resulting in neuroablative thermocoagulation on DRG or nerves near DRG (de Louw et al., 2002). By interruption of the afferent pathways of spasticity, CRF is able to reduce the spasticity of patients. However, this procedure is controversial because neuroablation can lead to lasting motor weakness, neuritis, dysesthesia, neuropathic pain, and urinary incontinence (Coleman, 1976; Krieger et al., 1982; Herz et al., 1990). Therefore, instead of CRF, we used PRF to treat our patients, and the results were successful in both cases. However, PRF was limited by the recurrence of spasticity, with the effects diminishing or beginning to diminish after approximately 6 months. These findings are similar to those observed for CRF, which has been shown to induce a limited period of improvement of 6–7 months (Herz et al., 1983; Kasdon et al., 1984; Reynolds et al., 2014). If durations of the effects of CRF and PRF are not significantly different, we believe that PRF is a better choice because it does not cause nerve damage, resulting in fewer complications than CRF. This is the first report to show the effective use of PRF for managing spasticity caused by SCI. However, this study is limited because it is a case study. Accordingly, further studies that involve larger case numbers are warranted. In addition, to achieve the optimal outcomes of PRF, further studies are needed to investigate the stimulation duration, mode, and intensity of PRF. Moreover, an evaluation of the action mechanisms by which spasticity is reduced is necessary.

In conclusion, we report two patients with SCI who showed a good response to PRF on DRG to reduce spasticity. The results of this study showed that PRF is useful for controlling the spasticity after SCI, especially in patients who were unresponsive to anti-spastic medications.

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Declaration of patient consent: *The authors certify that they have obtained all appropriate patient consent forms. In the form the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.*

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