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Spinal Cord Stimulation for Intractable Pain Caused by Sacroiliac Joint Dysfunction: A Case Report

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Abstract

Sacroiliac joint dysfunction is one of the causes of lower back pain, and although it has characteristic pain locations and aggravating factors, it is difficult to diagnose and is often overlooked. A case of relief of typical pain symptoms due to sacroiliac joint dysfunction by spinal cord stimulation is presented. A 60-year-old woman presented with severe chronic pain in the left lumbar, lower buttock, iliac, and groin areas that worsened even when sitting for short periods, as well as numbness in the right lower extremity. The patient had chronic lower back pain since experiencing acute lumbosacral sprains in her 20s and 40s, and her symptoms worsened without any trigger in her 60s. Standard imaging examinations showed no lesions that could be causing the pain, and blood tests showed no inflammation or other abnormalities. Although pharmacological treatment did not provide sufficient analgesia, sacroiliac joint block provided a significant analgesic effect, leading to a definitive diagnosis of sacroiliac joint dysfunction. A spinal cord stimulation trial was performed using percutaneous 8-contact leads placed at the thoracic vertebra 8-11 level, and pain relief was confirmed. One month later, 2 new percutaneous 16-contact leads and an implantable pulse generator were implanted simultaneously. One month after implantation, the visual analog scale and the quick inventory of depression symptomatology scores decreased dramatically from 83 to 8 and from 16 to 4, respectively. In addition, the numbness of the right lower extremity disappeared. These analgesic effects were sustained for 12 months.

Keywords: spinal cord stimulation, neuropathic pain, sacroiliac joint dysfunction, SIJ, SCS

Introduction

The sacroiliac joints (SIJs) are located in the pelvis linking the iliac bones to the sacrum, which facilitates load transfer from the lumbar spine to the lower extremities. Their functions include mobility, stability, and resistance against shear forces.[1)] Disorders of these joints impair the shock-absorbing function and result in characteristic clinical symptoms, a condition known as SIJ dysfunction. The SIJ is increasingly being recognized as a potential cause of lower back or buttock pain, and recent studies have reported that 15-30% of lower back pain cases are due to SIJ dysfunction.[2,3)] However, accurate diagnosis is difficult, and it is often overlooked because SIJ dysfunction is not easily differentiated from other similar conditions, and standard imaging studies fail to provide definitive diagnostic findings. Identifying characteristic pain locations and aggravating factors, such as iliac pain, groin pain, and pain that worsens with sitting, is important in diagnosing SIJ dysfunction.[4,5)] In addition, SIJ dysfunction may be accompanied by symptoms such as numbness or tingling in the legs, which are also important physical findings.[6)]

When conservative treatments do not adequately relieve pain, interventional treatments for SIJ dysfunction may be considered and include radiofrequency ablation of the SIJ, lumbar and sacral radiofrequency neurotomy, and SIJ fu-

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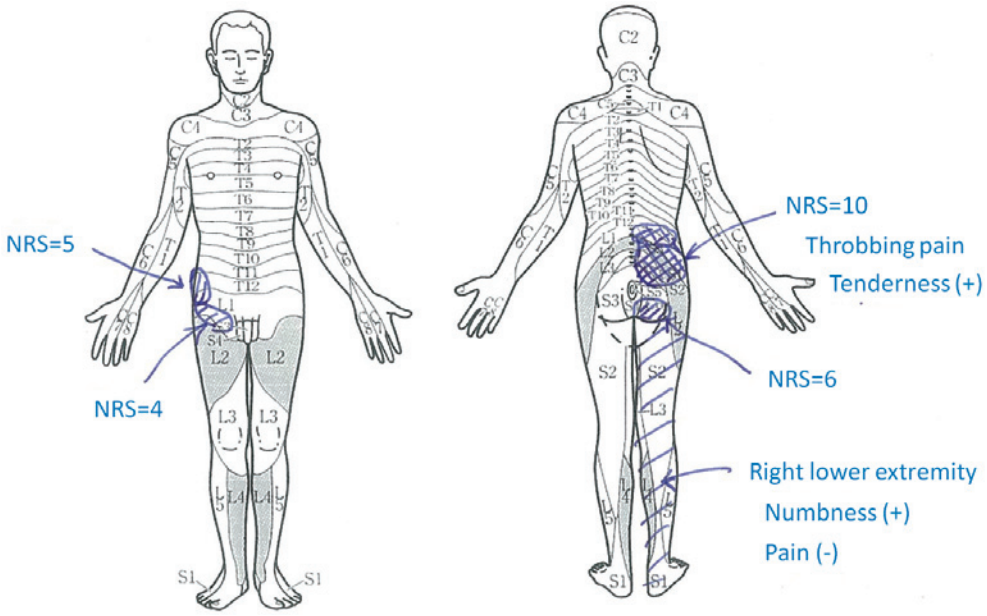


Fig. 1 The schematic diagram recorded in actual clinical practice shows the pain locations.
The numerical rating scale scores for pain in the lumbar, lower buttock, iliac, and groin are 10, 6, 5, and 4, respectively.

sion.[7-10] Spinal cord stimulation (SCS), a neuromodulation therapy that is widely used worldwide for the treatment of chronic pain, is particularly well-known to be effective for chronic lower back pain.[11,12] However, there have been few reports of its use in treating lower back pain due to SIJ dysfunction.[13-15] An instructive case demonstrating the efficacy of SCS for a patient presenting with typical pain symptoms and diagnosed with SIJ dysfunction is reported.

Case Report

A 60-year-old woman presented with severe chronic pain in the left lumbar, lower buttock, iliac, and groin areas. These pain areas were not connected but were separate. A schematic diagram of pain locations recorded in actual clinical practice is shown in Fig. 1. The numerical rating scale (NRS) scores for pain in the lumbar, lower buttock, iliac, and groin areas were 10, 6, 5, and 4, respectively. The pain worsened after about 5 mins of sitting and after about 2 hours of standing upright. She would have liked to work long hours, but the pain had forced her to cut back on her hours. In her 20s, she had her first acute lumbosacral sprain, with a second sprain in her 40s, and she then had lower back pain ever since. In her 60s, the lower back pain worsened without any apparent trigger, and numbness also developed in the posterior region of the right lower extremity (Fig. 1). She visited several hospitals and clinics, but standard imaging examinations such as X-ray, computed tomography, and magnetic resonance imaging showed no obvious lesions that could be causing the pain (Fig. 2A and B). Pelvic computed tomography also showed no obvious abnormalities in the SIJs (Fig. 2C and D). Blood tests showed no signs of inflammation or other abnormalities. Pharmacological treatment with nonsteroidal anti-inflammatories, pregabalin, tramadol, and antidepressants did not provide sufficient pain relief. A sacral block and an L3/4 epidural block each provided pain relief, so the patient was referred by the anesthesiologist to a neurosurgeon for SCS. Before surgical intervention, she was referred to a spinal surgery specialist for examination. On examination by the specialist, the location of pain was characteristic, and imaging and blood tests showed no abnormalities, so SIJ dysfunction was suggested as a possible diagnosis. Therefore, the anesthesiologist was asked to perform a fluoroscopically guided SIJ block, which provided a significant analgesic effect and led to the diagnosis of SIJ dysfunction.

Then, an SCS trial was performed using 2 percutaneous 8-contact leads (Model 977A190; Medtronic Inc.) placed at the Th 8-11 levels, confirming the paresthesia by intraoperative stimulation (Fig. 3A and B). During the SCS trial, 3 stimulation patterns were applied over a 9-day period: tonic, differential target multiplexed (DTM), and fast-acting subperception therapy (FAST).[16] The results of the SCS trial showed that the visual analog scale (VAS) scores before and after each stimulation were as follows: tonic 81 to 26; DTM 85 to 26; and FAST 83 to 21. After the SCS trial was completed, the inserted electrodes were removed, and the patient was discharged. One month later, 2 new percutaneous 16-contact leads (Infinion CX lead; Boston Scientific) and an implantable pulse generator (WaveWriter Alpha 32; Boston Scientific) were implanted simultaneously under general anesthesia referring to the previous X-ray of trial lead placement (Fig. 3C and D). The threshold of par-

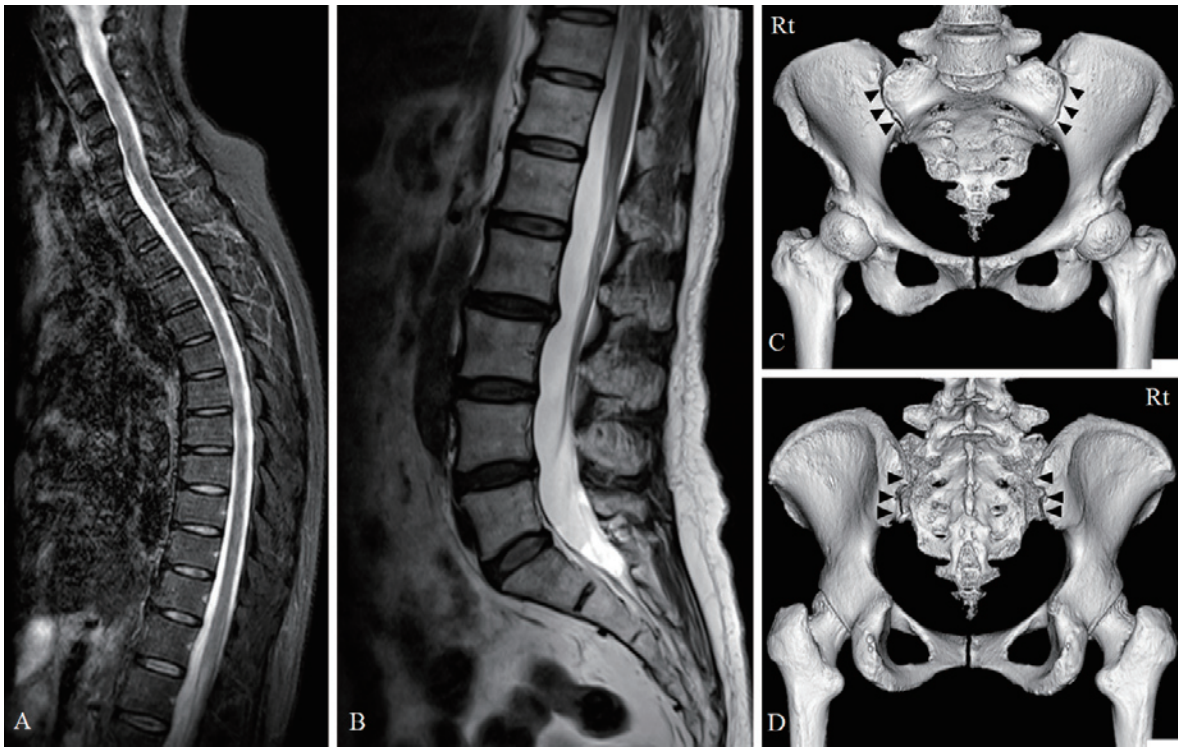


Fig. 2 A, B: Magnetic resonance T2-weighted images of the spinal cord show no obvious lesions causing the pain. C, D: 3D images of pelvic computed tomography show no obvious abnormalities in the sacroiliac joints (arrowheads: sacroiliac joints, C: anterior-posterior view, D: posterior-anterior view).
3D: three-dimensional

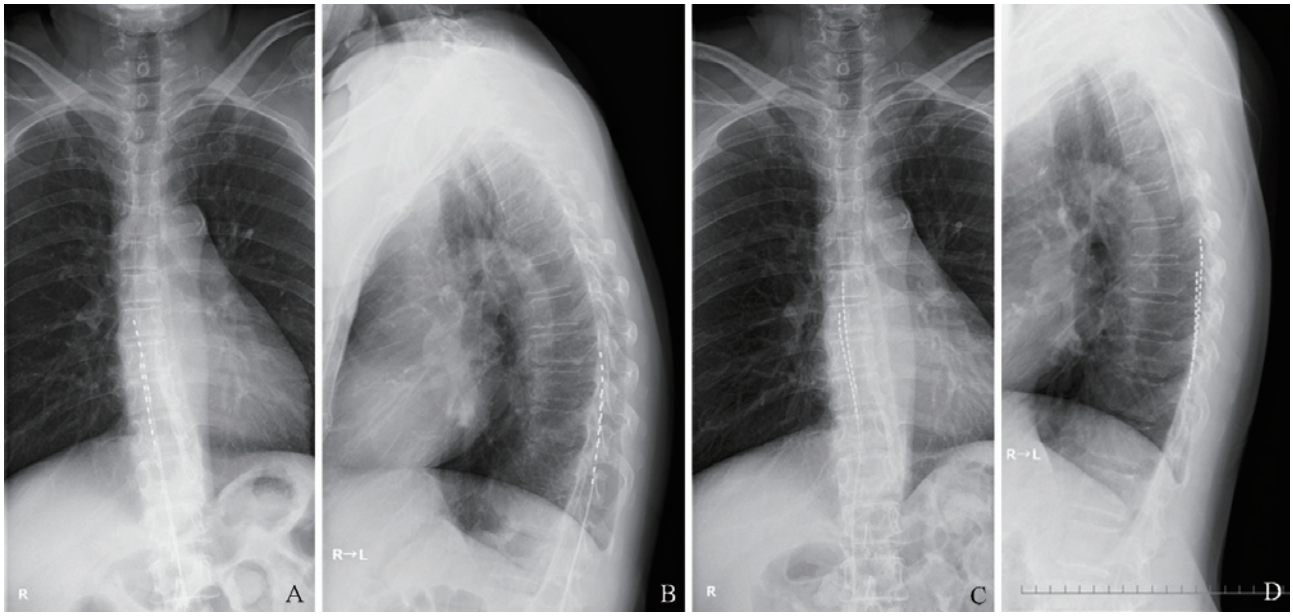
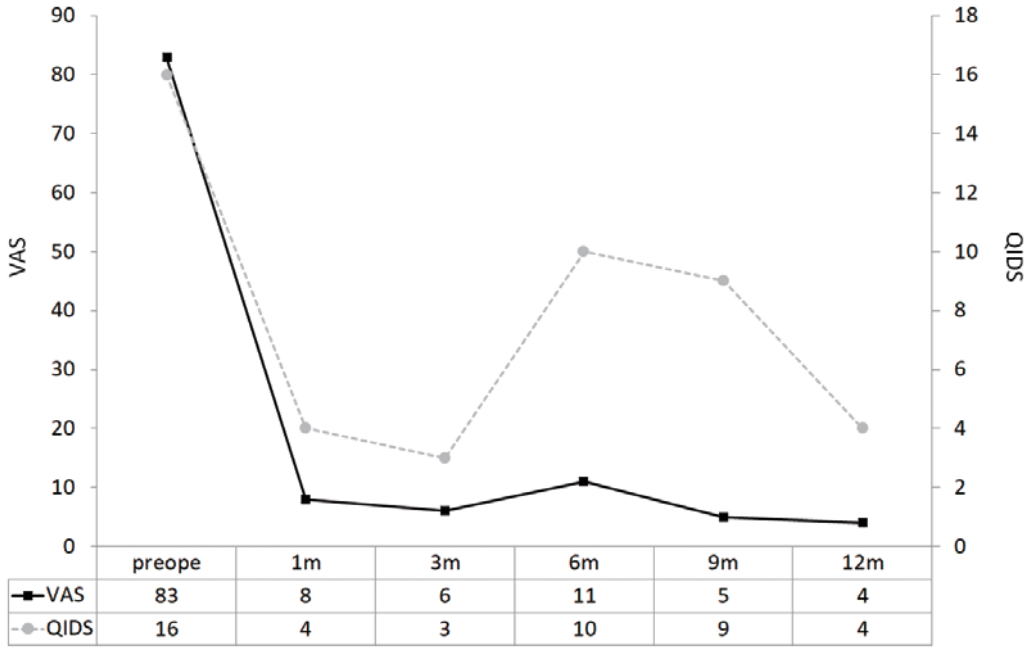


Fig. 3 A, B: During the spinal cord stimulation trial, two percutaneous 8-contact leads are placed at the Th 8-11 levels (A: anterior-posterior view, B: lateral view). C, D: Two new percutaneous 16-contact leads and an implantable pulse generator are implanted simultaneously under general anesthesia (C: anterior-posterior view, D: lateral view).

esthesia using FAST stimulation (frequency 90 Hz, pulse width 210 μs) of the painful area was approximately 3.5 mA. The power setting of FAST stimulation is approximately 30% of the paresthesia threshold, so the actual



	preope	1m	3m	6m	9m	12m
VAS	83	8	6	11	5	4
QIDS	16	4	3	10	9	4

Fig. 4 The line graph shows the changes in the visual analog scale (black line) and the quick inventory of depression symptomatology (gray dotted line) scores after implantation.
m: month, QIDS: quick inventory of depression symptomatology, VAS: visual analog scale

stimulation power was always on at 1.0 mA. One month after implantation, FAST stimulation dramatically reduced the VAS and the quick inventory of depression symptomatology (QIDS) scores from 83 to 8 and from 16 to 4, respectively (Fig. 4). In addition, the numbness in the posterior region of the right lower extremity that was present before implantation had disappeared by this time. The pain relief effects lasted for 12 months and enabled her to work long hours (Fig. 4).

Discussion

The diagnosis and management of SIJ dysfunction remain challenging. One of the main reasons is that imaging examinations do not provide conclusive findings to make a definitive diagnosis of SIJ dysfunction. Standard imaging examinations are used to rule out other etiologies such as spondylolisthesis, hip osteoarthritis, fracture, tumor, inflammation, and degenerative changes. Single-photon emission computed tomography shows increased scintigraphic uptake at the SIJ ligament attachment site in patients with SIJ dysfunction.[17,18] This is one of the useful imaging findings for diagnosing SIJ dysfunction, but it is an auxiliary finding and cannot be used to make a definitive diagnosis. Therefore, in order to include SIJ dysfunction in the differential diagnosis of lower back pain, it is important to recognize the characteristic clinical symptoms such as iliac pain, groin pain, and pain that worsens with sitting. A diagnostic scoring system for SIJ dysfunction has been proposed, in which 6 clinical symptoms are weighted, and a total score is assigned ranging from 0 to 9.[5] A validation study of the scoring system identified 4 characteristic clinical symptoms of SIJ dysfunction: one-finger test results, pain while sitting on a chair, sacroiliac joint shear test results, and tenderness of the posterosuperior iliac spine. When the cut-off value was set at 5 points, sensitivity was 77.4%, and specificity was 76.4%.[19]

If SIJ dysfunction is suspected based on clinical symptoms, diagnostic procedures should be performed to confirm the diagnosis. Intra-articular SIJ blocks are the means of establishing a diagnosis of SIJ dysfunction, with fluoroscopically guided blocks being the gold standard procedure.[20-22] Blocking pain from SIJ is accomplished by injecting 1-2 mL of local anesthetic. If over 75% of the pain is reduced after a single diagnostic block, this test is considered positive. If around 50-75% of the pain is reduced, the SIJ may be considered a major contributor to pain. Because a single block may include false-positive cases, the diagnostic yield can be improved by performing dual blocks, injecting lidocaine in the first block and bupivacaine in the second block.[22] The evidence levels for diagnostic accuracy are Level II for dual diagnostic blocks with at least 70% pain relief and Level III for a single diagnostic block with at least 75% pain relief.[22]

Conservative treatments such as physical therapy, chiropractic care, and medications are the first-line treatments for SIJ fusion. If these treatments do not adequately relieve pain, interventional treatments for SIJ dysfunction are considered and include radiofrequency ablation of the SIJ and lumbar and sacral radiofrequency neurotomy.[7] SIJ fusion is

an effective surgical treatment for SIJ dysfunction with a success rate of 60% to 75%. However, the procedures are invasive and have problems with delayed fusion-related complications.[8-10] The reason why there have been few reports of SCS for SIJ dysfunction is likely because the disease concept and diagnostic methods for SIJ dysfunction have not been established, and the disease has simply not been diagnosed in patients who have undergone SCS. Meanwhile, although it is not the standard use of SCS devices, there are some reports of peripheral nerve/field stimulation targeting peripheral nerves by placing SCS electrodes subcutaneously to treat SIJ dysfunction.[23-25] The hypothesized mechanism of action is that electrical stimulation of the peripheral nerve/field in the painful area modulates cutaneous afferent pain-transmitting nerves and inhibits the transmission of nociceptive information in the dorsal horn of the spinal cord, resulting in an analgesic effect.

FAST stimulation is a paresthesia-free stimulation pattern that delivers 2 symmetrical biphasic waveforms to the leads with a frequency of 90 Hz and pulse width of 210-260 μs. Each wave is a rectangular phase of the charge-balanced stimulation cycle. The stimulation power is lowered to approximately 30% of the paresthesia threshold and stimulation is applied continuously. Therefore, the FAST stimulation settings do not vary significantly depending on the cause of pain. In this case, the changes in QIDS and VAS increased at 6 months and decreased at 9 months, but the increase in QIDS was more significant than the increase in VAS (Fig. 4). This difference may be due to the influence of the questionnaire content. VAS evaluates the intensity of pain at a certain point in time, while QIDS is a psychological evaluation over a certain period of time (the most recent 2 weeks). VAS reflected the intensity of pain at the time the questionnaire was filled out, and it is assumed that there was a period when the pain actually worsened, causing QIDS to increase more than VAS.

It is important to recognize and diagnose SIJ dysfunction as a cause of unexplained lower back pain. As in the present case, when lower back pain continued for years and was accompanied by numbness, the pathology of the pain was likely to include not only nociceptive pain due to SIJ dysfunction but also neuropathic pain components. Neuromodulation therapy is expected to be effective for neuropathic pain, and in the present case, SCS was certainly effective, and the effects were sustained. The pain relief mechanism of peripheral nerve/field stimulation is almost the same as that of SCS. Since SCS is a safe, simple, and common procedure with proper use of SCS devices, evaluating its efficacy for chronic pain due to SIJ dysfunction may be an option.

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Not applicable.

Informed Consent

Informed consent for publication was obtained from the patient.

Disclaimer

Author Ryuta Saito is one of the Editorial Board members of the Journal. This author was not involved in the peer-review or decision-making process for this paper.

Conflicts of Interest Disclosure

There are no conflicts of interest.

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