Spinal Cord Stimulation for Intractable Visceral Pain Due to Sphincter of Oddi Dysfunction

Kang Hun Lee, Sang Eun Lee, Jae Wook Jung, and Sang Yoon Jeon

Sphincter of Oddi dysfunction (SOD) is a syndrome of chronic biliary pain or recurrent pancreatitis due to the functional obstruction of the pancreaticobiliary flow. We report a case of spinal cord stimulation (SCS) for chronic abdominal pain due to SOD. The patient had a history of cholecystectomy and had suffered from chronic right upper quadrant abdominal pain. The patient had been diagnosed as having SOD. The patient was treated with opioid analgesics and nerve blocks, including a splanchnic nerve block. However, two years later, the pain became intractable. We implanted percutaneous SCS at the T5-7 level for this patient. Visual analog scale (VAS) scores for pain and the amount of opioid intake decreased. The patient was tracked for more than six months without significant complications. From our clinical case, SCS is an effective and alternative treatment option for SOD. Further studies and long-term follow-up are necessary to understand the effectiveness and the limitations of SCS on SOD. (Korean J Pain 2015; 28: 57-60)

Key Words: Endoscopic; Intractable; Pain; Sphincter of Oddi dysfunction; Sphincterotomy; Spinal cord stimulation; Splanchnic nerves; Visceral pain.

CASE REPORT

A 58-year-old woman visited our pain clinic for consideration of a splanchnic nerve block. The patient presented had a three year history of right upper quad-

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Correspondence to: Sang Yoon Jeon
Department of Anesthesiology and Pain Medicine, Inje University Haeundae Paik Hospital, 1435, Jwa-dong, Haeundae-gu, Busan, Korea
Tel: +82-51-797-0440, Fax: +82-51-797-0499, E-mail: maestro@paik.ac.kr
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rant abdominal pain. The patient had undergone a laparo-
soscopic cholecystectomy three years earlier. After this
operation, she suffered from chronic recurrent abdominal
pain and underwent repeated multiple abdominal work-
ups and hospitalization. After several trials of
endoscopic retrograde cholangiopancreatography, the
patient was diagnosed as having SOD. Her visual analog
scale (VAS) score for pain intensity was 8–9/10. Pain
relief was not achieved by numerous medications in-
cluding opioids, anticonvulsants, antidepressants, and
others. Medications included 40 mg of oxycodone at ev-
every 8 hrs, a fentanyl citrate patch, 150 mg of tramadol
at every 12 hrs, 200 μg of fentanyl oral tablets at PRN,
1,000 mg of gabapentin at every 8 hrs, and 50 mg of
milnacipran at every 12 hrs. The patient required emer-
gency room visits and hospitalization. Endoscopic
sphincterotomy was recommended for the patient by a
gastroenterologist, but the result of an endoscopic
sphincterotomy was disappointing.

Therefore, a splanchnic nerve block was performed
with local anesthetics and corticosteroid, providing a benefit
for a short duration. This was followed by splanchnic nerve
destruction with 100% dehydrated alcohol, and the patient
was satisfied with the result. The VAS score for pain in-
tensity was reduced to 3–4/10. The procedure was re-
peated three times in two years, but the duration of the
effect was reduced gradually, and the last procedure did
not reduce the pain at all. The patient did not respond to
numerous medications, nor did splanchnic nerve block help.
It was difficult for her to live a normal life due to pain,
and she fell into depression.

We decided to proceed with a trial of spinal cord stim-
ulation which utilized a percutaneous octopolar catheter-
type lead (Octrode lead, Advanced Neuromodulation
Systems, Plano, TX, USA). The trial SCS lead was placed
at the T5–7 level corresponding to the painful area. During
the trial, the patient reported good paresthetic coverage
consistent with her pain site. The VAS score for pain in-
tensity decreased to 2–3/10. After this successful trial, a
permanent percutaneous octopolar catheter-type lead was
placed at the T5–7 level (Fig. 1). Epidural access was at
the T10/T11 intervertebral space by the paramedian
approach. The stimulation parameters were a pulse width
of 450 μs, amplitude of 6.0 mA and a frequency of 60
Hz.

The patient reported significant pain relief with de-
creased VAS scores for pain intensity. The amounts of pain
medications were reduced gradually. Postoperative medi-
cation included 40 mg of oxycodone, 400 mg of gaba-
pentin at every 8 hrs, and 50 mg of milnacipran at every
12 hrs when she finally visited our pain clinic. The pa-
tient was followed-up with for more than six months,
and she demonstrated excellent improvement in pain
without complications. She could therefore return to her
daily life.

**DISCUSSION**

SOD is characterized by symptoms of functional pan-
creatico-biliary obstruction without anatomic abnormality.
Three clinical categories may cause SOD symptoms:
post–cholecystectomy syndrome, acalculous biliary pain
with an intact gallbladder, and recurrent idiopathic pan-
creatitis [2]. The prevalence of SOD in patients after
cholecystectomy is 1%, but it is 14–23% in patients
with post–cholecystectomy syndrome (biliary pain with
elevated liver enzymes) [2]. The Milwaukee classi-

![Fig. 1. Anteroposterior radiograph showing a SCS lead placement at T5-7.](image)

ification classifies SOD according to clinical character-
istics based on elevated liver enzymes, a dilated common
bile duct and the presence of abdominal pain [2]. Type
I SOD patients have abdominal pain, abnormal liver en-
zymes and a dilated common bile duct. Type II SOD pa-
tients have abdominal pain and only one objective sign,
Type III SOD patients have biliary pain only.

A diagnosis of SOD requires a strong suspicion of the disease after ruling out anatomical causes. Manometry of the sphincter of Oddi is the most objective and direct diagnostic method of SOD [3]. However, invasive diagnostic techniques such as manometry have limited availability given the risk of potential complications [2].

Medical treatment of SOD includes smooth muscle relaxants and calcium channel blockers, but the effectiveness of these agents is limited [2]. There are a few reports which found that a medical treatment with trimebutine and nitrates is as effective as surgical treatment [4]. However, thus far the most effective treatment of SOD is endoscopic sphincterotomy [2]. Despite the availability of extensive pharmacologic, physical therapeutic, surgical, and cognitive-behavioral treatments, some patients will develop debilitating, refractory pain. Endoscopic sphincterotomy was also performed for the patient in this study, but she did not experience a decrease in VAS scores for pain intensity.

Visceral pain is described as deep, dull and cramping pain. The pain from a visceral origin is classically described as poorly localized with dull, cramping characteristics. It is diffuse in character and poorly localized. It is typically referred rather than being felt at the source. It is produced by stimuli different from those adequate for the activation of somatic nociceptors. Adequate stimuli for the production of visceral pain include the distention of hollow organs, traction on the mesentery, ischemia, and chemicals typically associated with inflammatory processes [5]. It is associated with emotional and autonomic responses typically greater than those associated with somatic pain [5].

The mechanism of pain relief by SCS remains unclear. A hypothesis that SCS is gating visceral nociception at the spinal level, i.e., the “gate theory” of Melzack and Wall, has been proposed. SCS is promoting the descending inhibitory pathway which acts via the supraspinal mechanism by pretectal nucleus stimulation [6,7]. Another hypothesis includes sympathetic suppression and antidromic activation through neuromodulatory substances [6,7]. Case series which have gained good results about ventral epidural space SCS in patients suffering from visceral pain have been reported, suggesting that there may be another mechanism of action for the effect of SCS [8].

SCS has been applied to patients with intractable pain syndrome as an effective treatment modality and its indications have been expanded. Many studies about SCS as a treatment for neuropathic pain have been reported, and complex regional pain syndrome is the disease most commonly treated with SCS. Recently, case reports of patients successfully treated for visceral pain with SCS have increased. The initial report of SCS for visceral pain published by Ceballos et al. [9] described a 78-year-old man with mesenteric ischemia who was not determined to be a surgical candidate for revascularization. Since the first clinical trial, neuromodulation has been applied to chronic pancreatitis, pelvic visceral pain, angina chest pain, Crohn’s disease, endometriosis and irritable bowel syndrome [10–13].

SCS lead placement varies from C3 to T12, and the “sweet spot” also varies compared with extremity pain, even when the pain is in a similar site [10–13]. Decreased VAS scores for pain intensity, a reduction of opioid consumption, and the recovery of daily life activities were described in previous studies. However, the effectiveness of the SCS treatment is not proven because there is no randomized controlled trial comparing SCS with a conventional treatment. In addition, results from long-term follow-up also have not been reported. SCS as a treatment for visceral pain involves complications, like other etiology cases, such as infection, lead migration, and unpleasant stimulation to extremities [14]. Therefore, SCS is not the first line of treatment for visceral pain, but it is worth applying for chronic intractable patients who do not respond to conventional treatment methods as an alternative treatment option.

After an application of permanent SCS, the VAS score for pain intensity of the patient was reduced to 2–3/10, and the pain was well controlled for six months without significant complications. SCS may provide moderate pain relief with an improved quality of life and less consumption of analgesics for patients with SOD. We report the application of SCS as an alternative treatment option for chronic intractable visceral pain due to SOD.

REFERENCES

1. Corazziari E, Shaffer EA, Hogan WJ, Sherman S, Tooul J. Functional disorders of the biliary tract and pancreas. Gut 1999; 45 Suppl 2: I48–54.
2. Bistritz L, Bain VG. Sphincter of Oddi dysfunction: managing the patient with chronic biliary pain. World J Gastroenterol 2006; 12: 3793–802.
3. Mariani A, Curioni S, Zanello A, Passaretti S, Masci E, Rossi M, et al. Secretin MRCP and endoscopic pancreatic manometry in the evaluation of sphincter of Oddi function: a comparative pilot study in patients with idiopathic recurrent pancreatitis. Gastrointest Endosc 2003; 58: 847–52.

4. Vitton V, Ezzedine S, Gonzalez JM, Gasmi M, Grimaud JC, Barthe M. Medical treatment for sphincter of Oddi dysfunction: can it replace endoscopic sphincterotomy? World J Gastroenterol 2012; 18: 1610–5.

5. Bielefeldt K, Gebhart GF. Chapter 51. Visceral pain: basic mechanisms. In: Wall and melzack’s textbook of pain. 6th ed. Edited by McMahon SB, Koltzenburg M, Tracey I, Turk D. Philadelphia, (PA,), Elsevier/Saunders. 2013, pp 703–17.

6. Scadding JW, Koltenburg M, Chapter 65. Painful peripheral neuropathies. In: Wall and melzack’s textbook of pain. 6th ed. Edited by McMahon SB, Koltenburg M, Tracey I, Turk D. Philadelphia, (PA,), Elsevier/Saunders. 2013, pp 939–50.

7. Prager JP. What does the mechanism of spinal cord stimulation tell us about complex regional pain syndrome? Pain Med 2010; 11: 1278–83.

8. Baranidharan G, Simpson KH, Dhandapani K. Spinal cord stimulation for visceral pain—a novel approach. Neuromodulation 2014; 17: 753–8.

9. Ceballos A, Cabezudo L, Bovaira M, Fenollosa P, Moro B. Spinal cord stimulation: a possible therapeutic alternative for chronic mesenteric ischaemia. Pain 2000; 87: 99–101.

10. Deer TR, Raso LJ. Spinal cord stimulation for refractory angina pectoris and peripheral vascular disease. Pain Physician 2006; 9: 347–52.

11. Kapural L, Cywinski JB, Sparks DA. Spinal cord stimulation for visceral pain from chronic pancreatitis. Neuromodulation 2011; 14: 423–6.

12. Kapural L, Nagem H, Tucek H, Sessler DI. Spinal cord stimulation for chronic visceral abdominal pain. Pain Med 2010; 11: 347–55.

13. Hunter C, Davé N, Diwan S, Deer T. Neuromodulation of pelvic visceral pain: review of the literature and case series of potential novel targets for treatment. Pain Pract 2013; 13: 3–17.

14. Jeon YH. Spinal cord stimulation in pain management: a review. Korean J Pain 2012; 25: 143–50.