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

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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.

Sphincter of Oddi dysfunction (SOD) is a syndrome of chronic biliary pain or recurrent pancreatitis due to the functional obstruction of the pancreaticobiliary flow in the absence of other structural causes [1]. It is most commonly seen in females between the ages of 20 and 50 years and the predominant symptom is localized right upper quadrant abdominal pain [2]. Medical treatment for SOD is ineffective and endoscopic sphincterotomy is recommended in some clinical cases (especially in cases involving elevated sphincter pressure as diagnosed by manometry) [2]. If prior treatment options have failed, the patient will experience repeated hospitalization and a poor quality of life due to intractable abdominal pain. Therefore, we assessed a percutaneous spinal cord stimulator as an alternative treatment method for SOD patients.

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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rant abdominal pain. The patient had undergone a laparoscopic 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 including opioids, anticonvulsants, antidepressants, and others. Medications included 40 mg of oxycodone at 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 emergency 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 intensity was reduced to 3-4/10. The procedure was repeated 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 stimulation 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 intensity 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 decreased VAS scores for pain intensity. The amounts of pain medications were reduced gradually. Postoperative medication included 40 mg of oxycodone, 400 mg of gabapentin at every 8 hrs, and 50 mg of milnacipran at every 12 hrs when she finally visited our pain clinic. The patient 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.

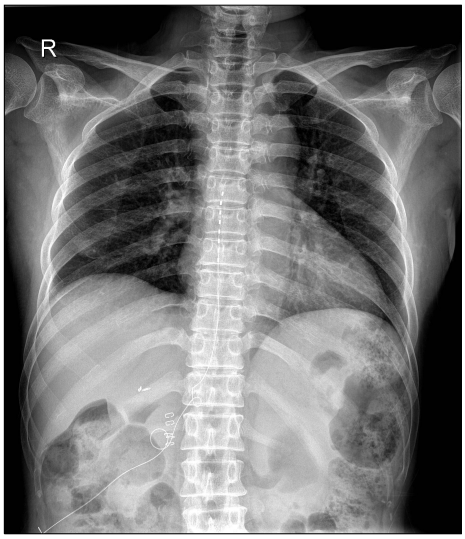

Fig. 1. Anteroposterior radiograph showing a SCS lead placement at T5-7.

DISCUSSION

SOD is characterized by symptoms of functional pancreaticobiliary obstruction without anatomic abnormality. Three clinical categories may cause SOD symptoms: post-cholecystectomy syndrome, acalculous biliary pain with an intact gallbladder, and recurrent idiopathic pancreatitis [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 classification classifies SOD according to clinical characteristics 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 enzymes and a dilated common bile duct. Type II SOD patients 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.

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