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Spinal cord stimulator implantation with immediate post-operative paraplegia: Case report

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ABSTRACT

The most common complication of dorsal column spinal cord stimulator implantation is hardware migration. Spinal cord injury following paddle or percutaneous lead implant is rarely reported, with an overall incidence of 0.42%. This report describes a case of immediate post-operative incomplete paraplegia following implantation of one thoracic paddle electrode to address post-laminectomy pain syndrome. Despite emergent removal of the electrode, post-operative corticosteroids, and a course of inpatient rehabilitation, the patient discharged with persistent incomplete paraplegia. Although there is rare occurrence of spinal cord injury with spinal cord neuromodulation, it is important to recognize risk factors which may lead to similar devastating complications.

1. Introduction

An estimated 50,000 spinal cord stimulators are implanted annually in the United States [1]. The most common indications for spinal cord stimulation (SCS) include post-laminectomy pain syndrome, complex regional pain syndrome, painful neuropathy, and ischemic limb pain [3]. Spinal stimulation may be accomplished via percutaneously inserted electrodes without disruption of osseous structures. However, the insertion and placement of paddle leads into the epidural space requires open surgery (e.g., laminotomy or partial laminectomy) [2,3]. The paddle lead is suitable for patients with a history of lead migration or challenges with placement of the trial lead. Ultimately, the decision for percutaneous versus paddle electrode implantation is based on clinician and patient discussion with various technical considerations and preferences. [4].

Spinal cord stimulator trial and implantation complications are generally minor. The most common complication was found to be hardware issues, including electrode migration [4]. The literature review by Turner et al. states the following incidences of complication: additional revision (23.1%), hardware malfunction (10.2%), infection (4.6%), biological complications other than infection or local pain (2.5%), pain at the pulse generator site (5.8%), and stimulator removal (11.0%) [5]. Although uncommon, there are reports in the literature of SCS implant resulting in significant neurologic damage. A retrospective study found that of 71,172 patients who underwent SCS implant surgeries, 0.42% developed postoperative spinal cord injuries (SCI) within 45 days [6]. Neurological damage is typically a result of infection or intra-operative spinal root injury [7]. In this case, we describe immediate post-operative paraplegia following thoracic paddle electrode placement.

2. Case presentation

A 70-year-old male with history of obesity, L2-S1 lumbar fusion (without history of revision surgery) presented with progressive, persistent axial low back pain and bilateral lower extremity radiculopathy. Previous medical conditions included insulin-dependent diabetes mellitus, hypertension, and hypothyroidism. Imaging revealed multilevel, moderate thoracic spinal stenosis most severe at T11-12. He underwent a trial with a thoracic percutaneous stimulator electrode and reported 90% improvement in pain and substantial functional gain. Prior to permanent implantation, patient had adequate strength against resistance of all extremities and was ambulatory without the need for assistive devices.

Placement of one paddle electrode was performed with a T11 laminectomy under general anesthesia and without intra-operative neuromonitoring. Intra-operative positioning with electrodes at the superior endplate of T9 was confirmed with fluoroscopy. Once alert in the post-anesthesia care unit, the patient noted significant weakness and minimal sensation throughout the bilateral lower extremities. Strength exam

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was noted to be 0/5 of hip flexors, knee extension and dorsiflexion and plantar flexion bilaterally. Therefore, the patient underwent emergent surgical removal of the stimulator system, including the paddle lead and the pulse generator, via the prior incision site. There was no sign of epidural hematoma.

Following completion of the second surgery for SCS removal, thoracic spine MRI was completed which showed T2 hyperintensity in the cord from approximately T9-10 intervertebral disc to T11-12 level. On T9-10 and T11-12 level, there was disc osteophyte complex, facet hypertrophy, and ligamentum flavum thickening. Moderate canal stenosis was noted on T8-T9 level without cord impingement. Patient was treated with dexamethasone for two days with slight improvement in sensory deficits. However, his pre-operative back pain with radiation into the bilateral extremities persisted with the new addition of severe bilateral lower extremity weakness. Hospital course was further complicated by neurogenic bladder and bowel. He was classified with American Spinal Injury Association Impairment Scale C incomplete paraplegia. He was discharged to inpatient rehabilitation for participation in spinal cord injury-focused treatment and subsequently to a skilled nursing facility for prolonged rehabilitation and nursing care with minimal improvement in lower extremity neurological function with continued neurogenic bladder and bowel symptoms.

3. Discussion

While most complications from spinal cord neuromodulation are rare, the most common complications are hardware malfunction and infection. More serious complications are rare, and may include spinal nerve root and spinal cord injuries [7]. More serious and potentially devastating complications such as neurological injury can occur. Neurological injury within 45 days of SCS implantation occurs in 0.42% of cases, according to a recently updated retrospective study focusing on mechanical causes of SCI [6]. Furthermore, development of epidural hematomas after SCS implantation may result in neurological deficits [8,9]. Neurological injuries post-procedure can present from a range of symptoms including loss of vibration and position sense, paresthesias, bowel and/or bladder incontinence, and paraplegia [8–12]. Smith et al. reported a case series of four patients who suffered spinal cord injuries after SCS implantation; two were caused by epidural hematomas, one from spinal cord contusion, and one with progressive spinal cord compression.

There are other spinal cord injury complications after SCS implantation that have been previously documented. Wang et al. reported an 82-year-old patient with history of lumbar radiculopathy and spinal stenosis who underwent L1-L2 SCS implantation with percutaneous lead placement at T8 which was complicated by inability to stand or ambulate without assistive devices. The leads were removed, and imaging revealed T12-L1 stenosis in association with a new annular tear. In this case, it was theorized that the tear occurred after removal of the previous trial leads. Despite immediate complications, her symptoms progressively improved after 7 days [12].

Smith et al. reported a female patient with history of two previous lumbar fusion surgeries who underwent a SCS implant at the T8-T9 level to address persistent, severe pain. However, she experienced progressive numbness and weakness in bilateral lower extremities. MRI thoracic spine revealed new thoracic disc herniation, spinal cord edema, and severe thoracic spinal stenosis with multi-level calcified disc herniations. She underwent a thoracotomy and multi-level discectomy followed by arthrodesis. Post-operatively she continued to have uncontrolled pain and lower extremity weakness requiring the need for wheelchair and rolling walker for locomotion and ambulation [8].

Delayed spinal cord compression as a result of excessive fibrosis after paddle lead placement have also been reported [15]. While epidural fibrosis is an inflammatory reaction that is usually found in all cases where electrodes are present in the epidural space, this reaction is usually mild and does not result in significant cord compression and neurologic complications [6]. However, severe fibrosis as a result of lead placement is a possible complication which might contribute to or cause cord compression.

Our case highlights a rare and important example of severe and acute neurological complication immediately following spinal cord stimulator implantation. This is thought to be secondary to mechanical spinal cord compression from electrode lead mass effect. It is important to reiterate that he had undergone thoracic MRI a few months prior to permanent spinal cord stimulator implant which showed evidence of multilevel thoracic spinal canal stenosis, most severely at the level of T11-L1. This MRI was obtained after a successful percutaneous lead trial period without neurological complication.

Our patient also had a thoracic MRI after SCS removal which did not reveal new intervertebral disc pathology or acutely progressive spinal canal stenosis. Furthermore, during the SCS removal, no epidural hematoma was found. Therefore, the two key contributing factors leading to the spinal cord injury are likely the preexisting thoracic spinal canal stenosis and the decision for implantation of the paddle electrode.

Choi et al. examined the position of post-operative paddle leads within the T9 posterior spinal canal using CT in SCS implant patients which found that paddle leads were inclined to slide even after use of fluoroscopic guidance, neurophysiological modeling, and appropriate placement. This sliding contributed to an overall reduction in the T9 spinal canal space [13,14]. Al Tamimi et al. used three dimensional myelographic CT scans before and after placement of paddle leads at T9 and found that the cross sectional areas of the thecal sac and spinal cord under each contact lead was significantly decreased after placement, resulting in deformation of the spinal cord at the location under the leads [15,16]. SCS lead placement takes place close to the dorsal column, and results in some deformation of the spinal cord even with proper placement. This mechanical phenomenon may be a significant cause of cord compression leading to neurological complications as observed in this patient's case.

Due to thicker paddle electrodes in comparison to percutaneous leads, there may be an assumed increased risk of SCI with paddles compared to percutaneous leads. However, a retrospective study comparing the incidence of spinal cord injury after percutaneous and paddle lead implants did not find a significant difference between the two [6]. The limitation to this study, however, was that the incidence of SCI between the two lead types were not focused on patients with history of thoracic spinal stenosis, which would have better assessed the risk between lead types.

The next question we must address is whether thoracic stenosis increases the risk of neurological injury for those undergoing SCS lead placement. Chan et al. conducted a retrospective study which found that having a diagnosis of cervical or thoracic spinal canal stenosis within 1 year of SCS implantation significantly increased the risk of developing a spinal cord injury [17]. The elevated rates of SCI in these patients is likely due to increased narrowed cross-sectional area surrounding the spinal cord, which increases the risk of nerve compression, and ultimately peri-operative SCI [18].

4. Conclusion

This noteworthy case emphasizes the potentially devastating risk of SCI with SCS implantation in patients with history of moderate-to-severe thoracic stenosis. The risk of neurological sequelae may be further increased with paddle leads in patients with narrow epidural cross-sectional area. While SCS implantation is often safe and effective for pain management, possible severe neurologic complications such as cord compression should be addressed with patients prior to surgery. Extra precautions with attention placed on patient selection should be taken for risk mitigation prior to undergoing implantation.

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Declaration of competing interest

The authors declare that they have no known conflicts of financial interests or personal relationships to reveal that could have appeared to influence the work reported in this paper.

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