PAIN MEDICINE CASE REPORTS

ISSN 2768-5152

Volume 10, Number 6, pp. 535-537

Seizure Provocation Following Low-Energy Burst Dorsal Root Spinal Cord Stimulator Implant in Patient with Chronic Back Pain and Pre-Existing Seizures: Case Report

Josephine Nwankwo, MD[1], Angela Nwankwo, MD[1], Renato Martin Jr., DO[1], Ovie Enaohwo, MD[2], Yacoub Khatab, MD[1], and Emanuel Husu, MD[1]

Background: Spinal cord stimulation (SCS) is an established treatment of chronic pain refractory to conservative management. While it is thought to work through neuromodulation of spinal cord pathways, its impact on central nervous system excitability is not well understood. There are afew reports highlighting seizures temporally associated with SCS.

Case Report: This case involves a 78-year-old man with chronic low back pain secondary to lumbar facet arthropathy and a history of seizure disorder. He underwent spinal cord stimulator implantation after failing conservative management for back pain. The implant provided significant pain relief and functional improvement. However, within 9 months of implantation, he developed recurrent seizures. Seizure activity ceased after removal of the SCS, but unfortunately, his pain returned.

Conclusions: This case highlights the unusual and poorly understood complication of SCS-induced seizures, emphasizing the need for caution when considering neuromodulation therapy in patients with a known seizure history.

Key words: Seizure disorder, spinal cord stimulator, chronic low back pain, epilepsy, case report

BACKGROUND

Spinal cord stimulation (SCS) has become a common intervention for chronic pain syndromes that do not respond to conservative measures, including failed back surgery syndrome, radiculopathy, and facet arthropathy. In these populations, patients were found to have 50% or greater pain relief and satisfaction with the SCS (1). Placement of a SCS involves the implantation of leads in the epidural space, which send pulsed electrical energy to the spinal cord to help relieve pain (2). The primary mechanism of action involves neuromodulation to inhibit pain signaling pathways, yet the influence of SCS on central nervous system excitability is complex and not fully understood (3). SCS also activates the thalamus, a potential component of seizure generation, which may contribute to seizure susceptibility (3). In an animal model study examining the effect of SCS frequency on seizure susceptibility, it was found that higher SCS frequencies resulted in a decrease in seizure susceptibility. Several mechanisms have been proposed to explain how SCS may induce seizures. One mechanism is that neuromodulation alters spinal cord pathways and potentially impacts cortical and subcortical excitability, particularly in individuals with a lower seizure threshold

From: [1]H. Ben Taub Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX; [2]Department of Physical Medicine & Rehabilitation, McGovern Medical School at The University of Texas Health Science Center at Houston, Houston, TX

Corresponding Author: Josephine Nwankwo, MD, E-mail: josephine.nwankwo@gmail.com
Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.
Patient consent for publication: Consent obtained directly from patient(s).
This case report adheres to CARE Guidelines and the CARE Checklist has been provided to the journal editor.
Accepted: 2026-03-05, Published: 2026-08-31

(4). Reports linking SCS placement for chronic pain and seizures are extremely limited. Here we present a case of a 78-year-old man with a history of chronic low back pain and seizure disorder who developed recurrent seizures after spinal cord stimulator placement.

CASE REPORT

The patient is a 78-year-old man who presented with complaints of chronic low back pain that began in 2018 after a fall. The pain was described as burning and stabbing, predominantly in the right paraspinal region, and exacerbated by walking and standing. It was partially relieved by sitting and medications, including nonsteroidal anti-inflammatory drugs, acetaminophen, and topical cannabidiol. The patient reported a surgical history of an L2-L3 laminectomy in 2010 and an L4-S1 laminectomy in 2019 without significant improvement in pain. The patient tried physical therapy and lumbar facet injections with only short-term relief, and other conservative treatments were similarly ineffective. Neurosurgery had stated that surgical interventions were not indicated, as there was no clear surgical target. The patient was then referred to physical medicine and rehabilitation pain management specialists for further evaluation and treatment.

The patient's medical history was notable for a seizure disorder since adolescence, characterized by generalized tonic-clonic seizures, controlled for many years on antiepileptic medication. His neurological examination showed no focal deficits, with intact lower limb strength and sensation. A magnetic resonance imaging of the lumbar spine revealed mild to moderate bilateral degenerative foraminal stenosis and facet arthropathy, consistent with lumbar spondylosis but without significant central canal stenosis.

Due to persistent pain and poor response to conservative measures, the patient was offered a SCS trial. The trial resulted in complete pain relief, allowing improved mobility and daily functioning, which led to the decision to proceed with permanent SCS implantation. The patient experienced a significant reduction in pain but began to have recurrent seizures within 9 months following SCS implantation. The implanted device was programmed in burst stimulation mode. These episodes, characterized by loss of consciousness, tongue-biting, drooling, and post-ictal confusion, were notably different from the patient's prior well-controlled seizures, the last of which occurred more than 30 years prior to his recent SCS implantation. The seizures were temporarily associated with periods of increased SCS stimulation and occurred mostly at night while the patient was resting in a recliner.

The patient underwent multiple evaluations in the emergency department following seizure episodes, including head computed tomography scans, which showed no acute abnormalities. Neurology consultation and electroencephalography were unremarkable. During evaluation of the recurrent seizures, programming adjustments to the device were trialed, but despite these changes, seizure activity persisted, and occurred even after adjustments to the manufacturer's highest stimulation settings. To assess the relationship between SCS implantation and seizure activity, the device was temporarily turned off, resulting in the complete cessation of seizures over several months. However, disabling pain returned, significantly impacting the patient's quality of life. Consequently, the SCS was eventually explanted, and the patient remained seizure-free thereafter.

DISCUSSION

This case illustrates an unusual scenario where SCS therapy, although effective for pain control, may have contributed to the recurrence of seizures in a patient with a remote history of epilepsy. While SCS provided significant pain relief, the cessation of seizures after its removal supports a possible association. The mechanism by which the SCS contributes to increased seizure frequency remains unclear. SCS has been linked to an inhibition of cortical excitability and activation of thalamic structures, which may interfere with seizures (4).

While the exact pathophysiology remains speculative, the relationship between stimulation parameters and seizure activity suggests that certain patients may be predisposed to this complication. In this patient, his seizures recurred during periods of high stimulation, which may suggest that programming features like frequency, variations in pulse width, or improper lead placement closer to central neural structures may increase the risk of aberrant neuronal firing (1,3). Moreover, the patient's remote history of seizure disorder and the temporal association with SCS implantation raised suspicion that stimulation may have exacerbated his condition.

It is important to note that seizure activity ceased once the device was turned off, suggesting a possible causal link between SCS activity and seizure provocation. However, the neurologic workup and literature review revealed that such an association is not well-established. Neurologists evaluating this case indicated that the

patient's seizures could represent a natural progression of his epilepsy, which had been quiescent for several years. Nevertheless, this case adds to the body of scant evidence that SCS could pose an unrecognized risk for patients with a history of seizures.

The management of this case highlights the need for a multidisciplinary approach involving neurology, pain management, and spine specialists, particularly when balancing seizure risk against pain control. For patients with a history of seizures who are candidates for SCS, conservative programming, careful titration of parameters, and close monitoring of neurologic symptoms may be advised. Additionally, thorough patient counseling about the risks of seizure exacerbation is critical.

This report adds to the limited literature suggesting that SCS placement may alter seizure threshold in patients with an existing seizure history. Further studies are warranted to better understand the potential neurologic side effects of SCS, identify risk factors in those susceptible, and develop guidelines for managing patients with seizure disorders who may benefit from SCS placement.

CONCLUSIONS

This case describes seizure exacerbation following SCS implantation in a patient with well-controlled preexisting epilepsy. The temporal relationship between SCS activity and seizure recurrence suggests a possible, although poorly understood, association between neuromodulation and seizure induction. This case underscores the need for careful patient selection and comprehensive risk assessment before considering SCS therapy in those with neurologic comorbidities. The lessons learned from this case suggest that conservative device programming and close interdisciplinary follow-up are essential for optimizing outcomes. Future research is needed to establish best practices for SCS use in seizure-prone populations and to further elucidate the mechanisms by which SCS may provoke or exacerbate seizures.

REFERENCES

1. Sdrulla AD, Guan Y, Raja SN. Spinal cord stimulation: Clinical efficacy and potential mechanisms. *Pain Pract* 2018; 18:1048-1067.
2. Garcia K, Wray JK, Kumar S. *Spinal cord stimulation*. [Updated 2023 Apr 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: www.ncbi.nlm.nih.gov/books/NBK553154/
3. Harreby KR, Sevcencu C, Struijk JJ. The effect of spinal cord stimulation on seizure susceptibility in rats. *Neuromodulation* 2011; 14:111-116.
4. Jiao J, Jensen W, Harreby KR, Sevcencu C. The effect of spinal cord stimulation on epileptic seizures. *Neuromodulation* 2016; 19:154-160.