High-frequency spinal cord stimulation causing cardiac paresthesias after lead migration: a case report

British Journal of Pain
2018, Vol 12(4) 217–219
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DOI: 10.1177/2049463718762970
journals.sagepub.com/home/bjp

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Abstract

Introduction: High-frequency (HF) spinal cord stimulation (SCS), a relatively new form of spinal cord stimulation, provides stimulation frequencies of up to 10 kHz and allows for paresthesia-free pain relief, an advantage that distinguishes it from traditional stimulation therapy. Without paresthesias, patients with HF SCS do not experience position-dependent painful stimulation and do not have to experience treatment interruption during sleep. Lead migration is a well-known complication of conventional spinal cord stimulation and usually results in a loss of efficacy along with other unpleasant sensory symptoms. In this case report, we present an incidence of lead migration in HF SCS that resulted in paresthesias, a symptom not expected to occur in this novel therapy.

Case: The patient, a 60-year-old female with post-laminectomy syndrome, underwent a trial of HF SCS with standard lead placement at T8-T9. She initially had pain relief, but returned to the office on post-operative day 2 complaining of left chest wall and cardiac paresthesias, without frank pain or palpitations, in addition to loss of efficacy for her back and leg pain. Imaging showed that the leads had migrated, with one lead reaching the levels of T1-T3.

Conclusion: While HF SCS has emerged as an effective paresthesia-free means of reducing back and leg pain, we provide the first report of paresthesias occurring with the HF SCS system as a result of cephalad lead migration. As HF SCS is only now being utilized as a treatment modality, we must remain cautious of potential adverse outcomes in patients, in particular above T8.

Keywords

Paresthesias, HF SCS, post-laminectomy syndrome, lead migration, FBSS, complex regional pain syndrome

Introduction

The application of neuromodulation to treat pain has grown exponentially since its advent almost 50 years ago. Over the years, the indications for spinal cord stimulation (SCS) implantation have broadened in order to treat a variety of pathologies that cause chronic pain, with failed back surgery syndrome (FBSS) and complex regional pain syndrome being the most common. While SCS has been proven to be effective, many patients describe the SCS-induced paresthesias as particularly uncomfortable.

A relatively new modality, high-frequency (HF) SCS (Senza SCS System, Nevro® Corp.) provides

stimulation frequencies of up to 10 kHz, which allows for paresthesia-free pain relief, an advantage that distinguishes it from traditional stimulation therapy.

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Without paresthesias, patients with HF SCS do not experience position-dependent painful stimulation.^{1,2} Furthermore, studies have indicated that HF SCS provides significant back and leg pain relief.¹

The complications and adverse events of HF SCS are similar to those of traditional SCS and include pocket pain, wound infection, lead migration, loss of therapy effect and skin erosion.^{2,3} While numerous case reports exist regarding conventional SCS and adverse events, few exist regarding HF SCS complications as it is a relatively recent treatment method. Lead migration is a well-known complication of SCS placement and commonly results in loss of adequate pain relief along with other sensory disturbances. We describe the first published report, to our knowledge, of the paresthesia-free HF SCS system causing paresthesias after lead migration.

Case

The patient described in this case report is a 60-year-old female with a history of L3-S1 spinal fusion in 2011, who subsequently developed adjacent segment disease. In 2015, she underwent direct lateral interbody fusion at L2-L3, which unfortunately resulted in persistence of burning, dysesthetic, low back and bilateral leg pain, consistent with neuropathic pain from FBSS. Subsequent failure of conservative management, which included physical therapy and epidural steroid injections, along with her complaints of significant low back pain made her an excellent candidate for a trial of HF SCS using the Nevro[®] system (Senza SCS System, Nevro[®] Corp.).

In September 2015, the patient was brought electively to the operating room for a trial of stimulation. The Nevro[®] 1 × 8 arrays were placed under fluoroscopic guidance, in the standard fashion: one lead was placed at the top of T8, while the other at mid aspect of T9. Lateral fluoroscopic imaging was performed to confirm proper dorsal epidural placement. The needles and guidewires were removed under live fluoroscopy, and the leads were secured with 2-0 silk sutures to the patient's skin. The leads were dressed with cotton gauze and bio-occlusive adhesive dressing. Initially, the patient reported improvement in her pain and denied any paresthesias.

Despite the uncomplicated procedure, the patient presented on post-op day 2 with complaints of recurrence of her low back and leg pain, which is not unexpected with HF SCS as there is a 'wash in' effect that may delay the onset of pain relief a couple days after implantation. Furthermore, she complained of marked numbness and paresthesias in her 'heart', left anterior chest wall and left upper extremity regions primarily in the T1-T3 dermatomes. Along with basic laboratory testing which included cardiac enzymes, imaging studies were ordered and an approximately 15.0-cm rostral

lead migration was observed. Although T1-T3 paresthesia would be a highly unlikely presentation for acute coronary syndrome, serial cardiac enzymes were ordered and were within normal limits. The electrocardiogram (ECG) also was unremarkable. Imaging demonstrated one of the leads to be at the T1-T3 vertebral levels. Immediately, the stimulation was stopped, and the patient no longer reported the uncomfortable chest paresthesias. The leads were then subsequently repositioned caudally to the appropriate location, but overall pain relief remained poor most likely due to the 'wash in' effect. The patient subsequently elected to have the leads permanently removed at that time. They opted to follow-up with pain management, and no further neurosurgical intervention was indicated.

Discussion

Lead migration was the primary diagnosis in the differential for the cause of this patient's symptoms, but it was necessary to rule out possible cardiac causes as she complained of left-sided chest and upper extremity discomfort. The HF SCS has been marketed as a paresthesia-free system, which, in addition to the system's benefits in relieving lower back pain, is seen as a major benefit to patients' quality of life. While HF SCS has been studied at vertebral levels and shown to be paresthesia free at vertebral levels at T8 and below, the safety of programming above 1200 Hz has not been studied at vertebral levels above T8;³ levels which may become stimulated following a migration adverse event. Although lead migration has been included as a complication of HF SCS implantation, the symptoms following migration have not been published.

Considering that routine and uncomplicated HF SCS therapy does not produce paresthesia symptoms, the adverse process of lead migration should be examined to explain how paresthesias could develop in such a setting. One potential explanation for the transition from paresthesia-free coverage to the patient's symptoms involves the distance between the electrodes and the target for stimulation. There is an anatomically normal relative thickening of the dorsal cerebrospinal fluid (CSF) layer from T2 to T12, peaking at T5-T8, which increases the distance between the electrodes and the dorsal columns.⁴ The effects of electrode to dorsal column distance are evident when the patient experiences uncomfortable symptoms with changes in position. The low resistivity of CSF in combination with the changes in thickness of fluid results in postural changes. Under normal conditions, we don't expect HF SCS to affect the dorsal columns, but in this case, the extreme migration may have caused it to affect them and would explain the paresthesia. Such extreme lead migration is rare, but similar occurrences have been

reported before.^{5,6} In these other instances, similar paresthesias have also been noted, further indicating that the migration was the underlying cause. The cause of the migration is unknown and could be seen in cases of excessive movement or manipulation; however, the patient denies either occurred. Other contributing factors to the migration may be the lack of adequate scar tissue formation by post-op day 2, which forms later and aids in securing the leads.

As a result of lead migration, the distance between the stimulator and target changed and the applied HF stimulation most likely fell outside of the appropriate stimulation window. In order to achieve paresthesia-free stimulation without eliciting uncomfortable sensation, the stimulation must remain within a particular stimulation window. Changing the distance can bring stimulation outside of this therapeutic window, thereby causing uncomfortable sensations or paresthesias.⁷ We believe that this change in distance between the leads and the target accompanying the change in vertebral level lead to overstimulation and was the most likely cause of her symptoms in the T1-T3 dermatomes.

Investigations into the mechanisms of kilohertz frequency SCS have shown that in supra-threshold stimulation of nerve fibres at higher frequencies, there is initially an asynchronous firing, followed by a conduction block.⁸ After the migration of the leads occurred, the new target fibres of the stimulation would be activated and begin the asynchronous firing leading to the paresthesia symptoms in the T1-T3 dermatomes. The conduction block most likely did not occur in this case, as paresthesia would not be expected to occur if the dorsal column fibres were blocked. Conduction block most likely did not occur because the thresholds for conduction block are higher than the thresholds for activation.⁸ Patients receiving HF SCS may experience a 'wash out' effect where the effects of the therapy persist even after the stimulation is turned-off. However, in this case, the symptoms resolved as soon as the stimulation ceased, most likely due to the stimulation being ended shortly after the migration. Similarly, there is often a 'wash in' effect when initiating the therapy, which may explain why the patient did not receive pain relief when the leads were repositioned, and stimulation was resumed. The leads were then explanted and not enough time had passed to see if the trial would have again succeeded in restoring pain relief.

Conclusion

In this report, we describe the first case of lead migration-induced paresthesia following standard use of HF SCS in a patient with FBSS. The patient underwent a trial of HF SCS for significant lower back and leg pain after two fusion surgeries. After 1 week, she returned to

the office complaining of left chest and upper extremity paresthesias along with loss of efficacy for her back and leg pain. Imaging showed that the array had migrated to the vertebral levels of T1-T3.

While HF SCS has emerged as an effective paresthesia-free means of reducing back and leg pain, this is the first report of paresthesias occurring with the HF SCS system, specifically in the setting of lead migration. Similar to the adverse symptoms seen in conventional SCS lead migration, HF SCS lead migration can result in loss of efficacy. In addition to this loss in pain relief, migration of the HF SCS system can still result in paresthesias, something not discussed in the existing literature. As HF SCS is now slowly being utilized as a treatment modality in most centres across the world, its safety, efficacy and appropriate use will continue to be studied and reported.

Conflict of interest

Dr. Mammis is a paid consultant for Nevro Corp.

Funding

The author(s) received no financial support for the research, authorship and/or publication of this article.

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