Pain Medicine Case Reports

SEROMA FORMATION 20 YEARS AFTER SPINAL CORD STIMULATOR INSERTION: A CASE REPORT

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- **Background:** Spinal cord stimulation is a common treatment modality for chronic neuropathic pain. Device complications can include infection, hardware malfunction, and seroma formation.
- **Case Report:** A 78-year-old woman presented to the pain management clinic 20 years after spinal cord stimulator insertion with 6 months of pain and swelling around the stimulator insertion site. Aside from localized pain and swelling, the patient did not experience any neurological or musculoskeletal symptoms. A computed tomography scan revealed a large seroma around both the spinal cord stimulator and the extension wiring. The patient was referred to neurosurgery, where she underwent successful explant of the stimulator without further complication.
- **Conclusions:** This case is an excellent example of complications that can occur with the extension wiring of spinal cord stimulators. Many physicians are aware of the complications that occur with the generator and electrodes, but extension wiring is a forgotten source of adverse outcomes.

Key words: Neuropathic pain, seroma, spinal cord stimulator

BACKGROUND

Spinal cord stimulators (SCS) are a common treatment option for patients with chronic neuropathic pain that is refractory to less invasive therapies (1). They are useful in the treatment of chronic back pain, chronic postoperative pain, complex regional pain syndrome, and other causes of neuropathic pain. These devices use low-level electrical impulses to stimulate Aß afferent fibers, in turn inhibiting pain signals from A δ and C efferent fibers (2,3). The exact mechanism of action is not completely understood, but release of pain-modulating neurotransmitters such as GABA, substance P, and serotonin may play a role (4-6).

SCS are composed of electrodes placed in the epidural space and an internal pulse generator; in some cases, an extension wire connects the electrodes to the pulse generator (7). Common complications of SCS insertion include hardware malfunction (lead migration, breakage, connection failure), infection, pain at the generator site, cerebrospinal fluid leak, hematoma, and seroma (8-11). Seroma around the pulse generator site is a less common complication of SCS insertion but can lead to infection if untreated (12,13). Formation is caused by lymphatic obstruction, inflammatory mediators, tissue shearing, and creation of dead space (14).

Seroma development can be mitigated by making the pulse generator pocket small and minimizing movement in the subcutaneous space (10). Treatment of SCS seroma should be based on the progression of the seroma; options include conservative observation, prophylactic antibiotics, percutaneous aspiration, and surgical evacuation (15). There are currently no documented

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instances of seroma formation around a SCS extension wire. Informed consent was obtained from the patient for the publication of this case report.

CASE PRESENTATION

The patient presented in this case is a 78-year-old woman with a past medical history of lumbar radiculopathy, cervical radiculopathy, cervical spondylosis, and atrial fibrillation. She was referred to the interventional pain management clinic for localized swelling around her SCS generator. She has a history of SCS insertion for lumbar radiculopathy, which was placed at another facility 20 years earlier. Six months prior to her presentation, she began to notice swelling and pain around the SCS generator site. The quality of her pain was dull, aching, and pressure; the pain was worse with compression of the site. She described the severity as a 4 out of 10 on a numerical pain scale. Aside from localized pain, the patient did not report any other symptoms; she did not experience any numbness or tingling of her extremities, bowel or bladder incontinence, or fevers. On exam, an area of mildly tender, compressible swelling was noted on the right lateral flank. The skin overlying the area was warm, dry, intact, and non-erythematous. Her extremity muscle strength, range of motion, and reflexes were normal and symmetric.

Computed tomography (CT) of the chest demonstrated a 1.1-cm-wide fluid collection bordering the SCS generator in the right lateral lower chest. Two additional fluid collections were noted in the right posterior paramedian chest at the T12 level around the extension wire: the medial collection measured 4.2 cm in length by 1.9 cm in depth; the lateral collection measured 3.2 cm in length by 1.9 cm in depth. The fluid collections were consistent with the diagnosis of uncomplicated seromas. Alternative diagnoses considered included abscess, hematoma, and SCS hardware migration.

Expectant management was not chosen due to the nature of the patient's symptoms and age of the implanted device. Percutaneous drainage was not recommended due to the location of the multiple seromas. Additionally, definitive resolution of the issue would be established with surgical removal of the device. The patient was referred to neurosurgery for elective surgical explant of the device. Explant was performed without any adverse outcome.

The patient was adherent to both pain management and neurosurgical follow-up appointments. The patient presented to her postoperative visits with resolution of symptoms; she was satisfied with the treatment course and outcome of the surgery. She verbalized relief of her pain, along with satisfactory resolution of local swelling.

DISCUSSION

This patient presented with only localized swelling and pain at the SCS generator site; she did not experience any other neurological complications from the development of this seroma. Seroma development can result in nerve compression and peripheral nerve symptoms such as pain, numbness, and tingling. Additionally, seromas can grow in size or progress to infection.

Prompt identification of seroma can guide treatment and prevent further complications. There is no way to predict the likelihood of developing a seroma postoperatively after SCS insertion but minimizing surgical dead space and tissue shearing can help prevent development (16). Similarly, there is often no indication of where a seroma is likely to develop. Factors such as tissue shearing, creation of dead space, and inflammation are associated with seroma formation; they often occur around the generator site (17). This case is unique due to the location of the seroma in conjunction with the long latency period before formation. Seromas typically present within one month of the initial tissue insult, as opposed to 20 years in this case (18). Possible causes of the late presentation of seroma in this patient include trauma to the area, tissue shearing from hardware, and dead space creation from movement of hardware (14). Measures should be taken to avoid the inciting factors of seromas and to accurately identify this complication.

The care of this patient was strengthened by the ability to refer to expert definitive treatment through neurosurgical removal of the SCS. Prompt identification of the seroma, attainment of advanced imaging studies, and adherence to referrals by the patient led to a quick and uncomplicated resolution of this uncommon complication. Our presentation is limited due to the fact that the CT images are not available for publication.

CONCLUSION

To our knowledge, this is the first case where a seroma has not only developed around the SCS generator site, but also the extension wiring. This case can guide physicians in the monitoring of complications of SCS insertion. Additionally, this case draws attention to the fact that SCS complications can occur at any stage of treatment, even 20 years post insertion. Seromas, and their complications, should be considered at any time after SCS placement.

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