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Spinal Cord Infarction as an Immediate Complication of Spinal Cord Stimulator Placement

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Background: Spinal cord stimulators (SCSs) are a widely used intervention for managing chronic neuropathic conditions. SCSs are considered safe, but rare complications can arise, typically attributed to spinal cord compression or contusion. Spinal cord infarction as a complication of SCS placement has not been previously reported in the literature.

Case Report: A 76-year-old woman developed spinal cord infarction resulting in right leg paralysis following SCS placement. Immediately postprocedure, there was bilateral lower extremity paralysis with partial recovery of the left leg but persistent right leg paralysis. Extensive changes consistent with cord infarction from C7-L2 were seen on spinal magnetic resonance imaging, and vascular compromise of spinal arterial supply was suspected. Despite medical management and rehabilitation, no significant improvement in right leg function was observed.

Conclusions: This case highlights spinal cord infarction as a rare but serious complication of SCS implantation. Physicians should be aware of potential vascular injuries and counsel patients accordingly.

Key words: Spinal cord stimulator, paralysis, spinal cord ischemia

BACKGROUND

Spinal cord stimulators (SCSs) were introduced in 1968 for chronic lower back pain management (1). Since then, several randomized controlled trials (2,5) have demonstrated that SCSs can be a viable treatment option for complex regional pain syndrome, chronic back and leg pain, failed back surgery syndrome, ischemic limb pain, and intractable angina.

The procedure involves placing electrodes in the epidural space of the spinal cord to stimulate the dorsal columns. These electrodes are inserted through a small laminectomy or laminotomy, inducing paresthesia to replace pain (3).

Previous case series and reports (3) suggest that SCS is generally safe. A retrospective review of 12,297 SCS cases by Labaran et al (4) found at 90 days postoperatively, 4.2% of patients presented to the emergency department, while 0.3% required SCS removal or reimplantation. Other surgical complications included wound infections (4.3%), hematoma (0.5%), and seroma (0.4%) (4). There are a limited number of published cases (2,7) of paralysis occurring after SCS placement, all of which have been attributed to cord compression or contusion. To our knowledge, no cases of spinal cord infarction due to SCS placement have been reported.

Here, we present a case of neurologic injury from spinal cord infarction following SCS implantation, which resulted in right leg paralysis.

CASE PRESENTATION

On hospital day 1, a 76-year-old female patient with a history of hypertension, chronic kidney disease, multiple

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myeloma, obesity, and prior lumbar spine surgery underwent temporary SCS placement under conscious sedation for chronic lower back pain that was worse on the right side. Immediately postprocedure, she experienced bilateral leg numbness and paralysis, and was brought to the emergency room. She reported no bowel or bladder symptoms or saddle anesthesia. Her vital signs were stable, though she reported left-sided chest discomfort. An emergent computed tomography scan of the spine was performed—although devoid of findings of acute cord compression or other obvious injury—did reveal a small focus of intraaxial air at the level of T2 (Fig. 1). Neurosurgical services were consulted, and suggested stimulator lead-induced spinal cord compression as the etiology of her symptoms; steroid treatment was begun, and further imaging with magnetic resonance imaging (MRI) was recommended.

On hospital day 3, MRI of the spine was performed, revealing extensive T2 signal abnormality throughout the thoracic region, involving the central cord from C7-T1 to T7-T8, and right of midline from T7-T8 to L2, suggestive of cord infarction (Fig. 2). At this time, the patient's left leg strength had improved significantly, but her right leg remained flaccid except for some minimal movement in the toes. The remainder of the neurological exam showed reduced pain and temperature sensation from the right lower trunk throughout the right leg without a marked sensory level; normal vibration and proprioception sense in both legs; and absent deep tendon reflexes in both legs. Arm strength and sensation were normal, although the patient did develop transient tremors in both arms, thought likely to be due to anxiety, and resolving within a day.

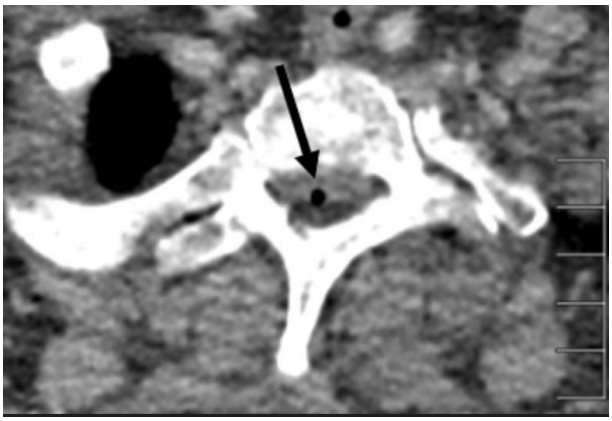

Fig. 1. CT scan of the thoracic spine demonstrating a focus of intraaxial air at T2. CT, computed tomography.

Also on day 3, cardiac telemetry revealed new-onset atrial fibrillation, and the patient was started on low-molecular-weight heparin. By day 5, she had also developed urinary retention. In addition, she had an episode where the fingers of her left hand became markedly pale-colored and cold to touch, consistent with localized vasoconstriction, thought possibly due to autonomic dysreflexia from the spinal cord injury. Her neurological exam would remain unchanged for the duration of her hospitalization.

On hospital day 9, while still on anticoagulation for newfound atrial fibrillation, the patient developed a large retroperitoneal hematoma leading to hemorrhagic shock, which required open surgical intervention and transfer to the intensive care unit. She was moved to the general medical floor on hospital day 15 and subsequently discharged to acute rehabilitation on day 23. She was seen in clinic 34 days after initial symptom onset, but unfortunately, at that time, the patient continued to experience no improvement in her neurological function.

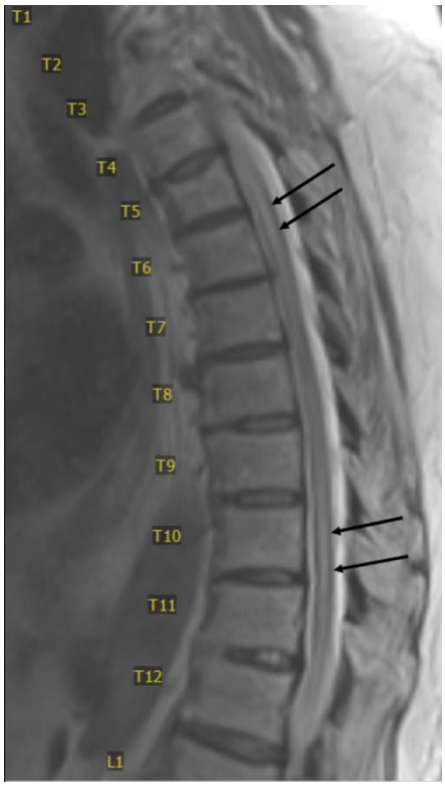

Fig. 2. Axial T2-weighted MRI of the thoracic spine showing increased signal intensity within the spinal cord. MRI, magnetic resonance imaging.

DISCUSSION

SCS is used for treating complex regional pain syndrome, chronic back and leg pain, failed back surgery syndrome, ischemic limb pain, and intractable angina (2,5). This case demonstrates that, although rare, neurologic injury is a serious possible complication of SCS placement. We present a case of a woman who developed suspected spinal cord infarction as a consequence of the SCS procedure. To the best of our knowledge, spinal cord infarction as a direct complication of SCS placement has not previously been reported.

There are several indicators in this case suggesting that cord infarction occurred, rather than cord contusion, despite the latter being the primary observed cause of paralysis following these procedures (2,7). These include the primarily centralized location of the cord injury as seen on MRI, as well as the extent of cord injury as high as C7, a finding that would not be expected with contusion caused by needles or lead placement from lumbar access. Additionally, the intraaxial air at T2 is particularly suggestive of vascular injury; periprocedural injury to spinal arterial supply may have introduced the air, which subsequently moved through the vasculature. Imaging revealed no overlying hemorrhage or other injury to suggest other mechanisms of air entry, and cord contusion would also not be expected to result in intraaxial air.

Although the definitive cause of infarct in this case is not known, a possible mechanism is a mechanical interruption of blood supply to the spinal cord caused by the stimulator lead during placement, specifically involving the artery of Adamkiewicz. This artery has high anatomical variability and is a primary source of blood flow to the anterior cord from the midthoracic region caudally (6). Injury to this artery could therefore cause extensive spinal cord infarct and substantial neurological deficits. This could occur if the stimulator lead inadvertently tracks anteriorly during advancement, or if anatomical variation places this artery in greater proximity to the normal position of the lead, or a combination of both. Spinal cord angiography may have provided additional insight into the infarction mechanism, but it was not expected to alter management and was therefore not performed.

This patient also experienced a spontaneous retroperitoneal hematoma, discovered on day 5, after 2 days of anticoagulation with low-molecular-weight heparin for newfound atrial fibrillation. This was considered unrelated to the SCS procedure, due to the time interval in between these 2 events, the absence of any retroperitoneal bleeding seen on the spinal MRI on day 2, and the unlikelihood of the SCS leads entering the retroperitoneal space and compromising vasculature there.

CONCLUSIONS

This case report describes spinal cord infarction during SCS implantation, highlighting an exceedingly rare but serious potential neurological complication associated with this procedure. While SCS has proven effective for managing various chronic pain conditions, the potential for serious spinal cord injury remains. Given the rarity of this event, and the invasive nature of spinal angiography, we do not believe this warrants screening for vascular anatomy preoperatively. However, further research into the spinal and paraspinal vascular anatomy may enhance surgical planning and outcomes. Counseling patients regarding the risks of this procedure should therefore encompass all potential outcomes, including the risk of serious and irreversible neurological injury.

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