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SPINAL EPIDURAL HEMATOMA MANAGED CONSERVATIVELY AFTER A SPINAL CORD STIMULATION TRIAL: A CASE REPORT

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Background: Spinal cord stimulation (SCS) is used to treat chronic pain but carries a rare risk of spinal epidural hematoma (SEH), which can cause serious neurologic injury. Urgent surgical decompression is often considered when neurologic deficits develop.

Case Report: A 73-year-old man developed severe back pain during an SCS trial and was found to have a thoracolumbar SEH after trial lead removal. He remained neurologically stable during hospitalization and was managed with close observation. His pain improved, and follow-up imaging showed a reduction in the hematoma without surgery.

Conclusions: This case illustrates conservative management of SEH in a neurologically stable patient with close monitoring. Further evidence is needed to define which patients may be managed without surgical decompression.

Key words: Case report; spinal cord stimulation; spinal epidural hematoma; chronic back pain; conservative management.

BACKGROUND

Spinal cord stimulation (SCS) is a treatment option for chronic pain when other therapies have been inadequate. As its use increases, complications warrant attention. West et al. (1) examined 40 publications and reported a pooled incidence of 0.81% for any hematoma and 0.32% for neuraxial hematoma after SCS procedures.

Spinal epidural hematoma (SEH) may present with acute back pain, radicular symptoms, or neurologic deficits. Magnetic resonance imaging (MRI) is generally used for diagnosis. Because spinal cord compression can cause permanent injury, urgent assessment and, when indicated, surgical decompression are essential (2).

Giberson et al. (3) reported 2 patients who required surgical evacuation of SEH after removal of percutaneous SCS trial leads. The first patient developed acute severe back pain and paraplegia after lead removal. MRI showed a T5–L1/L2 hematoma with cord compression. Despite laminectomy and evacuation, the patient had persistent left lower extremity weakness.

The second patient developed severe thoracic pain and lower extremity weakness shortly after trial lead removal, 4 days after placement. A T8–L1 laminectomy and evacuation were performed for a T8–L3 SEH, and the patient recovered without neurologic deficits (3). These cases show the urgency of evaluating new pain and neurologic symptoms after lead removal.

SEH can be a neurosurgical emergency, particularly when neurologic function deteriorates. Yet management depends on symptoms, neurologic findings, imaging, and clinical course. We describe a patient who

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improved without surgical evacuation while undergoing close monitoring.

CASE PRESENTATION

A 73-year-old Caucasian man returned to the clinic with worsening chronic right-sided low back pain and recurrent peripheral neuropathy in both feet. His history included hypertension, hypercholesterolemia, peptic ulcer disease, and benign prostatic hyperplasia; he reported no tobacco or alcohol use. He had previously responded to radiofrequency ablation at right L3–L4 and L4–L5. An SCS trial with leads at T9–T10 was planned. Lumbar MRI before the trial showed anterolisthesis at L4–L5 and L5–S1, with bilateral foraminal narrowing most pronounced at L5–S1. The percutaneous trial was performed approximately 6 months after this visit without reported procedural complications. Before lead placement, the patient denied taking anticoagulants and reported no bleeding disorder or thrombocytopenia.

Five days after placement of the SCS trial leads at T9–T10, the patient returned for removal. He reported approximately 50% relief of neuropathic paresthesia but had developed severe back pain. Review of fluoroscopic spot images showed no substantial lead migration. His pain persisted after lead removal, and his blood pressure was elevated. He was sent to the emergency department. Thoracic MRI with and without contrast showed a left dorsolateral epidural collection, likely hematoma, extending from T10–T11 through the L1–L2 disc space, with canal stenosis at T11–T12 and T12–L1 (Figs. 1 and 2). The on-call orthopedic spine surgeon assessed him; he denied bowel or bladder incontinence and lower extremity weakness. He was admitted to the intensive care unit for frequent neurologic examinations, and his pain was treated with acetaminophen. The team deferred surgery because his neurologic examinations remained stable.

Approximately 2 weeks after hospital discharge, the patient followed up with the orthopedic spine surgeon. His back pain had improved substantially, and he reported no new lower extremity weakness or paresthesia. A repeat thoracic showed decreased mass effect from the hematoma (Figs. 1 and 2). At another visit 1 week later, he continued to do well without new neurologic symptoms and was advised to follow up as needed.

DISCUSSION

This case illustrates an observed improvement without surgery in a patient with SEH who remained neurologically stable under close surveillance. The decision to

Fig. 1. Sagittal MRI of the thoracolumbar spine showing a left dorsolateral epidural collection from T10–T11 through the L1–L2 disc space, with canal stenosis most pronounced at T11–T12 and T12–L1 (left). Follow-up imaging shows decreased mass effect and improved canal caliber (right).

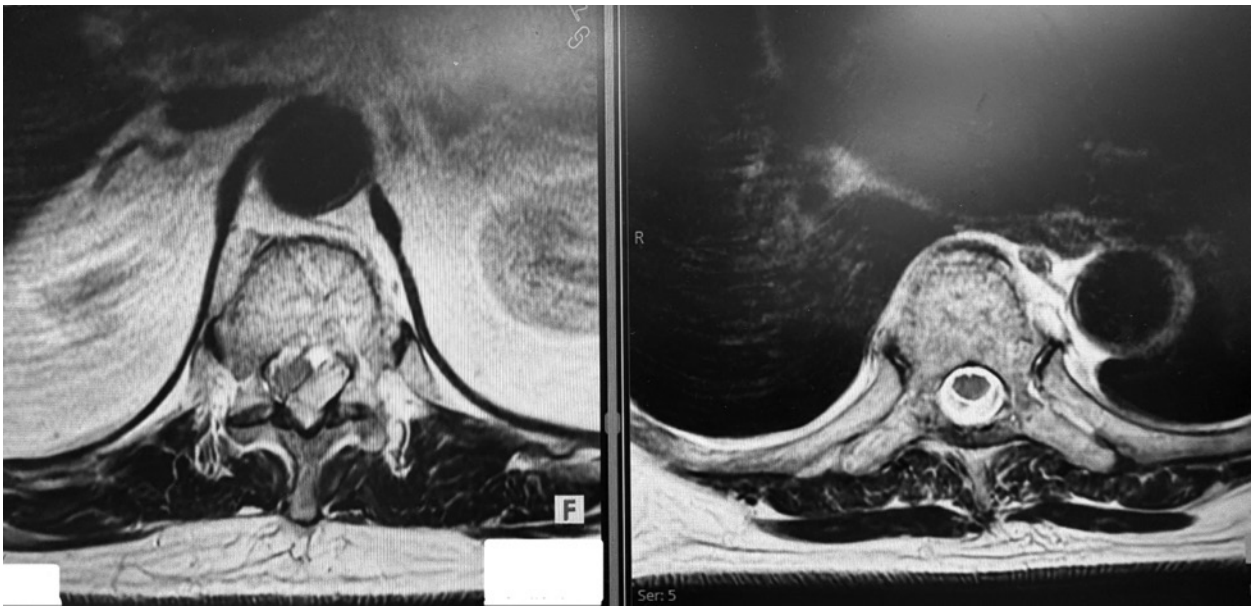

Fig. 2. Axial MRI showing a left dorsolateral epidural collection with thecal sac compression and canal narrowing (left). Follow-up imaging shows a smaller collection and decreased mass effect (right).

observe rather than decompress should be interpreted in the context of the patient's examination, imaging, and clinical course.

Urgent decompression is frequently considered when SEH causes progressive neurologic impairment. Lenga et al. (4) reported surgical outcomes in octogenarians with spontaneous SEH; that population differs from a patient with hematoma identified during an SCS trial. Du et al. (5) described a patient with SEH after combined spinal–epidural anesthesia whose hematoma resolved with conservative treatment. Together, these reports illustrate the importance of matching treatment to the clinical course and neurologic findings.

Noori et al. (6) described an SEH after SCS trial lead removal in a patient with chronic thrombocytopenia. Her symptoms resolved without lasting neurologic sequelae. In our case, observation was chosen because repeated examinations showed no neurologic deterioration.

CONCLUSIONS

This case describes improvement of an SEH without surgery in a patient who remained neurologically stable under close observation. It does not change the need for urgent evaluation and timely decompression when neurologic deficits develop. Further study is needed to identify patients for whom observation may be appropriate.

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Author Contributions

Conceptualization and visualization: L.P.; writing of the original draft: L.P.; data curation and resources: J.D.; resources: J.R.; review and editing: L.P., S.M., and J.D.; project administration: S.M.

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