

Progression of Spinal Stenosis at the Spinal Cord Stimulator Percutaneous Lead Entry Point

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Purpose

Spinal cord compression (SCC) following spinal cord stimulator (SCS) implantation is a rare, but serious complication that can occur secondary to progressive spinal canal stenosis. Furthermore, SCC can occur at lead entry locations in the lumbar spine. Physicians should be aware of the clinical symptoms of SCC including loss of efficacy of the SCS device and treatment options.

Background

- ❑ Spinal cord stimulation implants comprise the third-highest number of adverse events from medical devices behind only insulin pumps and total hip arthroplasties [1]
- ❑ Most complications are attributed to hardware malfunctions, primarily including lead migration and lead fractures [2]
- ❑ Spinal cord compression is rare and typically manifests from fibrotic scar tissue formation

Case Report

- ❑ A woman in her 70s presented to a pain management physician with complaints of low back pain and lower extremity radicular pain which began several years prior
- ❑ Past medical history: L3-S1 laminectomy with L4-L5 instrumental fusion
- ❑ Nonresponsive to conservative interventional treatments and pharmacological management
- ❑ Underwent a Nevro spinal cord stimulator (SCS) trial and implant with two percutaneous leads and a rechargeable generator
- ❑ Pre-operative lumbar magnetic resonance imaging (MRI) → mild spinal stenosis at L1-L2 (Fig. 1)
- ❑ 18 months post SCS implant → increased pain in her lumbar spine despite programming optimization sessions
- ❑ X-rays to trace SCS system → leads adequately positioned, no evidence of lead migration
- ❑ 26 months post SCS implant → increased low back pain and lower extremity radicular pain with associated perceived weakness
- ❑ Lumbar MRI → moderate to severe spinal canal stenosis at L1-L2 (Fig. 2)
- ❑ Referred to neurosurgeon → L1-L2 decompressive laminectomy with removal of the SCS leads and battery. Surgery confirmed ligamentum and facet joint hypertrophy.
- ❑ Resolution of low back pain, lower extremity radicular pain, and perceived lower extremity weakness

Authors	Age and Sex	Type of lead (s)	Clinical signs	Location of SCS	Compression location	Imaging	Interval	Surgical Treatment	Sequelae	Histology/Surgical findings	Comments
Reynolds, 1983	26, M	Paddle	Hyperactive reflexes of the bilateral upper and lower extremities; unstained clonus at the ankles and upgoing toes	C1-C4	Cervical	n/a	2 Years	Scar tissue removal; SCS explant	Resolution of spastic quadriparesis	Dense fibrous tissue; fibroblastic nuclei arranged in straight lines	n/a
Dam Hieu, 2010	66, F	Paddle	Gait disorder, quadriparesis with sensory impairment; pyramidal syndrome	C4-C5	Cervical	MRI	16 Years	Scar tissue removal; SCS explant	Complete resolution of symptoms	Fibrosis; chronic inflammatory reaction without specificity	n/a
Dam Hieu, 2010	58, M	Paddle	Motor deficit in the left inferior limb; dysesthesias and hypesthesia of the left hemi body	C4-C5	Cervical	MRI	5 Years	Scar tissue removal; SCS explant	Neurological disorders disappeared; symptoms related to CRPS recurred	Fibrous scar tissue	n/a
Wada, 2010	42, M	Percutaneous	Spastic quadriparesis; exaggerated deep tendon reflexes of the lower extremities	C4-C5	C1-C5	CT with myelography	5 Years	Unilateral open-door laminoplasty from C3 – C6; SCS explant	Gradual recovery	Fibrous scar tissue without inflammatory cells	n/a
Cicuende z, 2012	66, M	Paddle	Spastic paraparesis; weakness of the lower extremities	T7	T7-T8	MRI	1 Year	Thoracic laminectomy; SCS explant	Improvement in neurological symptoms and signs	Fibrosis; non-specific chronic inflammatory reaction	n/a
Wloch, 2012	69, M	Paddle	Hemihypesthesia; mild motor deficit; burning pain of the left arm; decreased tendon reflexes; gait ataxia	C2-C4	C2-C4	Plain X-rays; CT myelograms	17 Year	C3-C4 hemilaminectomy; SCS explant	No further progression of gait disorder	Fibrous scar tissue	n/a
Lennarson, 2010	56, F	Paddle	Lack of effectiveness; upper extremity weakness; concern for infection	C3-C5	Cervical	MRI; Plain X-ray; CT scan	2 Years	C3-C4 hemilaminectomy; SCS explant	Resolution of paresthesia and burning pain; improved strength	Fibrous tissue; foreign body giant cell reaction; no significant acute inflammation	Revision of SCS secondary to electrode fracture, MRSA infection
Al Tamimi, 2017	48, F	Percutaneous	Progressive neck pain in addition to increased limb pain; paresthesia; extremity weakness	Cervical	Cervical	MRI	Not specified	Decompressive laminectomy; SCS explant	Improvement in weakness; unchanged cervicalgia	Fibrosis; granulomatous tissue formation; nonspecific chronic inflammatory reaction	n/a
Benfield, 2016	61, M	Paddle	Progressive paraplegia; thoracic radicular pain; bowel and bladder incontinence	T7-T9	T7-T9	CT Myelogram	10 Years	Decompressive T7-T9 laminectomy; SCS explant	Pre-SCS implant pain returned	Fibrotic scar tissue	n/a
Guzzi, 2015	59, F	Paddle	Paraesthesias; gait ataxia; lower extremity weakness; increased muscle tone and tendon reflexes in the lower extremities	C3-C5	C3-C5	CT; MRI	7 Years	C6 left hemilaminectomy; SCS explant; C3-C4-C5 laminectomy	Progressive but incomplete improvement of neurological signs without developing new deficits	Fibrosclerosis; aspects of reparative type and without inflammatory cells.	Compression occurred following SCS revision surgery
Damar, 2016	61, M	Paddle	Decreased grip strength; increased paresthesia, numbness, and weakness of upper extremities; midline paraspinal tenderness	Cervical	Cervical	CT	10 Years	SCS explant, decompression and fusion C2-C5; C4 vertebrectomy	Resolution of numbness and paresthesia of the bilateral upper extremities	Silicone granuloma formation	2 SCS implants; 4 SCS revisions; intrathecal pain pump with revision
Fransen, 2014	42, M	Percutaneous	Limited gait, progressive weakness	T8	Thoracic	n/a	12 Years	Decompressive laminectomy	Improvement in gait and pain control	Fibrous scar tissue	Three lead replacements; two cases of infection
Anoun, 2018	85, F	Paddle and Percutaneous	Lower extremity weakness bilaterally; intractable thoracic pain	T8-T9	Thoracic	CT	8 Years	Laminectomy at T8 and T9; SCS explant	Myelopathic symptoms resolved	Organized fibrous tissue	Sectioning of paddle lead wires; insertion of percutaneous leads
Kim, 2018	46, M	Paddle	Cervical pain; bilateral upper extremity numbness; bilateral lower extremity weakness; gait abnormality	Cervical	Cervical	CT	10 Years	C2-C6 Laminectomy and cervical spine fixation	Immediate pain relief; improvement in neurological symptoms	Metallosis and fibrosis; granulomatous inflammation; phagocytic reaction of neutrophils and macrophages on the metal debris	Metallic irritation of the dura mater
Scranton, 2015	41, F	Percutaneous	Spastic gait; upper extremity weakness; hyperreflexia of the upper and lower extremities	C2 –C5- C6	C3-C4, C4-C5	MRI	9 Months	C2-C7 Laminectomy; SCS explant	Improvement in bilateral upper and lower extremity strength	Dense fibroconnective tissue; marked chronic inflammation; multinucleated giant cells; several noncaseating granulomas	n/a
de Eulate-Bernard, 2016	55, M	Paddle	Dysesthesias of the lateral 3 fingers of the left hand; motor deficit; hypoalgesia	C4-C6	C4-C6	MRI	7 Years	Removal of fibrotic mass; SCS explant	Neurological improvement; improvement in motor and sensory evoked potentials	Fibrous scar tissue	n/a

Table 1. Literature review of all case reports of spinal cord compression following spinal cord stimulator implant. “n/a” denotes not applicable.

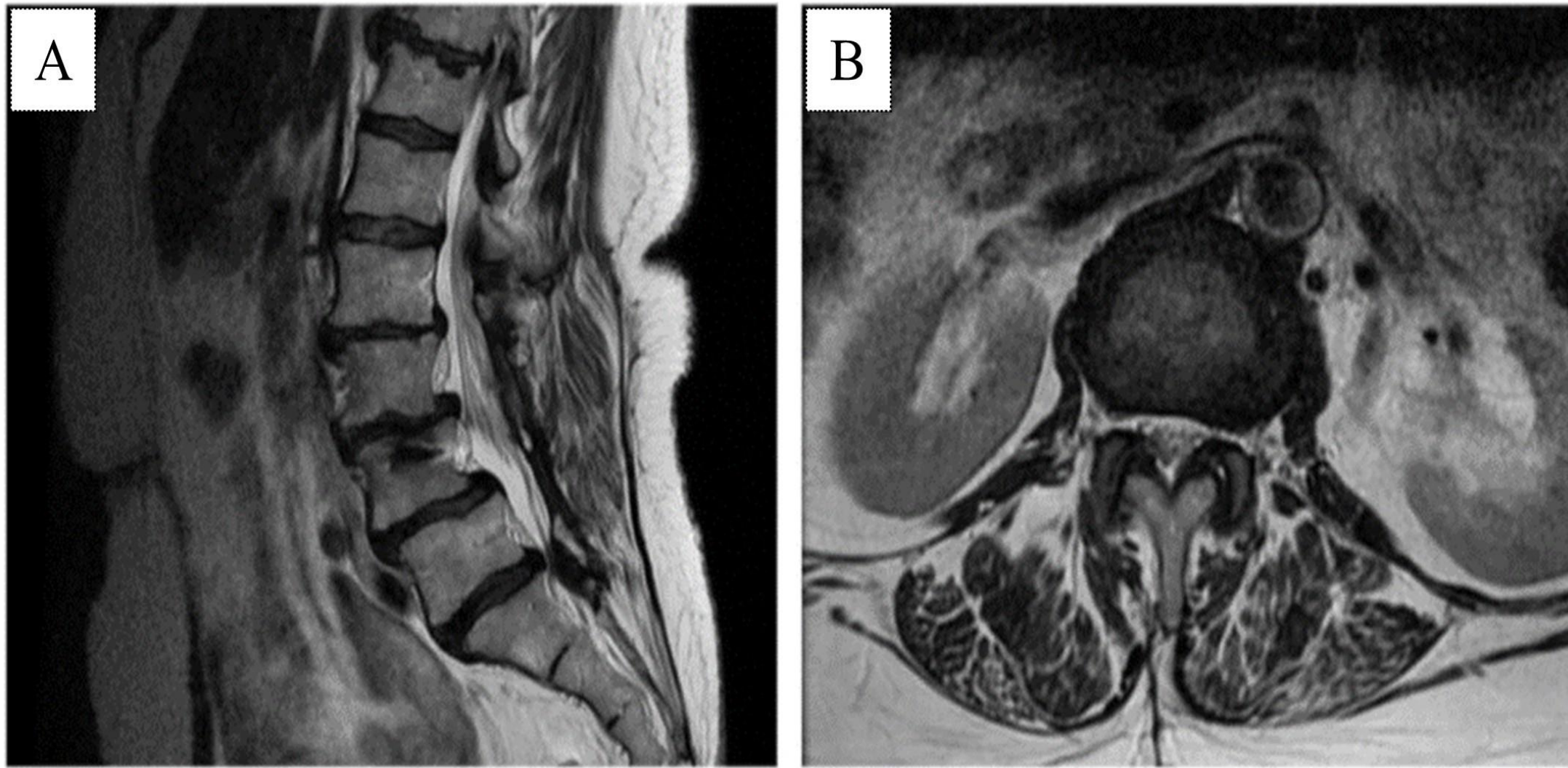


Fig. 1. Magnetic resonance imaging scan obtained two months prior to the spinal cord stimulator trial. (A) T2 sagittal and (B) T2 axial images demonstrating mild central canal stenosis at L1-L2. Appearance is unchanged compared to lumbar magnetic resonance imaging scan obtained six months prior.

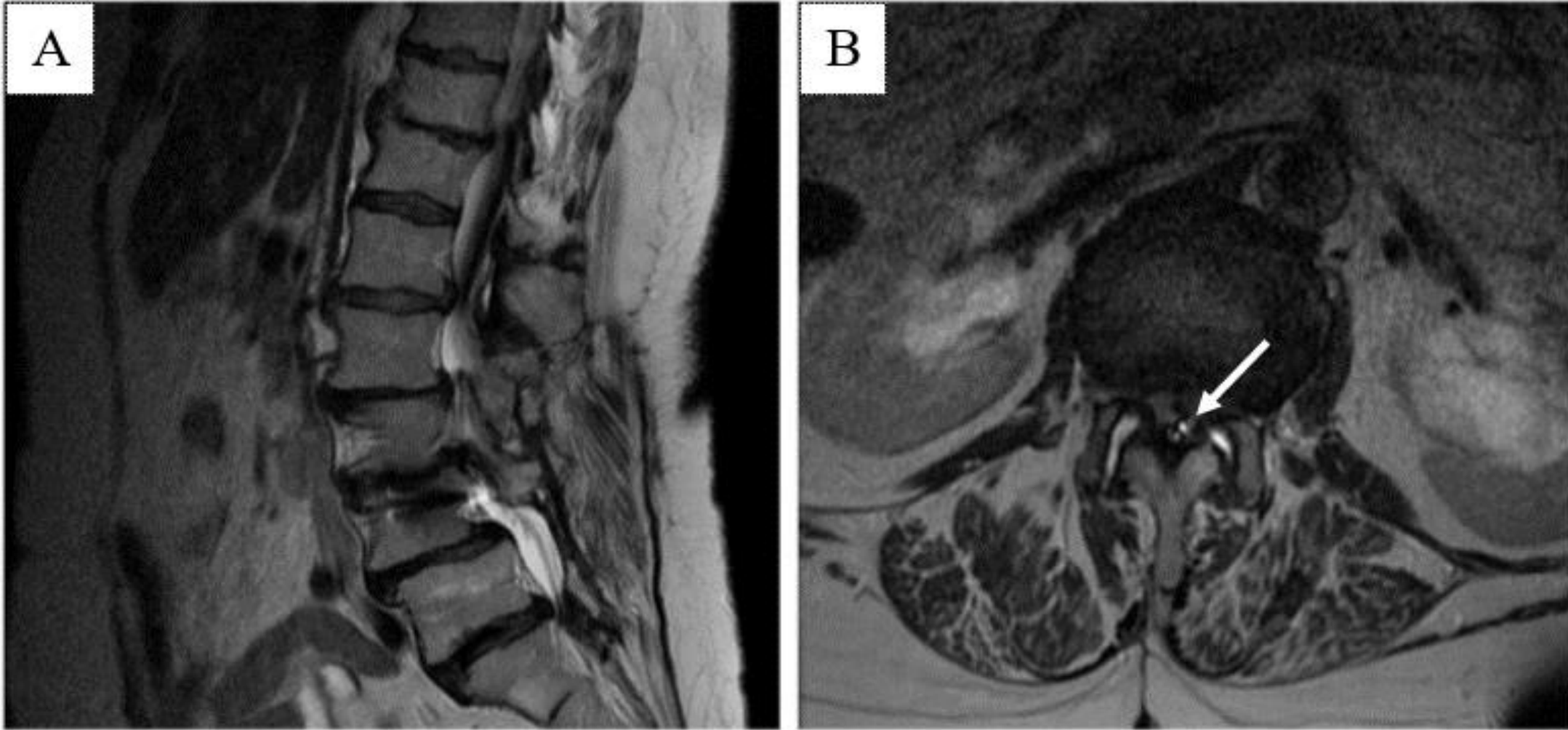


Fig. 2. Magnetic resonance imaging scan obtained thirty months following the spinal cord stimulator implant. (A) T2 sagittal and (B) T2 axial images demonstrating a notable interval progression at L1-L2 resulting in moderate to severe spinal canal stenosis and moderate right and moderate to severe left neuroforaminal narrowing secondary to bilateral facet arthropathy, small broad-based disc bulge, and bilateral ligamentum flavum hypertrophy. White arrow denotes SCS electrode.

Key Points

- ❖ SCC following SCS compression can occur at lead entry levels in the lumbar spine
- ❖ SCC can occur secondary to progressive spinal canal stenosis
- ❖ MRI conditionality is vital to identifying spinal cord compression
- ❖ Loss of efficacy of SCS device warrants investigation
- ❖ Treatment includes SCS explant

Discussion

- ❑ 16 published cases of SCC in patients 1—17 years post-implant (Table 1)
- ❑ Clinical indications: loss of efficacy of SCS device and perceived weakness
- ❑ All cases demonstrated SCC in the thoracic and lumbar spine at the final lead placement location (Table 1)
 - i. Case Report: lead entry location of *one lead* in the lumbar spine, L1-L2
- ❑ From T12-L1 to L5-S1, the lowest incidence of spinal canal stenosis occurred at L1-L2 (1.4%) and T12-L1 (0.4%) [3]
 - i. Spinal canal stenosis is rare at lead entry levels
- ❑ Paddle electrodes can decrease the cross-sectional area of the spinal cord, the anterior and posterior CSF space, and the thecal sac [4]
- ❑ Most instances of SCC following SCS implantation occurred with paddle leads (Table 1)
 - i. Larger surface area of the paddle electrode and disruption of native anatomy
- ❑ Nearly all previously reported cases of SCC occurred secondary to fibrotic scar tissue formation (Table 1)
 - i. MRI conditionality is vital
- ❑ Hypothesize ligamentous hypertrophy, facet hypertrophy, disc degeneration, and the SCS leads themselves contributed to SCC
 - i. Ligamentum flavum hypertrophy is a known contributor to spinal canal stenosis [5]
- ❑ Typical treatment: removal of SCS hardware and decompressive laminectomy

References

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