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# Perioperative Management of a Patient With Primary Adrenal Insufficiency Undergoing Spinal Cord Stimulation: A Case Report

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**Background:** Spinal cord stimulation (SCS) is an established therapy for the management of chronic neuropathic low back pain (LBP) that is refractory to conventional measures. Although considered minimally invasive, SCS constitutes a surgical intervention that may induce significant physiological stress, placing patients with primary adrenal insufficiency (PAI) at an elevated risk of adrenal crises during perioperative periods.

**Case Report:** The authors present a woman with chronic LBP and PAI who successfully underwent SCS trial and permanent implantation with stress-dose glucocorticoid supplementation perioperatively, which resulted in 90% pain reduction, improved functional capacity, decreased analgesic reliance, and no adrenal crisis. To the authors' knowledge, there are no published clinical guidelines that address perioperative glucocorticoid management in PAI patients undergoing SCS implantation.

**Conclusions:** Our case highlights a safe and effective perioperative protocol for SCS in PAI patients and underscores the importance of endocrine comorbidities when undergoing interventional procedures.

**Key words:** Case report, spinal cord stimulation, adrenal insufficiency, pain

## BACKGROUND

Chronic low back pain (LBP) is one of the most prevalent health conditions worldwide, affecting up to 84% of the population at some point during a lifetime (1). This condition is a leading cause of disability with substantial social and economic burden (2). Although many patients respond to conservative approaches, a subset of patients develop refractory neuropathic pain syndromes, including failed back surgery syndrome or radiculopathy (3,4).

Spinal cord stimulation (SCS) has emerged as an effective neuromodulatory intervention for the management of chronic neuropathic LBP (5,6) . By delivering targeted electrical pulses to a dorsal root ganglion of the spinal cord, SCS modulates pain-gating pathways and reduces nociceptive input (7). The standard procedure involves a percutaneous trial phase to determine the device's efficacy before proceeding to permanent implantation. Patients who report at least a 50% reduction in pain from percutaneous SCS trials are determined to be suitable candidates for permanent implantation (8). While both trial and permanent implantation are considered minimally invasive interventions, they are nevertheless surgical procedures with inherent risks of active infection, uncontrolled systemic disease, such as diabetes, anatomical abnormalities, or even physiological stress responses.

Primary adrenal insufficiency (PAI), known as Addison's disease, presents unique perioperative challenges. PAI results from inadequate production of adrenal

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cortisol, most commonly from autoimmune destruction, congenital causes, or other pathological processes (9). These patients often require lifelong glucocorticoid replacement and are particularly vulnerable during periods of physiological stress, such as surgery, when cortisol demands are increased (10). Without appropriate glucocorticoid supplementation, PAI patients are at high risk for adrenal crisis, a potentially life-threatening condition leading to severe hypotension, electrolyte abnormalities, and acute abdominal symptoms (9). This risk is further amplified in those receiving chronic glucocorticoid therapy, which suppresses the hypothalamic-pituitary-adrenal (HPA) axis, reducing the capacity to mount an adequate stress response (11). Current endocrine and anesthesiology guidelines generally recommend perioperative stress-dose glucocorticoid supplementation for surgical cases; however, no guidance exists for interventional pain procedures (12,13).

Additionally, the spinal cord contributes to autonomic regulation of adrenal function. While data is derived from spinal cord injury literature, disruption of the HPA axis has been observed, sometimes causing either secondary adrenal insufficiency or, paradoxically, increased cortisol secretion and subsequent immunosuppression, raising theoretical concerns in patients undergoing SCS (14-16). Taken altogether, these aforementioned issues underscore the lack of clinical guidance and highlight the need for evidence-based protocols tailored to PAI patients undergoing an SCS trial and permanent implantation.

## CASE PRESENTATION

A 58-year-old woman with a history of seizures, PAI on long-term glucocorticoid replacement, and prior adrenal crisis after thyroidectomy presented to pain management with chronic LBP characterized by left-sided radicular features radiating posteriorly to her left foot associated with cramping and paresthesias. Symptoms worsened with standing and walking, which impaired activities of daily living, including transfers, grooming, and bathing. Her physical exam demonstrated positive lumbar facet loading tests and restricted flexion and extension. Her replacement regimen included fludrocortisone 0.1 mg once daily and hydrocortisone 15 mg every morning, followed by 5 mg twice daily.

Lumbar magnetic resonance imaging (MRI) revealed multilevel degenerative changes, including lumbar spondylosis, facet arthropathy, spinal stenosis with lumbar ligamentum flavum hypertrophy, and idiopathic levoscoliosis. Her pain was refractory to multiple conservative modalities (i.e., physical therapy, acupuncture, pilates), pharmacotherapies, including nonsteroidal anti-inflammatory drugs, antiepileptics, topical analgesics, opioids, and interventional treatments, including epidural steroid injections, which were limited due to her PAI. Although surgical intervention was considered, the patient opted for an SCS trial and subsequent permanent implantation.

During the SCS trial, perioperative management was tailored to the patient's PAI. She received a stress dose of 100 mg intravenous (IV) hydrocortisone one hour prior to the procedure to mitigate the risk of adrenal crisis, which successfully maintained hemodynamic stability initially. Although she developed brief intraoperative hypotension, this was rapidly resolved with 10 mg IV dexamethasone and a 500 mL saline bolus, and she remained stable subsequently. The epidural space was accessed at T12-L1 using a Tuohy needle with a paramedian approach; two 16-contact leads were advanced under fluoroscopic guidance to T9 and programmed using an external trial device, with needle removal confirming proper lead placement. During the trial, the patient experienced complete pain relief, improved daily function, increased activity, and reduced her gabapentin dose from 600 mg to 300 mg nightly.

Posttrial thoracic MRI was unremarkable for acute injury (Fig. 1).

Following the favorable trial, the patient underwent permanent SCS implantation 3 months later. Two percutaneous leads were placed at T9 under fluoroscopic guidance, and a pulse generator was successfully implanted (Fig. 2). The glucocorticoid protocol was intensified for surgery: she again received 100 mg IV hydrocortisone preoperatively, followed by 50 mg IV every 8 hours for 24 hours postprocedure. Her hydrocortisone was then tapered orally over 48 hours before resuming her baseline regimen. At 2 months postimplantation, she reported 90% pain relief, discontinued analgesics, and increased activity tolerance, including walking 1.5 miles daily and scooter use. She remained free of adrenal crises or hemodynamic instability.

## DISCUSSION

Our case is noteworthy as, to the authors' knowledge, it is among the first reports to detail the perioperative management of SCS in a patient with PAI. It highlights the absence of evidence-based protocols for perioperative steroid management during neuromodulation

Fig. 1. Sagittal MRI of the thoracic spine reveals multilevel degenerative spondylosis characterized by neuroforaminal narrowing, with no evidence of central canal stenosis. The spinal cord demonstrates preserved signal intensity, and an incidental vertebral hemangioma is identified at T10.
MRI T-spine 12/21/2024
Abbreviation: MRI, magnetic resonance imaging.

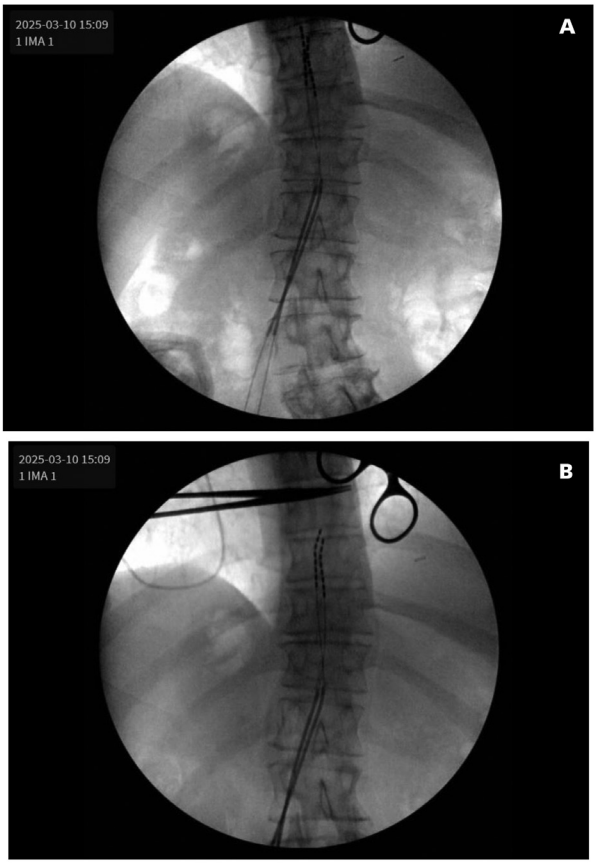


Fig. 2A-B. Fluoroscopic images showing dual-lead SCS placement at the T9 vertebral level: (A) during trial stimulation, and (B) at permanent implantation. Leads are positioned epidurally in a parallel configuration to optimize dorsal column coverage for neuromodulation.
XR Fluoroscopy 3/10/2025
Abbreviation: SCS, spinal cord stimulator.

procedures in this specific population, who are at increased risk for adrenal crisis. Our case addresses the challenge of balancing stress-dose glucocorticoid coverage to prevent an adrenal crisis without minimizing unnecessary steroid-related adverse effects in the context of SCS, a minimally invasive but physiologically stressful intervention.

Unlike open or major surgery, SCS is a minimally invasive procedure often performed percutaneously and on an outpatient basis (17). Although recovery is typically rapid and complications are fewer compared to open surgery, SCS still poses significant physiological stress, often underestimated for patients with underlying endocrine vulnerabilities, such as PAI (11-13,18). This procedural profile complicates perioperative risk assessment and often results in less standardized monitoring or steroid coverage than traditional open interventions. In addition to steroid management, clinicians should be aware of SCS-specific perioperative considerations, such as device programming, risk of electromagnetic interference, and the need for device interrogation and reactivation postprocedure (18).

Existing literature and perioperative guidelines on steroid management in PAI are based primarily on open and major surgical procedures. International guidelines (12,19) recommend administration of 100 mg of hydrocortisone at induction, followed by a continuous infusion of 200 mg hydrocortisone over 24 hours, then a postoperative oral glucocorticoid tapering regimen to baseline dosing within 48 hours. However, there is a lack of data or formal recommendations specific to interventional pain and neuromodulation procedures, such as SCS. As such, practice is extrapolated from major surgery or guided by expert consensus. The authors'

regimen in this case mirrored this approach, with the patient receiving 100 mg of hydrocortisone at induction, then 50 mg of hydrocortisone every 8 hours for 24 hours during inpatient hospitalization, followed by a 48-hour oral tapering regimen to return to baseline. This stepwise and vigilant steroid administration safeguarded against adrenal crisis and ensured the patient remained hemodynamically stable perioperatively and postoperatively compared to her trial. This adjustment was made to provide sufficient stress-dose coverage while minimizing potential steroid-related adverse effects, including hyperglycemia, impaired wound healing, or immunosuppression.

The pathophysiology of adrenal crises centers on the inability of patients with PAI to produce an appropriate cortisol response to physiological stress, resulting in symptoms ranging from hypotension to shock, abdominal pain, nausea, vomiting, and electrolyte abnormalities (hyponatremia, hyperkalemia) (10,20-22). The HPA axis plays a central role in endocrine homeostasis, with disruption reported in the setting of spinal cord injury. However, the impact of SCS on neuroendocrine function within the HPA axis remains incompletely understood and warrants systematic study, given there are no published studies assessing SCS effects on neuroendocrine regulation or adrenal crisis risk (9-11). Moreover, the symptoms of adrenal crisis in the perioperative setting may be subtle, easily misattributed to surgical recovery or medication effects of anesthesia, and therefore risk being missed.

Our case underscores the importance of multidisciplinary, individualized perioperative planning, close patient monitoring, and thorough patient education to mitigate risks and improve recognition of adrenal insufficiency in neuromodulation candidates. A practical approach for SCS candidates with adrenal insufficiency includes: preoperative endocrinology consultation, administration of stress-dose glucocorticoids tailored to both patient and procedure, close intraoperative and postoperative monitoring, prompt recognition of subtle signs of adrenal crisis, and thorough patient education (23). Further prospective studies are recommended to better define risk, management, and outcomes in this high-risk patient group to establish evidence-based protocols.

## CONCLUSIONS

Our case underscores the importance of individualized perioperative glucocorticoid management in patients with PAI undergoing SCS. In the absence of formal guidelines, tailored dosing and multidisciplinary coordination are essential to minimize adrenal crisis risk. A preoperative bolus followed by scheduled dosing and taper is recommended, mirroring general surgical principles but tailored to the patient's comorbidities and procedural invasiveness (24). Clinicians should maintain a high suspicion for adrenal insufficiency and provide thorough patient education regarding warning signs and crisis management. Further prospective studies are warranted to establish evidence-based protocols to guide best practices for neuromodulation in patients with complex endocrine disorders.

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