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Lumbar epidural steroid injection for spasticity in paraplegic spinal cord injury: A case report

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Abstract

This is the case of a 33-year-old male with traumatic paraplegic lumbar spinal cord injury after knife assault, who was unable to participate in an intensive inpatient rehabilitation course due to bilateral lower limb spasticity. For therapeutic management of spasticity at the bedside in the inpatient rehabilitation setting, we performed an epidural steroid injection to the right L4-L5 interspace. After the intervention, a significant decrease in spasticity was noted. The patient could subsequently tolerate sit-to-stand transfers with a standing frame and ambulate with the an exoskeleton device in inpatient physical therapy, significantly improving his overall functional level in therapies. This case demonstrates that bedside epidural steroid injection can dramatically improve paraplegic spasticity secondary to lumbar spinal cord injury in the inpatient rehabilitation setting.

Keywords

Muscle spasticity, paraplegia, spinal cord injuries, epidural steroid injection

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Introduction

Spasticity is a common complication of spinal cord injury (SCI). Seen in around 80% of SCI patients,[1] spasticity may progress to joint contracture if left untreated. Management includes physiotherapy, oral medication, botulinum toxin injection, and intrathecal infusion of baclofen.[2–5] In this report, we present a patient with painful lower extremity spasticity secondary to incomplete paraplegia whose mobility significantly improved after lumbar epidural steroid injection (ESI) and discuss bedside ESI as another treatment option to consider in the management of SCI spasticity.

Case report

A 33-year-old man with no significant past medical history was injured by a knife assault and unable to stand. He was initially admitted to an acute care community hospital, where a Computed Tomography (CT) scan demonstrated cervical cord transection with air inside the thoracolumbar spinal canal. Diagnosis of acute traumatic paraparesis secondary to thoracolumbar SCI was made. Subsequently, the patient was transferred to an inpatient rehabilitation facility (IRF).

Patient arrived with a neurological level of injury at T1, an American Spinal Injury Association Impairment Scale grade B, and knee flexor and extensor spasticity Modified Ashworth Scale (MAS) grade 3 on the right. He was previously independent in Activities of Daily Life (ADLs) and ambulation.

Three months after admission, patient still presented 8/10 pain on the numerical rating scale (NRS), radiating to the right posterolateral calf. Spasticity persisted in the bilateral lower extremities (BLEs), more on the right side (Figure 1). Decreased volitional movement and weakness persisted, severely interrupting physical therapy and sleep. Bed mobility was performed with minimal assistance for repositioning BLEs due to muscular hypertonicity. ADLs required moderate assistance. Transfers were performed with supervision secondary to BLE spasticity. Standing and ambulation could not be performed due to safety concerns from the SCI. Initial treatment with Baclofen 20 mg and Gabapentin 300 mg three times daily provided minimal relief.

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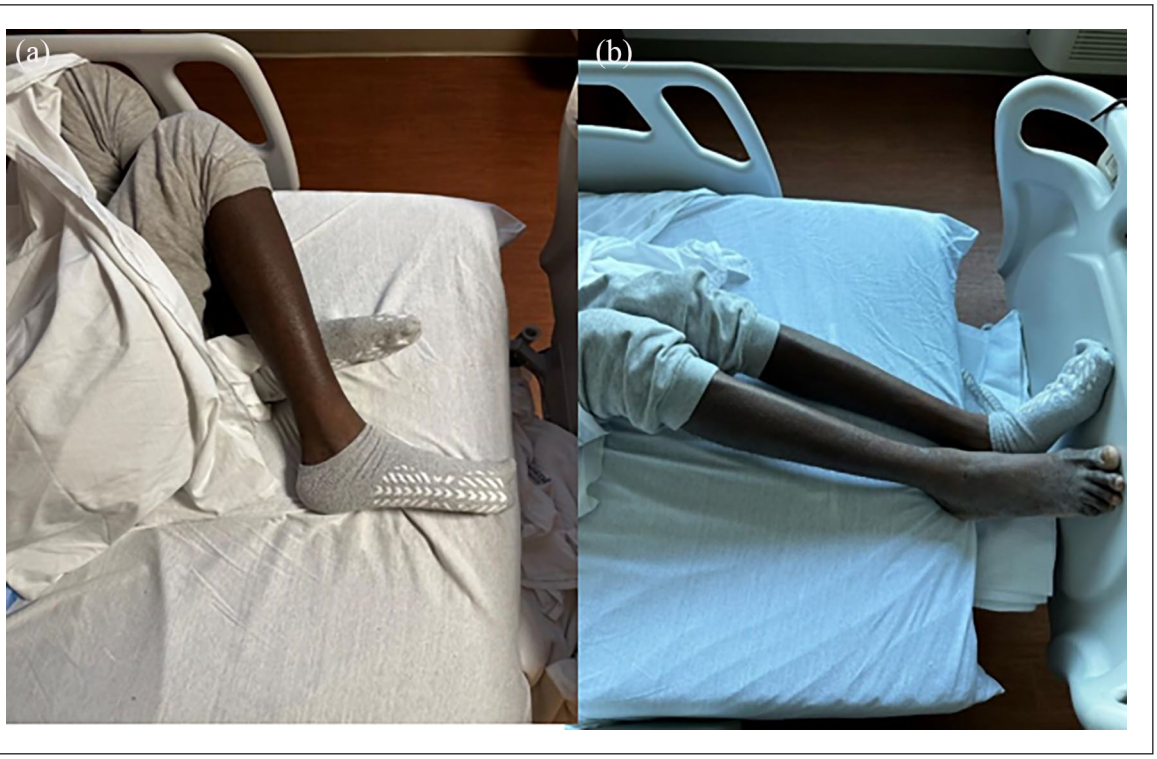

Figure 1. (a) and (b) Resting posture of spastic bilateral right worse than left lower extremities before the first bedside epidural steroid injection (left) versus 2 days after (right).

Therefore, bedside L4-L5 ESI for therapeutic purposes was scheduled, and written informed consent was obtained from the patient for ESI to be performed for spasticity at bedside without imaging guidance. The L4-5 level was chosen for the initial ESI due to L3-5 involvement of air inside the thoracolumbar spinal canal upon initial imaging in the acute care setting. These neurological levels appeared to manifest in reciprocal quadriceps weakening and hamstring spasticity, consistent with the patient's BLE clinical picture. ESI was performed by maintaining a sterile technique and cleansing the area with iodine. 9 cc of bupivacaine and 1 cc of methylprednisolone 40 mg were administered using a 22 g spinal needle. While 1 to 2 cc of local anesthesia are typically used for pain management, 9 cc of bupivacaine were used for this ESI. Based on prior experience by the attending neurorehabilitation physician, this relatively large amount of bupivacaine with 1 cc of methylprednisolone is the best protocol to confer the most benefit for reduction of painful spasticity in the inpatient rehabilitation setting.

Two days after the intervention, right knee flexor, and extensor spasticity subsequently decreased from 3 to 1+ on MAS (Figure 1), pain intensity decreased from 8/10 to 3/10 on NRS, and radiating pain decreased to the posterolateral calf from the right buttock as well. The etiology of the radiating leg pain is presumed to be related to the air inside the thoracolumbar spinal canal with the involvement of the L3-5 levels. Hospital course following ESI was complicated by mild intermittent clonus of the bilateral feet, right worse than left, along with constipation and urinary tract infection (UTI). Symptoms subsided with scheduled suppositories, constipation medications, and antibiotics.

Most importantly, the patient could perform gait training actively with significant improvements to BLE spasticity. Three days following intervention, the patient tolerated standing frame-assisted sit-to-stand transfers during occupational therapy (OT) for 15 min, with occasional symptoms of dizziness. He also demonstrated increased standing balance, evidenced by participating in coordination with bilateral upper extremities unsupported. One week following the initial ESI, the patient could actively flex his bilateral hips against gravity by 25°, compared to 10° before the intervention.

The patient's therapy participation would decrease for the next several months following the initial beneficial response to the first ESI. BLE spasticity returned to his pre-injection severity. Resting with both legs in significant flexion while lying in bed, he was unable to participate in sit-to-stand (STS) transfers or ambulate in therapies once again. After 4 months, a botulinum injection to the right lower extremity (RLE) was administered, but with less response than the ESI. Patient received three ESIs at three, seven, and twelve months after admission, with the last administered above the previous two at the right L3-4 level instead of L4-5. The three ESIs were administered more than 3 months apart, each with approximately 4 weeks of significant but temporary relief.

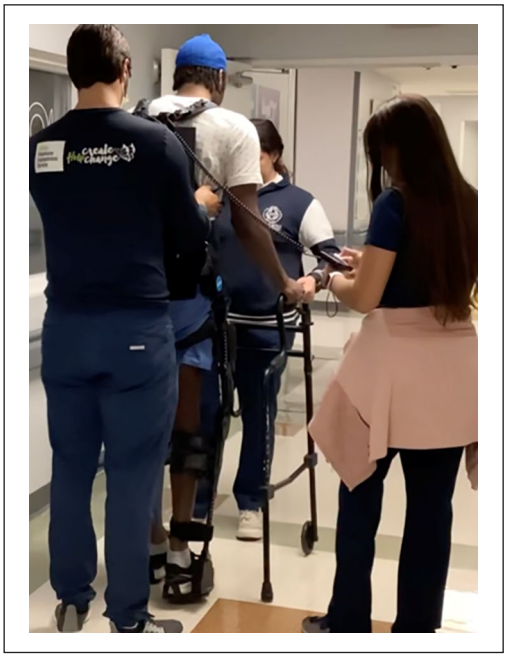

Figure 2. Participation in exoskeleton ambulation improved during therapies 1 week following the second bedside epidural steroid injection.

Following each ESI, radicular pain, spasticity, and sleep disturbance decreased, and participation in gait training improved accordingly. Notably, the patient ambulated with the EKSO exoskeleton for 12–13 min following the second ESI (Figure 2) and walked 367 steps without rest during therapy within 1 month following the third ESI. Relief dissipated gradually, but pain and spasticity improved significantly for approximately 1 month on average.

Discussion

If left untreated, spasticity after SCI can progress toward structural contracture as well as lead to complications with deep tissue pressure injuries, loss of range of motion, sleep disorders, and pain.[6] These complications from spasticity can impair functional participation in therapies and ADLs. ESI is an option in painful localized refractory spasticity and can improve functional outcomes accordingly.[7] This is because spasticity is known to result from noxious stimuli, whether intrinsic or extrinsic, below the level of the injury.[1] While still unclear, the etiology of SCI spasticity is thought to result from hyperexcitability of the spinal reflex.[2] The mechanisms of improvements in pain and spasticity from ESI after SCI seem related to radiculopathy as an extrinsic stimulus alleviated by the steroid.[2] ESI was chosen in this patient for this reason, as thoracolumbar radiculopathy was thought to be extrinsically stimulating the spinal reflex into BLE spasticity with good potential for relief of the hyperexcitability by the anti-inflammatory effects of the steroid.

This case report demonstrates that bilateral asymmetric SCI painful spasticity could improve from lumbar spine intervention at the bedside in the IRF setting. The patient in this report received three lumbar epidural injections administered to the L4-L5 and the L3-L4 levels more than 3 months apart, with temporary improvement to spasticity lasting around a month following each injection, increasing participation in therapies before returning to baseline spasticity. Patients in similar settings may benefit from the improvement in pain, strength, proprioception, and stability that each ESI confers.

Adjunct therapies and modalities should be scheduled around ESI to produce more long-term improvements in outcomes. Exoskeleton ambulation was beneficial in this patient, for instance, but only made possible when strategically scheduled immediately after ESI alleviated BLE spasticity (Figure 2). Temporary benefits of approximately a month following each intervention could be maximized by planning therapies accordingly.[8] One limitation of this study is the lack of long-term follow-up to determine if the multiple ESI therapies afforded any long-term benefit. Another limitation is that thoracic SCI with lower extremity spasticity, as in this case report, may present atypically and be more closely related to poor localization of pain instead of reflecting proper ASIA SCI deficit. Finally, the authors acknowledge the limitation of a single case report, which would benefit from pairing with randomized and double-blinded controlled studies comparing ESI with other spasticity management techniques in SCI, such as physiotherapy, oral medication, botulinum toxin injection, and intrathecal infusion of baclofen.

Conclusion

Overall, this case illustrates that ESI can significantly improve functional outcomes in SCI with BLE spasticity. If suspected, SCI-related spasticity may benefit from bedside intervention with ESI, and a series of repeat injections may also be indicated for functional improvement in the IRF setting over time. We recommend bedside ESI for diagnostic and therapeutic benefit to SCI patients with intractable lower extremity spasticity in the IRF setting. Although ESI relief is temporary, planning adjunct therapies strategically in the timeframe of maximum benefit to spasticity may improve long-term functional outcomes.

Author contributions

Author contributions are as follows: M.A. Writing—original draft; K.N. Supervision, Writing—original draft; R.A.R. Writing—review & editing.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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