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Selective Unilateral L5 Neuropathy Subsequent to Intrathecal Drug Delivery System Implant: A Case Report

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Background: Intrathecal drug delivery systems (IDDS) offer targeted pain relief with lower drug doses but come with risks. Our report describes a unilateral L5 neuropathy following an uncomplicated IDDS implant.

Case Report: A 45-year-old man with chronic back pain and prior L4-L5 laminectomy presented with severe pain after a fall. Imaging was unremarkable, and he was deemed a candidate for IDDS implantation. The procedure, which included catheter placement to the T6-T7 level and morphine pump insertion, was initially well tolerated without immediate complications. On postoperative day one, he developed right foot weakness and numbness. Magnetic resonance imaging showed no mechanical etiologies, and conservative management was recommended. By postoperative day 10, his symptoms had completely resolved.

Conclusions: Our case highlights the importance of early recognition of IDDS complications for effective management and prevention.

Key words: Unilateral neuropathy, L5 neuropathy, intrathecal pump, postoperative, case report

BACKGROUND

Chronic low back pain causes a significant health care burden, with an estimated 70% of individuals in developed countries being affected at some point in their life (1). Management of this condition typically involves oral analgesics, such as nonsteroidal anti-inflammatory drugs (NSAIDs), with some patients requiring opiates to manage their pain (2). Long-term use of these drugs can lead to adverse effects and tolerance, with patients requiring increasing dosages to alleviate their symptoms. Further, 10% to 30% of patients are unable to achieve adequate pain control with oral analgesics, and they require more invasive means of pain management (3).

Intrathecal drug delivery systems (IDDS) deliver drugs, such as opiates, directly to the intrathecal space, leading to more effective analgesia at lower drug concentrations while decreasing the amount of opiate metabolites (3). These devices consist of a pump with a reservoir, typically implanted just beneath the skin, connected to a catheter positioned within the intrathecal space (2). Most commonly, these systems are used to achieve analgesia in patients with conditions, such as spinal stenosis, spondylolisthesis, chronic regional pain syndrome, and other spine disorders.

Although these systems are effective in reducing pain burden for patients with chronic back pain, their implantation and use are not without risks. Risks associated with intrathecal pain pumps include infection, localized bleeding, dosing inaccuracies, cerebrospinal fluid (CSF) leakage, meningitis, device malfunction, and the development of hygromas or granulomas (4). Infections related to the pump may also compromise neurological function by causing inflammation and disrupting spinal cord integrity (4). Further, catheter-

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related complications, such as migration or inadequate placement, may damage nearby structures and lead to neurological deficits (3). In the following report, we outline a case of unilateral L5 nerve root neuropathy after an uncomplicated IDDS implant. The patient provided informed consent to be included in this case report.

CASE REPORT

A 45-year-old man with a history of chronic kidney disease, chronic neuropathic pain involving the lower back and bilateral lower extremities, and a prior L4-L5 laminectomy, in 2022, presented to the emergency department 2 days after sustaining a fall onto his buttocks and sacrum. Following the fall, he reported severe pain rated 10/10 on the numeric rating scale, unrelieved by multiple doses of intravenous narcotics. X-ray of the thoracic and lumbar spine revealed no acute or significant abnormalities (Figs. 1 and 2). Magnetic resonance imaging (MRI) of the thoracic and lumbar spine found no significant spinal canal or foraminal stenosis.

The patient was admitted for observation and evaluated by pain management, who deemed him an appropriate candidate for IDDS implantation due to his failed back surgery syndrome and chronic neuropathic pain. His pain has been refractory to physical therapy, NSAIDs, neuropathic pain medications, acupuncture, opioids, oral corticosteroids, and epidural steroid injections. After consultation, a 24-hour intrathecal opioid trial was performed, and the patient reported > 50% pain relief.

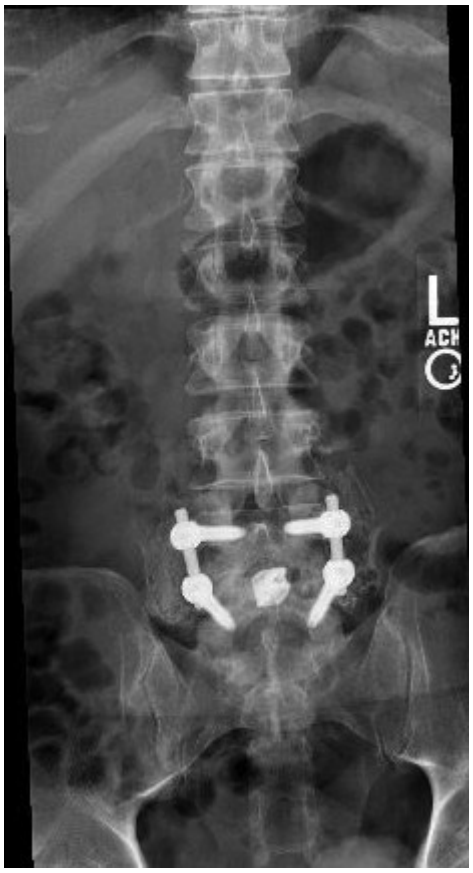

Fig. 1. AP x-ray thoracic and lumbar spine. AP, anteroposterior.

The procedure involved insertion of a 14G needle into the L3-L4 interlaminar space under fluoroscopic guidance and advancing the intrathecal catheter with the tip reaching the T6-T7 level. A subcutaneous pump reservoir filled with morphine (1 mg/mL, 20 mL) was implanted in the right abdominal quadrant and connected to the catheter via subcutaneous tunneling. The patient tolerated the procedure well and experienced no immediate neurological deficits following the procedure. Final device programming and testing were completed in the recovery area.

On postoperative day one following the IDDS implantation, the patient developed sudden-onset right lower extremity weakness and numbness despite a normal neurological exam immediately following the procedure. By the following morning, the patient reported inability to dorsiflex the right foot or bear weight on the right side. Physical examination was significant for 1/5 motor strength in right ankle dorsiflexion with sensory

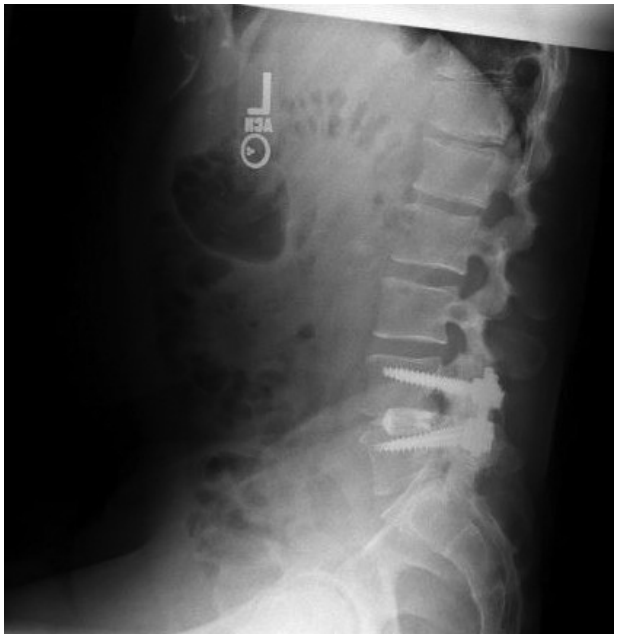

Fig. 2. Lateral x-ray lumbar spine.

loss in the L4-L5 dermatomes. Reflexes were intact, and no other extremity weakness was noted. The surgical sites were clean and intact without signs of infection, hematoma, or ecchymosis. The patient denied bowel or bladder dysfunction or saddle anesthesia.

The patient underwent an MRI of the lumbar spine (Fig. 3), which revealed stable postsurgical changes at L4-L5 with no evidence of spinal cord compression, disc herniation, or significant foraminal or central canal stenosis.

The patient was advised to proceed with conservative management of cold compress therapy, mobility support with a walker, and a home exercise program. The patient was discharged with instructions for outpatient neurology follow-up. On postoperative day 10, the patient's neurological symptoms had fully resolved.

On postoperative day 11, the patient returned to the clinic with complaints of a progressively worsening headache that had begun several days earlier. He described a significant exacerbation of pain when sitting or standing. He denied any associated symptoms, such as weakness, numbness, or visual disturbances. A physical examination was performed, and the risks, benefits, and potential complications of a lumbar epidural blood patch for treatment of a postdural puncture headache were thoroughly discussed. The patient consented to proceed with the procedure the same day.

A lumbar interlaminar epidural blood patch injection was performed at the L3-L4 level using an 18G, 2.5-inch Tuohy needle. A total of 15 mL of autologous blood, drawn from the patient's right antecubital vein, was slowly injected. The procedure was completed without complications and was well tolerated. The patient was observed postprocedure and reported no new neurological symptoms.

DISCUSSION

Unilateral L5 neuropathy is a rare complication following IDDS implantation. Given the absence of a structural/compressive pathology elicited on MRI, this must raise awareness toward potential nonmechanical postoperative complications. Thus, the neuropathy may be attributed to postoperative neuritis, localized neurapraxia, or chemical radiculitis involving the L5 nerve root. Although IDDS is a modality considered after failing more conservative treatments, the patient has trialed several modalities over several years without success. The acute on chronic pain with reduction of pain after the intrathecal opioid trial deemed the patient suitable for IDDS implantation. The L2-L3 interlaminar space is usually preferable in a patient with an L4-L5 laminectomy due to the risks of CSF leakage secondary to prior decompression, scarring, and risk of unroofing from the previous surgical site. However, the L3-L4 approach had more favorable anatomy under fluoroscopic guidance, showcasing more preserved epidural space compared to the L2-L3 space, which was more calcified and narrowed, preventing safe access.

Postoperative neuritis is a local inflammatory response of the nerve root during the surgical procedure or attributed to catheter tunneling that can lead to temporary dysfunction. Common causes of perioperative neuropathy are usually attributed to surgical trauma, intraoperative patient positioning, or regional anesthesia/nerve blocks. More uncommon causes of neuropathy may be less likely to be considered by surgery providers and anesthesia (5,6). Risk factors for postoperative inflammatory neuropathy have been associated with men, obesity, diabetes mellitus, peripheral vascular diseases, older age, alcohol and tobacco usage, arthritis, genetic predisposition, preexisting neuropathy, surgical technique, anesthetic used, and recent blood transfusions (7). Early recognition and treatment with corticosteroids in the acute phase can reduce the severity and duration of the symptoms (5).

Localized neurapraxia is the mildest form of nerve

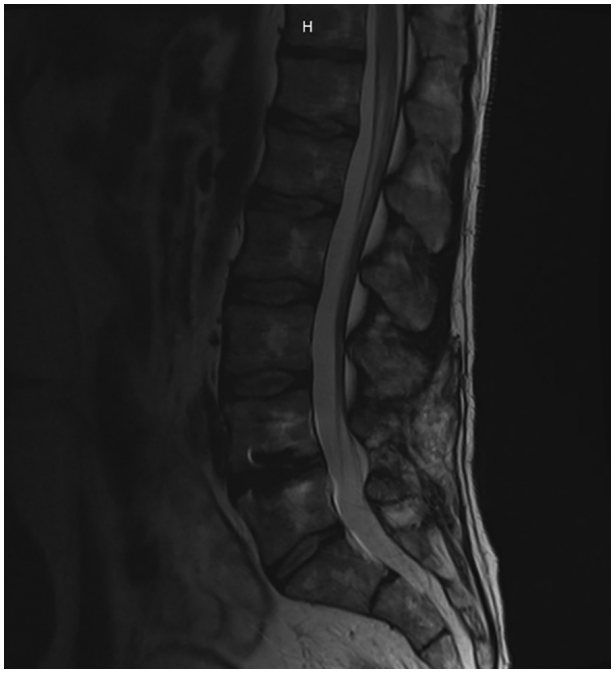

Fig. 3. MRI of lumbar spine. MRI, magnetic resonance imaging.

injury commonly caused by ischemia or demyelination, leading to temporary conduction blockage (8). During IDDS, difficult navigation using the catheter or anchor placement through fibrotic or challenging tissue planes may lead to localized neurapraxia.

Although the least likely of these 3 proposed etiologies, chemical neuritis may occur due to leakage or misplaced intrathecal medication, which can lead to a neurotoxic inflammation of the nerve root. The chemical neuritis theory was first put forth by Marshall et al in 1977, where a tear in the annulus fibrosus can leak material from the nucleus pulposus that irritates the nerve root (9). It is interpreted that electrophysiology is an ectopic eruption of an impulse from a nerve root, with animal studies demonstrating prostaglandin E2 and tumor necrosis factor-α being able to provoke this response, suggesting their roles in nerve root sensitization (9). Thus, chemical neuritis can be described as nerve root irritation from chemical mediators. Although unenhanced MRI may not find a pathology, T2-weighted MRI may demonstrate a hyperintense zone representing the annular tear (10). Periannular enhancement is caused by the inflammatory breakdown of the vessel wall barrier by proinflammatory cytokines. Therefore, if an MRI is negative for disc herniation or spinal stenosis but shows a hyperintense zone, clinicians can have a higher index of suspicion for chemical radiculitis (10).

We emphasize the importance of recognizing and managing nonmechanical causes of postoperative neuropathy symptoms. This is important to be aware of, even if the procedure was completed without complications. Patients presenting postoperatively with delayed or isolated neuropathic symptoms should have a high index of suspicion for both mechanical and nonmechanical (inflammatory/chemical) etiologies. Neurology consultation, along with electromyography/nerve conduction studies, with possible nerve biopsy and corticosteroid treatment, can help diagnose and manage the postoperative neuropathy.

Limitation

The main limitation of this study, inherent to a case report, is data only on one patient. Further long-term studies evaluating complications of IDDS, such as randomized controlled trials, observational studies, retrospective studies, and real-world evidence, can better characterize this risk and the causes of postoperative neuropathy.

CONCLUSIONS

IDDS implantation is a generally safe and well-tolerated procedure, but rare postoperative complications may occur. Clinicians must consider noncompressive etiologies, such as neuritis, neurapraxia, and chemical radiculitis, given a negative MRI. Timely diagnosis and management are essential to minimize nerve root irritation and help inform perioperative strategies to minimize this risk.

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