

◆ Case Report

Paralysis After Transforaminal Epidural Injection and Previous Spinal Surgery

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Objective: This case discusses a rare but devastating complication of transforaminal epidural injection. Elements in the patient's history that may be risk factors are discussed.

Case Report: A 64-year-old man was evaluated for chronic low-back pain after multiple spine surgeries. After the most recent surgery, he suffered transient cauda equina symptoms. Because conservative therapy was not helpful for spinal stenosis and neuroclaudication, a left L2 transforaminal epidural injection was attempted, but a posterolateral fusion mass made this procedure impossible. A left L1 transforaminal approach was successful, and 1 mL of iopamidol (Isovue) contrast was injected, followed by 5 mL of a solution of 0.125% bupivacaine and 40 mg of triamcinolone. Approximately 1 to 2 minutes after injection, the patient described discomfort in the lower abdomen, and 1 minute later, he was unable to move his lower extremities. An MRI showed T2 signal change in the conus medullaris gray matter at T11-12, consistent with an acute vascular infarct. Spinal shock protocol with high-dose methylprednisolone was begun without change. More than 4 years later, the patient continues to be troubled by persistent paraparesis and chronic pain.

Conclusions: This case report is part of a new and growing body of literature that demonstrates the potential risk of transforaminal injection. Further study is necessary to ensure that spinal vascular injuries can be kept to an acceptably rare level. *Reg Anesth Pain Med* 2004;29:494-495.

Key Words: Complications, Epidural, Risks, Spinal-cord injury, Steroid injection, Transforaminal.

Epidural corticosteroid injections are advocated as conservative therapy for patients with radicular pain syndromes.¹ Recent case reports of postprocedural spinal vascular events that resulted in paraplegia^{2,3} have brought into question the overall safety of the transforaminal epidural approach. We report a case in which a patient with prior extensive spinal surgery and remote cauda equina symptoms suffered an acute spinal cord infarct during transforaminal injection of corticosteroid for the treatment of worsening neuroclaudication from spinal stenosis.

Case Report

A 64-year-old man presented to the clinic with a 1-month history of worsening chronic low-back

pain. The pain was constant, exacerbated by walking or sitting, and radiated into the left buttock and groin. Opioid analgesics provided minimal relief. Past medical history included hypertension and 4 spinal surgeries. The most recent surgery was an L2 fusion 15 years before evaluation. Immediately after that surgery, he suffered cauda equina symptoms, with loss of bowel and bladder control, erectile dysfunction, and lower-extremity weakness. Two months after the L2 fusion the patient experienced a complete return of ambulation and bowel and bladder function, but erectile dysfunction and mild weakness persisted.

Physical examination showed mildly weak plantar and dorsiflexors, negative straight-leg raise, absence of palpation tenderness, and absent Achilles reflexes bilaterally. Patchy numbness was present in both lower extremities since his prior surgery. An MRI showed old T12 and L4 compression fractures, a subacute L1 compression fracture, moderately severe spinal stenosis at L1-2, and solid fusion mass from L2 to L5, with wide laminectomies at each level. In view of the patient's left leg pain and neuroclaudication and the MRI finding of L1-2 spinal stenosis, a left L2 transforaminal epidural steroid injection was attempted, but a posterolateral fusion mass made this procedure impossible.

Attempts with 25-gauge and 22-gauge, 3.5-inch Quincke needles were redirected to the L1 foramen, which was entered without incident. Injection of 1 mL of nonionic iopamidol (Isovue) contrast dem-

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onstrated excellent spread along the L1 nerve root, as well as into the epidural space, without evidence of vascular or intrathecal spread. Approximately 5 mL of a solution of 0.125% bupivacaine and 40 mg of triamcinolone were injected slowly. Approximately 1 to 2 minutes after injection, the patient described discomfort in the lower abdomen. Light touch sensation in the lower extremities was intact, however 1 minute later, he was unable to move his lower extremities. He was transferred back to his bed, and further sensory testing revealed a T10 level. He was monitored for 1.5 hours, and beginning resolution of a presumed subarachnoid block was documented based on the return of some sensation and some movement of his right leg. Over the ensuing several hours, he continued to manifest persistent paresis and sensory loss, as well as urinary incontinence. An MRI performed 18 hours after the procedure showed T2 signal change in the conus medullaris gray matter at T11-12, which was thought to be consistent with an acute vascular infarct. Spinal shock protocol with high-dose methylprednisolone was begun without change. More than 4 years later, the patient continues to be troubled by persistent paraparesis and chronic pain. The patient's current pain is disabling, dysesthetic, and spontaneous and more consistent with central pain than was his previous neuroclaudicatory pain.

Discussion

Anterior spinal-cord blood supply is through the single anterior spinal artery. Radicular feeding arteries arise from lumbar and intercostal arteries to help supply the anterior cord. The artery of Adamciewicz normally emanates from T5 to L2 segments but may have a high thoracic takeoff, and in that case, enlargement of an iliac-derived radiculomedullary artery may enter a single foramen at sites as caudad as S1.^{2,4} A recent case report revealed the inability of conventional contrast dye and fluoroscopy to visualize injection of a spinal cord-feeding radicular artery.⁵

Possible explanations for the injury to this patient include direct injury to the artery of Adamciewicz or radicular arteries, vasospasm caused by either the particulate steroid or the bupivacaine, end-capillary occlusion by steroid particles, or needle-related factors (volume deformation or postcontrast needle movement).⁵ The severity of these recently reported adverse events after transforaminal injection indicate the need for identification of risk factors and safety guidelines. In our patient, the risk factor may have been that an area of vulnerability

to ischemia persisted after his L2 fusion 15 years before.

In a series of 3 patients with anterior spinal-cord infarcts after transforaminal epidural injection, all had prior spine surgery.² These cases suggest that left-sided injections above L3 should be approached with particular caution in patients with extensive prior surgery or, as in our patient, previous cauda equina symptoms.

To date, no controlled studies that compared transforaminal and interlaminar epidural corticosteroid injections have been performed. This comparison is necessary, as the type and frequency of complications may differ. Further controlled studies are required to characterize the risks of these procedures in patients with prior surgery, better characterize the anatomy, or better define and protect arterial structures before injection. All injections carry risks, but if the risk of vascular infarct is higher in the transforaminal approach, and in the absence of compelling comparative efficacy studies, concerns for patient safety must be paramount.

In conclusion, we present a case of acute spinal-cord infarction occurring in a patient immediately after an L1 transforaminal epidural steroid injection. At the time our patient underwent this injection, no published reports documented similar complications. However, this case report is part of a new and growing body of literature highlighting the potential risk of this technique. Further study is necessary to ensure that spinal vascular injuries can be kept to an acceptably rare level.

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