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Conus medullaris syndrome after epidural steroid injection: Case report

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Abstract

Background: Given the risk of paralysis associated with cervical transforaminal injection, is it time to reconsider transforaminal injections of the lumbar spine? Arguments for discontinuing lumbar injections have been discussed in the anesthesia literature, raising concern about the risks of epidural steroid injections (ESIs).

Methods: In a 47-year-old man, paraplegia of the lower extremities developed, specifically conus medullaris syndrome, after he underwent an ESI for recurrent pain. Correct needle placement was verified with epidurography. Immediately after the injection, the patient felt his legs “going dead”; paraplegia of the lower extremities was noted.

Results: An initial magnetic resonance imaging study performed after the patient was transferred to the emergency department was unremarkable. However, a later neurosurgical evaluation showed conus medullaris syndrome, and a second magnetic resonance imaging study showed the conus infarct. We conducted a search of the PubMed database of articles from 2002 to 2011 containing the following keywords: complications, lumbar epidural steroid injection(s), cauda equina syndrome, conus medullaris infarction, spinal cord infarction, spinal cord injury, paralysis, paresis, plegia, paresthesia, and anesthesia.

Conclusions: Summarizing this case and 5 similar cases, we weigh the potential benefits and risks of ESI. Although one can safely assume that this severe, devastating complication is rare, we speculate that its true incidence remains unknown, possibly because of medicolegal implications. We believe that the rarity of this complication should not preclude the continued use of transforaminal ESI; rather, it should be emphasized for discussion with patients during the consent process.

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Keywords: Conus medullaris syndrome; Epidural; Injections; Polyradiculopathy; Steroids

Low-back pain is extremely prevalent; it affects an estimated 58% of the population.^{1,2} Inflammation of the nerve roots can lead to leg and low-back pain. Patients with lumbar spine pain can be treated with epidural steroid injection (ESI) through a transaminar or transforaminal approach. The benefits and risks related to ESIs in the cervical and lumbar spine, through either approach, are well documented.^{3–10} Complications include headache, increased back or leg pain, facial flushing, vagal responses, and procedural hypertension. Serious complications include epidural abscesses, paralysis, nerve damage, hematomas, hypersensitivity reactions, and conus medullaris syndrome.^{11–18} Complications related to cervical spine injections may pro-

duce irreversible paralysis.¹⁹ Botwin et al.⁹ reported an overall complication rate of 9.6% among patients who underwent transforaminal epidural injections, most often non-positional headache; no patient included in the review had conus medullaris syndrome as a result of ESI.

Our case report describes a patient in whom conus medullaris syndrome developed after undergoing a transforaminal lumbar steroid injection for lumbar pain, and we review the literature, including 5 other cases of paralysis after ESI (Table 1). Given the contention that ESIs in the cervical spine have the potential to cause devastating neurologic damage, we present our case and summarize the published evidence to further the discussion regarding use of ESI in the lumbar spine.

Case report

A 47-year-old man who had undergone an L4/L5 laminectomy and discectomy 5 years earlier presented with

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Table 1
Reported cases of cauda equina syndrome after ESI

Patient No.	Sex, age (y)	Intervertebral foramen injected	Aspiration performed	Epidurography performed	Computed tomography guidance used	Outcome
1	Female, 64 (Botwin et al. ⁹)	L3-4 and L4-5	Yes	Yes	No	Improvement from 3 of 5 bilaterally to 4 of 5 bilaterally at 1-mo follow-up
2	Female, 51 (Botwin et al. ⁹)	L3-4	Yes	No	Yes	No recovery of neurologic function at 8-mo follow-up
3	Male, 42 (Botwin et al. ⁹)	S1	Yes	No	Yes	No recovery of neurologic function at 5-y follow-up
4	Female, 71 (Florey ²⁶)	L2-3	Unknown	No	Yes	Improvement from 0 of 5 to 2 of 5 in L3 myotome but persistent dissociated sensory loss bilaterally at 6-wk follow-up
5	Male, 64 (Lutz et al. ¹⁰)	L1-2	Unknown	Yes	No	Persistent paraparesis and chronic pain
6	Male, 47 (current study)	L4-5	Yes	Yes	No	Improvement in strength from 0 of 5 to 4 of 5 in L5 myotome but persistent perineal numbness and urinary retention at 1-mo follow-up

recurrent pain in the left buttock and posterior leg. The patient chose conservative management by ESI at another institution. After receiving an ESI by a local anesthesiology group within the Cincinnati, Ohio, area, the patient was transferred from the outpatient pain clinic to our institution. Information regarding his medical history and prior treatment, as well as details of the procedure, was limited to that provided by the patient himself or the accompanying procedure report. The patient reported that he had previously received ESIs on the left side of his lower back; this was the second injection in a series of 3. A copy of the fluoroscopic image was neither provided nor available from the physician. Correct needle placement in the left L5-S1 neural foramen was verified with epidurography.

Immediately after the injection, the patient felt his legs “going dead”; paraplegia of the lower extremities was

noted. Because of the concern for intrathecal injection with resultant motor blockade, the patient was monitored for 4 hours. When no clinical improvement was observed, he was transferred to a nearby community hospital emergency department for neurologic examination. Although the initial magnetic resonance imaging (MRI) study performed in the emergency department was unremarkable (Fig. 1), a later neurosurgical evaluation showed conus medullaris syndrome. However, a second MRI study to evaluate the possibility of vascular complications obtained 48 hours after the injection showed a conus infarct (Fig. 2).

At 1-month follow-up, the patient could walk without assistance with a slow and calculated gait, had symmetric lower-extremity strength, and had dorsiflexion strength of 4 of 5 bilaterally. He reported persistent perineal numbness and the need for straight catheterization to void. His urinary

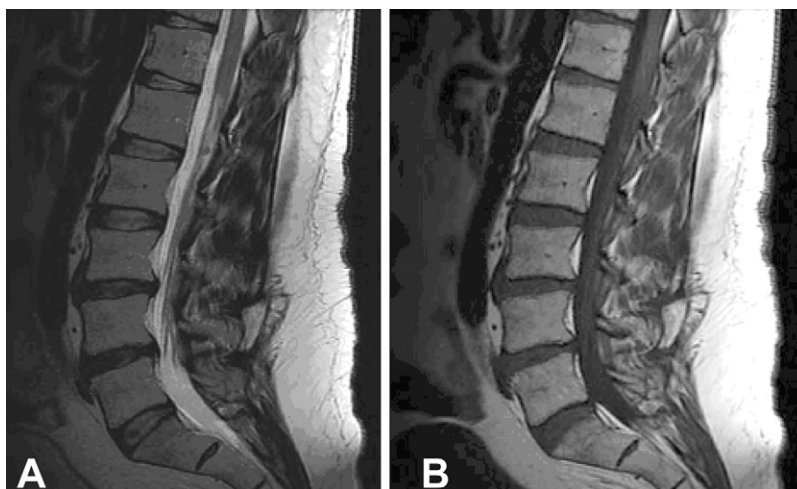


Fig. 1. Five hours after ESI in a 47-year-old man, magnetic resonance imaging scans performed at the emergency department were unremarkable, showing a normal appearing conus. (A) T2-weighted sagittal image (repetition time, 3516.7; echo time, 112.0). (B) T1-weighted sagittal image (repetition time, 416.7; echo time, 15.0).



Fig. 2. Magnetic resonance images (about 48 hours after the procedure) showing extensive signal abnormalities within the lower thoracic spinal cord and conus compatible with the clinical diagnosis of conus infarct. (A) T1-weighted sagittal image (repetition time, 675.0; echo time, 9.6). (B) T2-weighted sagittal image (repetition time, 3640.0; echo time, 102.0). (C) Short tau inversion recovery (STIR) sagittal image (repetition time, 4000.0; echo time, 58.0).

urge sensation had returned, and occasional episodes of fecal incontinence occurred in relation to bladder over-distention. He was able to have an erection but could not achieve orgasm.

Literature review

Our search of the PubMed database (2002–2011) included the keywords complications, lumbar epidural steroid injection(s), cauda equina syndrome, conus medullaris infarction, spinal cord infarction, spinal cord injury, paralysis, paresis, plegia, paresthesia, and anesthesia.

Discussion

ESI is regarded as an effective and conservative means of treating low-back pain resulting from nerve root inflammation. Recent case reports of post-procedural conus medullaris syndrome after spinal vascular compromise raise the question regarding safety.^{16–18} Our case represents a sixth patient in whom conus medullaris syndrome developed after sustaining an acute spinal cord infarct during ESI. In 5 other case reports (Table 1), this complication affected patients (aged 42–71 years) who had previous lumbar surgery and then underwent a transforaminal steroid injection.^{16–18} Of note, findings of spinal cord injury were not detectable on the initial MRI study within the first 24 hours in 1 case.¹⁷ Permanent paraplegia with loss of sphincter tone was observed in 1 patient.¹⁸ Outcomes in the remaining 4 patients varied from complete to partial recovery.

Injections are typically evaluated first by aspiration and injection of contrast. A nonvascular penetrating injection of contrast media verifies safe needle placement. However, in a series of 761 lumbosacral transforaminal injections performed, Furman et al.²⁰ reported that the sensitivity of positive flash or actual aspiration was only 44.7%, because only 38 of 85 patients had shown either flash or actual aspiration before injection. Specifically, they showed that there was an 11.2% rate of vascular injection in their series. In evaluating correct placement of 316 caudal-approach ESIs, Renfrew et al.²¹ implicated aspiration as an error-prone method for verifying needle placement, reporting a 9.2% incidence of vascular violation despite negative findings on aspiration.

Blood is supplied to the caudal-most portion of the spinal cord by the anterior spinal artery, 2 posterior spinal arteries, segmental radiculomedullary arterial branches, and most importantly, the artery of Adamkiewicz.²² The location of the artery of Adamkiewicz, which is the primary blood supply to the conus medullaris, is fairly unpredictable. It travels with the nerve through the foramen at or near the level of its origin from the thoracolumbar segmental arteries.²³ Lazorthes et al.²³ showed that the artery of Adamkiewicz originated from the left T9–12 posterior intercostal arteries in 75% of cases and from the L1–2 lumbar arteries in 10% of cases. In 31 cadavers Biglioli et al.²⁴ located this

artery between T12 and L3 in 26 cases (83.9%). In a 2002 review of more than 4000 spinal angiograms, Lo et al.²⁵ examined the variability of the artery of Adamkiewicz, noting that it originated at L2 in 1% of cases and L4 in 0.075% of cases.

Of the proposed mechanisms by which spinal cord medullary infarction may occur, one explanation may be the combined effect of an undetected direct arterial injection into a low-lying artery of Adamkiewicz and the resultant embolic incident from the injectate. Houten and Errico¹⁶ proposed that the collaterals surrounding the cord at the level of this artery were proximal to the injection site and thus allowed direct passage of the injected material into the conus. However, as discussed by Lo et al.,²⁵ the occurrence of a low-lying artery of Adamkiewicz is extremely unlikely. Considering this observation, we believe that it is unlikely that our patient had this low-lying artery that could have then been injected with an epidural steroid at this precise location.

Another plausible explanation is that an inadvertent sacral radicular artery injection carried injection material distally to the spinal cord. Lazorthes et al.²³ reported that material injected into the abdominal aorta below the level of the artery of Adamkiewicz would appear in the conus through collateralization.¹⁶ The most likely cause is injection of steroid particulate through either the artery of Adamkiewicz or collateral radiculomedullary arterial branches. Prior lumbar surgery may lead to compromise of normal vascular supply to the spinal cord, thus making it more susceptible to vascular injury. Direct arterial injury has also been cited as a cause of injury.¹⁶ Subsequent thrombus formation would result in an embolic infarct in the spinal cord supplied by the affected artery.

Other complications of transforaminal ESI include needle-induced vasospasm. Studies have shown that cerebral arteries constrict in response to mechanical stimuli. Florey²⁶ noted that such vasoconstriction was a local effect lasting 5 seconds to 10 minutes at the site of injury only. Simeone et al.²⁷ found that fine needle (30-gauge) arterial puncture in rhesus monkeys induced intense vasospasm, typically lasting anywhere from 4 hours to 4 days. Many studies have examined mechanically induced vasospasm of the cerebral vasculature and may be indirectly suggestive that a similar phenomenon can occur in the spinal cord and conus medullaris.

Of the multiple recommendations made to avoid such devastating complications as paralysis, first and foremost is the strict adherence to the standard and widely accepted techniques of transforaminal ESIs. These guidelines include the use of multiplanar fluoroscopy or computed tomography guidance together with contrast material to prevent complications. If recovery of neurologic function fails to occur in a patient within a 2- to 3-hour time period after ESI, an initial MRI study may be obtained to exclude an epidural hematoma. Repeat MRI after 24 hours should be performed as well. In our patient, as well as 1 other reported case,¹⁷ a

delayed effect was observed with MRI signal changes occurring after 24 hours.

Conclusion

Given the risk of paralysis associated with cervical transforaminal injection, is it time to reconsider transforaminal injections of the lumbar spine? Although the risk of permanent neurologic deficit is negligible, arguments for discontinuing lumbar injections have been presented in the anesthesia literature.^{19,28} However, when one considers the potential benefit of this noninvasive treatment modality, abandoning such injections may be premature. However, we speculate that more than 6 cases of conus medullaris syndrome after ESI have occurred, and medicolegal considerations may explain why they may go unreported. At the very least, patients should be clearly informed regarding the potential risks of ESI, including paralysis. Although one can safely assume that this severe, devastating complication is rare, its true incidence remains unknown. In our opinion, the rarity of this complication should, at this time, not preclude the continued use of transforaminal ESI for relief of pain in select patients but should be included in the consent process.

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