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# CONUS MEDULLARIS SYNDROME FOLLOWING TRANSFORAMINAL EPIDURAL STEROID INJECTION: A CASE REPORT

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**Background:** Lumbar transforaminal epidural steroid injection (TFESI) is a common procedure for management of obstinate lower back pain.

**Case Report:** We present a case of a conus medullaris infarction after TFESI and contrast it with previously reported cases to evaluate potential risk factors for this rare complication. The patient underwent a fluoroscopically guided TFESI using methylprednisolone for refractory back pain. Following the procedure, the patient lost sensation and strength in the lower extremities and experienced neurogenic bladder and bowel dysfunction. These deficits have persisted for a year with no improvement. Previous cases describe similar symptoms with minimal neurological improvement; however, the timing of radiological presentation on MRI were sometimes delayed.

**Conclusions:** The mechanism of infarction remains ambiguous, and while this complication is rare, the pattern observed for the timeline of deficits indicates they are often permanent and should be contemplated when pursuing TFESI.

**Key words:** Conus medullaris syndrome, infarction, lumbar spine, steroid injection

## BACKGROUND

Corticosteroid injections are frequently utilized for temporary relief of radicular back pain that is refractory to noninvasive medical management (1). Strong evidence indicates that steroid injections significantly reduce patient-reported Visual Analog Scale (VAS) for pain scores at one-, 3-, and 6-month follow-ups and have consistently relieved pain in 50% to 75% of patients in randomized trials (2,3). Physicians regularly utilize image-guided techniques, sometimes in addition to preprocedural aspiration or postprocedural epidurography, to reduce the risk of complications (4). Administration of transforaminal epidural steroid injections (TFESI) is generally considered a low-risk procedure for degenerative spine conditions. Common side effects of TFESI include syncope, dural puncture, facial flushing, paresthesia, and numbness and occur at an overall incidence rate of approximately 0.9% to as high as 16.8% (5-8). A rare but serious complication is infarction of the conus medullaris, which has been documented in a few reports. Herein we report an additional case of conus infarction after lumbar transforaminal injection with particulate steroid and review previously reported cases to further assess the timeline and risk factors for conus medullaris infarction after TFESI.

## CASE

Our case describes an elderly man in his 70s with a past medical history of lumbar radiculopathy, lumbar postlaminectomy syndrome, lumbar spondylosis, displacement and degeneration of lumbar intervertebral discs, lumbar spinal stenosis, and a 2014 lumbar fusion. Other medical diagnoses include hypertension, hypercholesterolemia, type II diabetes with peripheral neuropathy, renal insufficiency, aortic valve sclerosis, leukemia in remission, osteoarthritis, and chronic pain syndrome. The patient presented for medical evaluation of refractory left lower back pain and anterior left thigh

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radiculopathy which had been previously managed by left L2-L3 TFESI twice in March of 2017 and December of 2018 with satisfactory results. At the beginning of June 2020, the patient called his physician to request an additional ESI after reporting the recurrence of lower back pain and leg radiculopathy described as being identical to the pain experienced in prior years with new bilateral leg numbness. The patient consented to a left L2-L3 TFESI with particulate methylprednisolone at an outside institution. He underwent an uncomplicated L2-3 TFESI with no immediate apparent complications. Our patient's loss of sensation and strength were only discovered upon being unable to move to a wheelchair to proceed to the recovery area. The patient was subsequently transferred from the outpatient pain practice to a nearby hospital after experiencing decreased lower extremity sensation that did not resolve during office monitoring.

### TFESI Procedure

Left-sided TFESI were performed at the L2 and L3 in the following manner. The patient was provided moderate sedation, placed in the prone position, and draped in a sterile fashion. Anteroposterior (AP) fluoroscopic guidance was utilized to visualize the vertebral body endplates. After satisfactory visualization of the neuroforamen, the skin was anesthetized by 1% lidocaine. A 22-gauge, 5-inch spinal needle was advanced until bone was contacted. The needle was slightly "walked off" into the foramen using an AP view to assess the needle depth. Upon confirmation that the needle was placed at the midpoint of the pedicle, 4 mL of omnipaque contrast medium was injected under live fluoroscopy and appropriate neural spread was observed with no evidence of vascular uptake. Subsequently, 40 mg of particulate methylprednisolone and one mL of 0.25% bupivacaine were injected. The needle was then restyletted and removed. Post procedure, diagnostic epidurography was performed using 8 mL of omnipaque contrast medium. Flow characteristics demonstrated good bidirectional flow at L2 and L3, and absence of epidural spread was noted.

### Postprocedure Period

The injection report described that the patient tolerated the procedure well and was transported to the recovery area for additional monitoring. In less than 3 hours from the time of the final epidurogram, the patient experienced bilateral lower extremity weakness that did not subside during the time he was held in observation, and he was transported by ambulance to a nearby hospital. Neurological examination on admission revealed 0 out of 5 strength, altered sensation, and arreflexia in the lower extremities. Upper extremity and cranial nerve function remained intact. Magnetic resonance imaging (MRI) of the lumbar spine without contrast medium was completed approximately 6 hours after TFESI and displayed mild enlargement of the conus with an increased T2 signal centrally within the distal thoracic spinal cord and conus medullaris. There was no evidence of hematoma or cord compression (Fig. 1). A subsequent MRI with contrast medium revealed H-shaped conus gray matter hyperintensity and expansion (Fig. 2). As part of his workup, he underwent additional imaging of the cervical spine as well as a lumbar puncture which were unrevealing. Subsequent neurological exams performed 5 days after initial symptoms revealed that sensation to light touch, cold temperature, and proprioception had only slightly improved in the lower extremities. The patient also experienced urinary incontinence and constipation managed by foley catheter and supportive care respectively. After a thorough neurological workup, and evaluation by the neurology and neurosurgical teams, the imaging and presentation of symptoms post procedure were thought to be most consistent with spinal cord infarction. He was discharged to rehab with neurology follow-up. No resolution of any symptoms was noticed at one-, 3-, or 6-month follow-up appointments. As of the most recent one-year follow-up, no improvement in the lower extremity paralysis beyond a novel ability to twitch the right thigh has been documented. There was also no improvement in bowel and bladder incontinence and the patient is being medically managed for spastic bladder. Imaging performed at that time showed lower thoracic cord myelomalacia, most pronounced from T10 through the conus medullaris, consistent with the history of spinal cord infarction.

## DISCUSSION

TFESI is considered effective and safe in managing lumbar radiculopathy resulting from specific nerve root compression. In investigation of long-term outcomes, Kwak et al (9) found that 45% of patients had completely resolved radicular pain at least 4 years after TFESI. Rarely, TFESI can result in complications such as paralysis, epidural abscess, and even death (8). Conus medullaris infarction after TFESI is a rare complication

that has been reported several times in the literature (10-15). In this report, we detail a case of conus medullaris infarction following TFESI and review previous cases.

This case marks the 10th reported case of conus medullaris infarction after TFESI found in a literature review from 2002 to 2021 (Table 1).

Our patient's symptoms occurred soon after TFESI injection, following a similar timecourse to all other reported cases. However, our patient never reported feelings of nausea, flushing, shortness of breath, or pain at the onset of symptoms, which differed from several previous cases. Also, similar to our report, all patients failed to fully regain control over their bowels or bladder (10-15). There were varying gains in improvements of strength, but all patients had lasting paraparesis, hyporeflexia, or some degree of diminished mobility at various durations of follow-up from one month to 5 years (10-15). Some patients had moderate improvements in strength, but this was typically restricted to a single myotome and occurred in the first months after infarction. Together, our case and those in the literature suggest that most patients who suffer conus medullaris infarction after lumbar TFESI have permanent and disabling neurological deficits.

The proposed mechanisms for conus medullaris infarction include inadvertent radicular artery injury and dissection, transient vasospasm secondary to needle placement, and particulate steroid embolization (16). In the case of inadvertent harm caused to the vasculature surrounding the intended injection site, some estimate the rate of inappropriate needle placement to be as high as 30% in the absence of fluoroscopic guidance (17). A commonly used approach for steroid administration is the subpedicular technique, wherein Kambin's triangle is targeted under the inferior surface of the pedicle, where agents can be injected into the anterior extradural space near the inflamed spinal region and preserve the safety of vulnerable dura mater (18). However, the triangle typically contains the artery of Adamkiewicz (AKA) or radicular arteries in as many as 97% of patients (19). Furthermore, the anatomical location of AKA within the neural foramen varies among the population. In studies investigating its location, the AKA was found to be in the superior half of the foramen 97% of the time, on the left side of the foramen 88% of the time, and crossing the dura between T8 and T10 73% of the time (19,20). The unintended injection of agents in this region could result in the transfer of steroids into the vessel, resulting

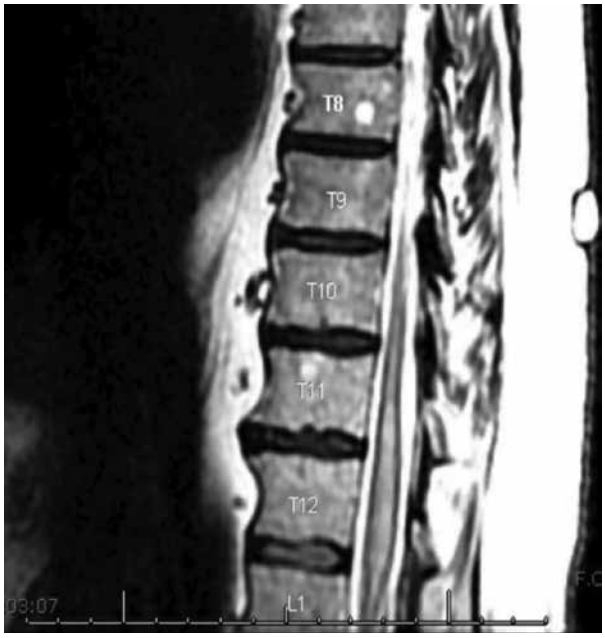


Fig. 1. Sagittal lumbar and thoracic spine MRI showing T2-weighted central cord intensity spanning from T10 to L1 and enlargement of the conus medullaris approximately 6 hours after transforaminal epidural steroid injection.

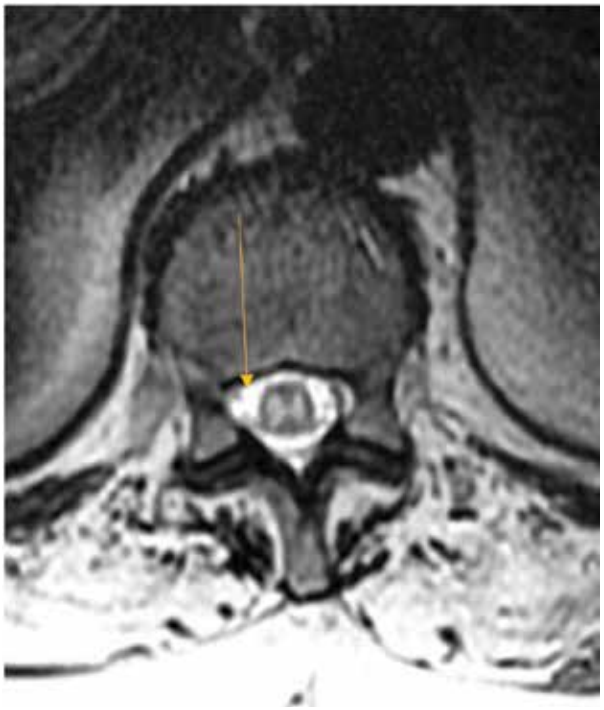

Fig. 2. Axial T2-weighted MRI demonstrating central H-shaped cord intensity at the level of T12 (indicated by yellow arrow).
Abbreviations: MRI, magnetic resonance imaging

Table 1. A summary of previously reported cases and their associated characteristics and outcomes

| Case | Age/Sex | Injection Level | Injection Type | Fluoroscopy | CT Guidance | Aspiration | Epidurography | Timeline of Deficits | Outcomes |
|---|---|---|---|---|---|---|---|---|---|
| Present case | 75/M | Left L2-L3 | Methylprednisolone | Anteroposterior | No | No | Yes | Immediate onset of symptoms with conus infarct confirmed on thoracic MRI within 24 hours | Unresolved lower extremity paralysis and bowel/bladder incontinence |
| Fakhry et al., 201610 | 60/NA | Right L4 | Dexamethasone | Anteroposterior, oblique, and lateral | No | Yes | No | Immediate onset of symptoms. Stat MRI revealed no changes. Repeat MRI the next day revealed conus infarction. | Unresolved lower extremity paralysis and bowel/bladder incontinence |
| Tackla et al., 201211 | 47/M | Left L5-S1 | Unknown | Unknown; no imaging provided by performing physicians | NA | NA | Yes | Immediate onset of symptoms. MRI 4 hours after injection revealed no acute findings. Repeat MRI performed after 48 hours showed a conus infarct. | Follow-up at one month demonstrated improvement of strength from 0 of 5 to 4 of 5 in the L5 myotome with continued voiding insufficiency. |
| Houten et al., 200212 | 64/F | Right L3-L4 & L4-L5 | Betamethasone | Biplanar | No | Yes | No | Immediate onset of symptoms. MRI performed within several hours revealed high signal intensity on T2 imaging consistent with spinal cord edema. | Follow-up at one month demonstrated improvement of strength from 3 of 5 to 4 of 5 bilaterally. |
| Houten et al., 200212 | 51/F | Left L3-L4 | Methylprednisolone | No | Yes | Yes | No | Immediate onset of symptoms. MRI performed after several hours revealed T2 signal intensity consistent with edema. | Unresolved lower extremity paralysis and continued bowel/ bladder incontinence |
| Houten et al., 200212 | 42/M | Left S1 | Methylprednisolone | No | Yes | Yes | No | Immediate onset of symptoms. MRI performed within 6 hours revealed high-intensity T2 signal and an expansion of the lower thoracic spinal cord consistent with infarct. | Follow-up at 5 years demonstrated unresolved 0 of 5 lower extremity paralysis and continued bowel/ bladder incontinence. |

Table 1 (cont.). A summary of previously reported cases and their associated characteristics and outcomes

| Case | Age/Sex | Injection Level | Injection Type | Fluoroscopy | CT Guidance | Aspiration | Epidurography | Timeline of Deficits | Outcomes |
|---|---|---|---|---|---|---|---|---|---|
| Somayaji et al., 200513 | 71/F | Left L2-L3 | Triamcinolone | No | Yes | NA | No | Immediate onset of symptoms. MRI performed within 6 hours revealed no significant findings. Repeat MRI performed after 48 hours revealed diffuse increased signal intensity consistent with infarct. | Follow-up at 6 weeks demonstrated improvement of strength from 0 of 5 to 2 of 5 in the L3 myotome with lower extremity hyperreflexia and continued bowel/ bladder incontinence. |
| Huntoon et al., 200414 | 64/M | Left L1-L2 | Triamcinolone | Unknown | NA | NA | No | Immediate onset of symptoms. MRI performed the same day showed T2 signal change consistent with acute vascular infarction. | Follow-up at 4 years demonstrated continued paraparesis and chronic pain. |
| Kennedy et al., 200915 | 83/F | Left L3-L4 | Betamethasone | Yes | No | No | No | Immediate onset of symptoms. MRI performed after 2 hours revealed no acute findings. Repeat MRI 2 days later revealed increased T2 signal consistent with acute spinal cord infarction. | Follow-up at 18 months demonstrated continued paraparesis with partial motor recovery with all muscles having at least 4 of 5 or 5 of 5 strength on testing, and continued bowel/ bladder incontinence. |
| Kennedy et al., 200915 | 79/M | Right L3-L4 | Methylprednisolone | No | Yes | No | No | Immediate onset of symptoms. Immediate CT revealed contrast medium in L3 foramen and extraforaminal zone. MRI performed 2 days after injection revealed increased T2 signal consistent with conus infarction. | Unresolved lower extremity paralysis and bowel/bladder incontinence |

Abbreviations: CT, computed tomography; F, female; MRI, magnetic resonance imaging; M, male; NA, not available

in vascular injury. It has been theorized that the needle tip used in injection also has the potential to cause vasospasm, as mechanical stimuli have been demonstrated to cause vasoconstriction at the site of injury for as long as 10 minutes (21). Further, particulate steroids (i.e., methylprednisolone) contain various properties which provide them the ability to coalesce into particles larger than 100 μm, drastically occluding capillaries, metarterioles and even arteries resulting in ischemia and infarction (22). While most cases of conus medullaris syndrome have been described following particulate steroid injection, one case of conus medullaris syndrome has occurred after a nonparticulate lumbar TFESI with dexamethasone (15). Interestingly, Derby et al (23) found that even when mixed with lidocaine, dexamethasone particles were uniformly 10 times smaller than red blood cells and did not appear to aggregate; triamcinolone and betamethasone particles varied appreciably in size and their particles were occasionally larger than red blood cells with a proclivity for aggregation. Lastly, methylprednisolone was found to be smaller than red blood cells without appearing to aggregate, but they were observed to be densely packed. These agents have been reported to be associated with higher incidence of cord infarction secondary to embolic events (17).

In our case, anteroposterior fluoroscopic guidance was used in real time to confirm appropriate needle placement. It is standard practice to use image guidance during TFESI for precise needle placement. Negative blood or cerebral fluid aspiration can be helpful to decrease the likelihood of vascular spread before proceeding with the steroid injection. However, Hong et al (24) found that vascular aspiration tests only have a sensitivity of 20.5% and suggested real-time fluoroscopy as the gold standard for injection imaging. Real-time fluoroscopic guidance and computed tomography are both represented in this patient population with infrequent use of aspiration and epidurography to confirm appropriate needle placement. In our case, an epidurogram was performed in the AP and lateral views to confirm adequate flow in the ventral and posterior epidural space at completion of the procedure. In 6 of the 10 cases where either epidurography or aspiration was used, the results indicated accurate needle placement after the injections were given or the absence of vascular spread before continuation of the procedure. As such, the use of these modalities to screen for intravascular penetration and compromise may not be 100% reliable.

A high degree of variability existed in the timing between the onset of patient symptoms and correlative imaging findings suggesting spinal cord infarction. Despite all patients experiencing lower extremity weakness and other symptoms immediately or within hours of the injection, 4 of 10 cases did not have MRI evidence of spinal cord infarction until > 24 hours after the TFESI. In the majority of cases, including our patient, MRI changes were seen within 24 hours of TFESI. High-intensity T2h signal was the most common imaging change reported in the literature. While MRI in the first day of symptoms may be used to exclude other items on the differential, it should not be considered conclusive to rule out spinal cord infarction immediately after TFESI (11). In the setting of a negative initial MRI with continued clinical symptoms following TFESI, providers should have a low threshold to order repeat MRI studies.

While lumbar TFESI is generally considered a safe, minimally invasive procedure, it is not without complications. Our case demonstrates a conus medullaris infarction as a result of methylprednisolone injection (24). Spinal cord infarction after TFESI is a rare, but devastating complication of TFESI. Imaging findings that demonstrate infarction may be delayed by up to 48 hours after TFESI. As demonstrated by the outcomes of these patients, there is often minimal neurological recovery made after injury. Consideration of this dramatic and long-term complication should be considered. The determination of best practices for TFESI should be continually developed to best mitigate the chances of this permanently disabling complication.

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