

Intravascular Injection of Contrast During Lumbar Discography: A Previously Unreported Complication

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ABSTRACT

Objective. Report a case of intravascular contrast injection during lumbar discography.

Setting. An academic University spine center.

Patient. Forty-year-old woman with L5-S1 degenerative disk disease and persistent low back pain.

Intervention. The patient failed to respond to multiple conservative treatments. In consideration of surgical treatment, discography was requested.

Results. A 22G needle was inserted to the central nucleus by a left posterolateral approach. Discography was performed at L4-5 and L5-S1. Testing was performed with pressure manometry, beginning with the L4-5 disk. The pressure increased with contrast injection in the manner of a normal disk. At 45 psi there was a sudden drop to 20 psi. Lateral views obtained during contrast injection demonstrated flow from a central nucleogram cephalad into the anterior epidural space. Images obtained with live fluoroscopy demonstrated a vascular flow pattern, confirmed with lateral and anterior-posterior views. The needle tip was repositioned anterior and superior. Repeat injection resulted in an identical vascular pattern. From the time the pressure dropped, contrast flowed in the vascular pattern without propagation of the nucleogram or increase in intradiscal pressure. The patient was asymptomatic throughout the injection at L4-5. Injection at L5-S1 demonstrated a severely degenerative nucleogram and reproduced the patient's usual low back pain.

Conclusions. Vascular uptake of contrast can occur during lumbar discography, even when the needle tip is ideally located.

Key Words. Spine; Lumbar Discogram; Intravascular Flow; Complication

Introduction

Lumbar discography was first described in 1948 for the diagnosis of herniated lumbar intervertebral discs [1]. With the arrival of advanced imaging technology, including CT and MRI, the original intended use quickly faded and a new use emerged. Since radiographic disk abnormalities are common in people without back pain [2], lumbar discography is now used to direct surgical intervention when suspected discogenic pain is refractory

to conservative treatments. The primary advantage of discography over advanced imaging studies is the subjective input from the patient regarding the location and reproduction of their typical pain during provocation, in addition to detailed objective information regarding intervertebral disk anatomy. The static, prone positioning of a patient during advanced imaging may provide similar anatomic detail, however, does not provide input regarding the source of their typical pain. Current controversies regarding the accuracy of such provocation testing during discography are reviewed elsewhere [3].

The most common complication of lumbar discography is transient increase in pain [4]. Less common but more serious complications include

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discitis, abscess formation, and acute disk herniation [5–7]. A case of a nucleus pulposus embolus has also been reported following discography [8]. Since intervertebral discs are largely avascular, it is no surprise that vascular complications from discography are rare. There is one report of hematoma formation adjacent to the carotid sheath following cervical discography [9]. Otherwise, we were unable to find other reports of vascular complication related to this procedure. Here, we report a case of intravascular contrast flow during lumbar discography.

Case Description

A 40-year-old woman presented to the University neurosurgery clinic with persistent, severe low back pain for 6 months, preceded by 6 years of episodic pain. Her MRI showed disk desiccation and narrowing with a small right paracentral protrusion at L5-S1. She had failed to respond to multiple conservative treatments including physical therapy, anti-inflammatory medications, osteopathic manipulation, and fluoroscopic-guided bilateral L5-S1 zygapophyseal joint blocks. The neurosurgeon requested that the patient undergo discography to evaluate the L5-S1 disk's role as a primary pain generator.

Discography was performed at L4-5 and L5-S1. The L4-5 disk was selected as a control due to its normal appearance on MRI, and proximity to the degenerative L5-S1 disk. A 22G 6" quinke point spinal needle was inserted to the central nucleus of each disk by a left posterolateral approach. Testing was performed with pressure manometry (Monarch Inflation Syringe, Merit Medical Systems, South Jordan, UT), and began at the L4-5 disk. The first blush of contrast into the disk (opening pressure) occurred at 12 psi. From here the pressure slowly and steadily increased with contrast injection in a manner typical of a normal disk.

Intermittent fluoroscopy was used to monitor the contrast dispersal pattern. At 45 psi there was a sudden and unexpected drop in pressure to 20 psi. A static lateral image, obtained during contrast injection, demonstrated flow from a central nucleogram through the posterior annulus into the region of the anterior epidural space and spreading cephalad (Figure 1). It was initially assumed that this pattern represented a small full thickness posterior radial annular tear with contrast flow into the adjacent anterior epidural space. However, no pain or other response was elicited



Figure 1 Initial spot fluoroscopy image taken after a rapid an unexpected drop in disk pressure from 45 psi to 20 psi. This static image shows contrast flow (→) posterior to the disk in what appears to be the anterior epidural space.

from the patient at this time causing the treating physician to pause for a moment. A few seconds later before resuming the contrast injection, another spot lateral image was obtained. On this image, all but the nucleogram had disappeared. To investigate why the contrast previously observed flowing posterior from the nucleogram had disappeared so rapidly, contrast was injected under live fluoroscopy. This rapidly demonstrated the previously observed contrast flow from the nucleogram through the posterior annulus into the region of the anterior epidural space. Under live fluoroscopy, this pattern clearly appeared and progressed in the manner of an intravenous contrast injection. The pattern quickly disappeared moments after discontinuing the contrast injection.

Both lateral and anterior–posterior views confirmed the vascular contrast pattern under live fluoroscopy (Figure 2). In an attempt to avoid this vascular injection, the needle tip was relocated approximately 0.5 cm superior and 0.25 cm anterior from its initial location in the central L4-5 nucleus. Repeat injection of contrast resulted in an identical vascular pattern (Figure 3). From the time the pressure suddenly dropped, all contrast flowed in the vascular pattern without propagation of the nucleogram or further increase in intradiscal pressure. No pain and no unusual response were elicited during or following the injection at L4-5.

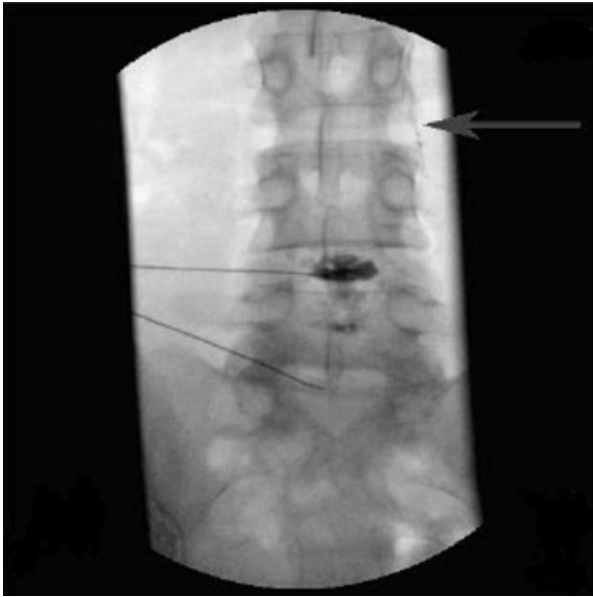


Figure 2 Image taken under live fluoroscopy in anterior-posterior view during contrast injection shows the vascular pattern (←) was not confined to the anterior epidural space.

Next, injection at L5-S1 demonstrated a severely degenerative nucleogram and reproduced the patient's usual low back pain in location and character. Throughout the discography, the patient did not complain of shortness of breath, focal weakness, paresthesias, or other focal neurological deficits. The patient spent 45 minutes in recovery following the procedure, and continued to do well. Examination prior to discharge confirmed this, and she was discharged home in stable condition. Three months later, she underwent an L5-S1 posterolateral fusion with a left iliac crest bone graft and subsequently experienced good relief of her low back pain.

Discussion

The healthy intervertebral disk is largely avascular and receives nutrition from two sources. First, branches of the periosteal arteries create metaphyseal anastomoses that perfuse the superficial portions of the annulus fibrosus [10]. Otherwise, there is no direct blood supply to the disk. Therefore, the majority of the disk receives nutrients by the second method: diffusion from the adjacent vertebral end plates via small arteries in marrow contact channels [11]. Venous drainage from here and the remainder of the vertebral body connects to the basivertebral veins, then to the intervertebral veins, before draining into the general venous cir-

ulation. An indirect correlation has been noted between a reduction in the number and caliber of endplate marrow contact channels and disk degeneration [12].

Given this anatomy, vascular contrast flow should not occur during discography, especially when the needle tip is located at the center of the nucleus pulposus. So how did this occur? The exact origin of the venogram could not be determined by fluoroscopy at the time of the injection, so we can only theorize. Following acute injury, or as a result of degenerative changes, neovascularization of intervertebral discs has been observed [12,13]. A study of surgically obtained frozen sections showed the presence of endothelial cells in both protruded and extruded disk material [14]. Another study examining sections obtained from patients that underwent anterior fusions for discogenic pain demonstrated microvessel formation in the nucleus pulposus of nonherniated discs [15]. These vascular channels provide an important conduit for macrophage migration [14] and subsequent resorption of herniated disk fragments [16]. Since the patient presented in this report had a history of degenerative disk disease, it is possible that neovascularization of the disk was involved in the observed vascular uptake.

However, there are a number of problems with this theory. First, this vascular uptake was seen in

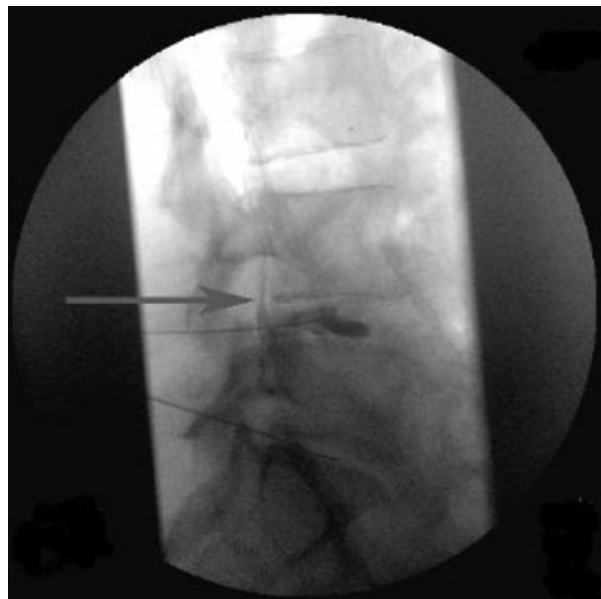


Figure 3 Image taken with live fluoroscopy in the lateral view after repositioning the needle. It continues to show the exact same vascular contrast flow pattern (→). Notice how the needle tip is repositioned in comparison to Figure 1.

an asymptomatic control disk without the degenerative changes expected for diffuse neovascularization of the nucleus pulposus to occur. Second, the final image presented above (Figure 3) was obtained following anterior and superior repositioning of spinal needle. Given the exact reproduction of the initial vascular flow pattern following repositioning, it is unlikely that the injected contrast entered the circulation via microvessels found within a degenerative nucleus pulposus. An alternative explanation is that during introduction of the spinal needle into the intervertebral disk, a vein confined by annular lamellae was ruptured and may have provided the path of least resistance for contrast injected into the disk.

Other potential routes for vascular contrast spread during discography exist, and are described in a handful of case reports. The first involves the venous sinusoidal vessels and describes the presence of nucleus pulposus pulmonary emboli on post mortem examination following a severe anaphylactic reaction during lumbar discography [8]. During the anaphylactic reaction the patient suffered from violent tonic-clonic seizures involving her lower extremities and back. Microscopic examination of the intervertebral discs demonstrated extrusion of the nucleus pulposus down to the vertebral marrow. The authors hypothesized that the disk material was able to enter the patient's venous circulation via the venous sinusoidal vessels from gross and microscopic fractures of the vertebral endplates that occurred during the tonic-clonic seizures.

Reports of fibrocartilaginous emboli resulting in spinal cord injuries [17–20] provide the second group of cases that demonstrate a potential mechanism for vascular contrast spread during discography. The first of these cases dates back to 1961 [20]. In each of these cases, patients between 14–16 years of age were performing innocuous flexion or extension movement of the neck or back when they experienced sudden onset of myelopathy. The exact mechanism behind these spinal cord injuries is unknown, but many have been suggested. In one of these cases, an MRI performed 5 months after onset of myelopathy demonstrated T11–12 intervertebral disk desiccation, multiple Schmorl's nodes, and intraspongious disk prolapse into the T12 vertebral body [18]. It was hypothesized that the nucleus pulposus may have herniated into the vertebral marrow via endplate damage such as a Schmorl node, and from there migrated into a previously damaged radicular artery causing occlusion and spinal cord infarction

[18]. Other theories include: nucleus pulposus entering a venous sinusoidal vessel then traveling through a spinal arteriovenous malformation to occlude a spinal artery [17], and disk material entering the circulation via neovascularization of the disk [17].

Each of these reports demonstrates a potential avenue for venous contrast uptake during discography. It is not clear which of these mechanisms were involved in the patient presented here and the patient suffered no known morbidity related to this complication. Regardless of the cause, additional increases in intradiscal pressure should be avoided if vascular flow is observed during discography in order to avoid potential morbidity associated with intravascular expulsion of nucleus pulposus.

Conclusion

Inadvertent intravascular injection can occur during lumbar discography even when the needle tip is ideally placed in the center of the nucleus pulposus.

References

- 1 Lindblom K. Diagnostic puncture of intervertebral discs in sciatica. *Acta Orthop Scand* 1948;18:132.
- 2 Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am* 1990; 72:403–8.
- 3 Cohen SP, Larkin TM, Barna SA, et al. Lumbar discography: A comprehensive review of outcome studies, diagnostic accuracy, and principles. *Reg Anes Pain Med* 2005;30(2):163–8.
- 4 Tallroth K, Soini K, Antti-Poika I, et al. Premedication and short-term complications in iohexol discography. *Ann Chir Gynaecol* 1991;80:49–53.
- 5 Fraser RD, Osti OL, Vernon-Roberts B. Discitis after discography. *J Bone Joint Surg Br* 1987;69:26–35.
- 6 Junila J, Niinimäki T, Tervonen O. Epidural abscess after lumbar discography. A case report. *Spine* 1997;22:2191–3.
- 7 Poynton AR, Hinman A, Lutz G, Farmer JC. Discography-induced acute lumbar disc herniation: A report of five cases. *J Spinal Disord Tech* 2005; 18(2):188–92.
- 8 Shreck RI, Manion WL, Kambin P, Sohn M. Nucleus pulposus pulmonary embolism. A case report. *Spine* 1995;20(22):2463–6.
- 9 Guyer RD, Ohnmeiss DD, Mason SL, Shelokov AP. Complications of cervical discography: Findings in a large series. *J Spinal Disord* 1997;10(2):95–101.

- 10 Ratcliffe JF. The arterial anatomy of the adult human lumbar vertebral body: A microarteriographic study. *J Anat* 1980;131(1):57-79.
- 11 Maroudas A, Stockwell RA, Nachemson A, Urban J. Factors involved in the nutrition of the human intervertebral disc: Cellularity and diffusion of glucose in vitro. *J Anat* 1975;120(1):113-30.
- 12 Benneker LM, Heini PF, Alini M, Anderson SE, Ito K. Vertebral endplate contact channel occlusions and intervertebral disc degeneration. *Spine* 2005;30(2):167-73.
- 13 Kauppila LI. Ingrowth of blood vessels in disc degeneration. Angiographic and histological studies of cadaveric spines. *J Bone Joint Surg Am* 1995;77(1):26-31.
- 14 Koike Y, Uzuki M, Kokubun S, Sawai T. Angiogenesis and inflammatory cell infiltration in lumbar disc herniation. *Spine* 2003;28(17):1928-33.
- 15 Freemont AJ, Watkins A, Le Maitre C, et al. Nerve growth factor expression and innervation of the painful intervertebral disc. *J Pathol* 2002;197:286-92.
- 16 Haro H, Shinomiya K, Komori H. Upregulated expression of chemokines in herniated nucleus pulposus resorption. *Spine* 1996;21(14):1647-52.
- 17 Toro G, Roman GC, Navarro-Roman L, et al. Natural history of spinal cord infarction caused by nucleus pulposus embolism. *Spine* 1994;19(3):360-6.
- 18 Tosi L, Rigoli G, Beltramello A. Fibrocartilaginous embolism of the spinal cord: A clinical and pathogenic reconsideration. *J Neurol Neurosurg Psychiatry* 1996;60(1):55-60.
- 19 Yousef O, Appenzeller P, Kornfeld M. Fibrogratigenous embolism: An unusual case of spinal cord infarction. *Am J Forensic Med Pathol* 1998;19(4):395-9.
- 20 Naiman JL, Donohue WL, Prichard JS. Fatal nucleus pulposus embolism of spinal cord after trauma. *Neurology* 1961;11:83-7.