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# Enhanced Lumbar Nerve Roots in the Spine without Prior Surgery: Radiculitis or Radicular Veins?

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**PURPOSE:** To evaluate the clinical significance of continuous intradural lumbosacral nerve root enhancement in symptomatic patients without prior lumbar surgery. **METHODS:** Fifty-three patients without prior back surgery, referred to our institution for evaluation of low-back pain and radiculopathy, were studied with gadolinium-enhanced MR (0.1 mmol/kg) of the lumbar spine. Scans were reviewed for the presence of lumbosacral nerve root enhancement and any associated nerve root compression. Results were correlated with clinical history and physical examinations. **RESULTS:** Seventeen continuously enhancing nerve roots and two enhancing fila terminale were observed in 13 patients. Eight of 17 (47%) had no referable symptoms. Nine of these nerve roots (53%) were not associated with any degree of nerve root compression. Seven cases (41%) were noted to have flow-related enhancement on the entry section of the T1-weighted axial sequence. **CONCLUSIONS:** Lumbosacral nerve root enhancement correlates poorly with clinical radiculopathy. The use of contrast enhancement to detect lumbosacral nerve root enhancement in cases in which the unenhanced scan is less than diagnostic is not warranted. The high association between lumbosacral nerve root enhancement and entry-section flow-related enhancement suggests that these enhancing structures within the cauda equina are vessels. It is likely that lumbosacral nerve root enhancement represents intravascular enhancement of radicular veins and not a breakdown in the blood-nerve barrier.

**Index terms:** Nerves, lumbar; Nerves, spinal; Spine, magnetic resonance; Radiculitis

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Intradural lumbosacral nerve root enhancement was first described by Boden et al as a common finding in asymptomatic patients studied within the first 6 months after successful laminectomy and diskectomy (1). These authors noted continuous lumbosacral nerve root enhancement from the level of previous nerve root compression extending cephalad toward the conus (1). A more recent study demonstrated that this phenomenon occurs in 5% of symptomatic patients without prior surgery, most often associated with nerve root compression from disk herniation (2). A high correlation was found between the level of lumbosacral nerve root enhancement and the patients' clinical symptoms in that study.

Having observed this phenomenon often in postoperative contrast-enhanced MR studies, it was our objective to confirm lumbosacral nerve root enhancement in lumbar spines not operated on and to determine the relationship between it and root compression. More specifically, we sought to determine a correlation between lumbosacral nerve root enhancement and clinical radiculopathy.

## Materials and Methods

Fifty-three patients without prior back surgery presenting with low-back pain or radiculopathy between August and November 1992 were included in this study. Clinical

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data were compiled from medical records and clinical questionnaires completed by the patients before imaging. Magnetic resonance (MR) studies were performed on a 1.5-T system and consisted of T1-weighted sagittal (600/15/2 [repetition time/echo time/excitations]) and fast spin-echo sagittal (2800/60, 4 echo train) images. T1-weighted axial (800/15) sequences were obtained contiguously from the L2–3 disk to the L5–S1 disk before and after intravenous administration of gadolinium contrast. All scans were reviewed by two neuroradiologists without benefit of the clinical data. MR images were evaluated for the presence of focal or continuous intradural lumbosacral nerve root enhancement and extradural compression of the thecal sac or nerve roots. Those patients noted to have continuous intradural lumbosacral nerve root enhancement were followed with repeat MR images performed between 3 and 6 months after the initial study.

## Results

Continuous intradural enhancement occurred in 17 nerve roots (13 patients), and focal enhancement occurred in 10 nerve roots (10 patients). Table 1 lists the 6 patients in whom continuous intradural enhancement was associated with nerve root compression. Table 2 lists the 7 patients in whom continuous intradural enhancement was not associated with nerve root compression.

Nerve root compression was identified in 22 patients with a total of 26 nerve roots involved. In all cases, nerve roots were compressed by herniated disks. Of these 26 roots, 8 (31%) demonstrated continuous intradural enhancement from the levels of compression cephalad to the L-2 level (Fig 1). In addition, one of the 6 patients also demonstrated continuous intradural enhancement along the distribution of the filum terminale not associated with compression. Focal enhancement limited to the compressed segment of the nerve was identified in an additional 10 cases.

Seven patients were noted to have continuous intradural lumbosacral nerve root enhancement without evidence of associated nerve root compression (Table 2). Four of the seven had single root enhancement. One had a combination of enhancing S-1 and filum terminale, and one patient subsequently determined to have sustained a conus infarction had enhancement of both L-5 roots and a single S-1 nerve root.

A small focus of hyperintense signal was noted on the most cephalad sections of precontrast T1-weighted axial sequences (Fig 2) in two of the eight compressed nerve roots with continuous intradural enhancement and in five of nine uncompressed nerve roots with continuous enhancement. The location of this hyperintense focus marked exactly the most cephalad extent of the subsequent nerve root enhancement on the postcontrast sequences. This observation was interpreted to represent flow-related enhancement and is recorded as such in the tables. No evidence of flow-related enhancement was detected in patients who did not demonstrate continuous intradural lumbosacral nerve root enhancement.

After the initial study, eight patients returned at 3 to 6 months for a follow-up contrast-enhanced MR. Five patients were lost to follow-up. Resolution of enhancement with marked regression of the disk herniation was seen in three of four patients with previously compressed lumbosacral nerve root enhancement. Persistent enhancement without regression of the disk herniation was seen in a single patient. Four patients with uncompressed lumbosacral nerve root enhancement underwent follow-up MR. Persistent enhancement was seen in two of the four patients, whereas resolution of enhancement was noted in the other two.

## Discussion

Enhancement of intradural lumbosacral nerve roots was reported by Boden et al in 10 of 16 asymptomatic patients studied at 3 weeks, 3 months, and 6 months after successful laminectomy (1). In all 10 cases, lumbosacral nerve root enhancement resolved between 3 and 6 months later. They observed that this enhancement tracked cephalad toward the conus medullaris from the level of the surgical decompression.

In a study of 200 symptomatic patients without prior back surgery, Jinkins recently observed focal or multisegmental (continuous) intradural lumbosacral nerve root enhancement in 5% (2). He reported an excellent correlation between the level of enhancement and the patients' symptoms. In a separate study, Jinkins et al reported 20 postoperative cases of focal and 6 cases of continuous intradural lumbosacral nerve root enhancement with an overall clinical correlation of 95.7% (3).

The mechanism implied or suggested by these authors as the cause for lumbosacral nerve root enhancement is a breakdown in the blood-nerve barrier. It is well established that

**TABLE 1: Nerve root enhancement with compression**

| Case | Age | Sex | Initial or F/U | Clinical Presentation | Level of NRE | FRE | Symptom Onset–Scan Interval | Additional MR Findings | Symptoms Referable to NRE? |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 25 | F | Initial | LBP→P + N, RLE | Right S-1 | – | 3 mo | Right L5-S1 disk extrusion<br>Right S-1 NR compression | Yes |
| | | | Lost to F/U | | | | | | |
| 2 | 34 | M | Initial | LBP→N, LLE | Left S-2 | – | 3 wk | Left L5-S1 disk extrusion<br>Left S-1 and S-2 NR compression | Yes |
| | | | Lost to F/U | | | | | | |
| 3 | 24 | M | Initial | LBP→P + MN, RLE | Left L-5<br>Right S-1 | +<br>– | 1 mo | Left L4-5 protrusion<br>Left L5 NR compression | No |
| | | | | | | | | Right L5-S1 extrusion<br>Right S-1 NR compression | Yes |
| | | | F/U | Stable | Stable | Stable | 14 wk | No change | |
| 4 | 21 | F | Initial | LBP→P + N, LLE | Left S-1<br>Left S-2 | –<br>– | 2 wk | Left L5-S1 extrusion<br>S-1 and S-2 NR compression | Yes<br>Yes |
| | | | F/U | Improved Minimal | Minimal | – | 11 wk | L5-S1 decreased in size | |
| 5 | 23 | M | Initial | LBP→P + N, LLE | Left L-5 | – | 8 wk | L4-5 extrusion<br>Left L-5 NR compression | Yes |
| | | | F/U | Mildly improved | Resolved | – | 6 mo | Extrusion mildly reduced in size | |
| 6 | 71 | M | Initial | LBP→P + N, LLE | Left L-5<br>Filum | +<br>+ | 2 mo | Left L4-5 disk extrusion<br>L-5 NR compression | Yes |
| | | | F/U | Stable | Resolved<br>Stable | –<br>+ | 5 mo | Extrusion markedly reduced in size | |

Note.—LBP indicates low back pain; P, pain; N, numbness; LLE, left lower extremity; RLE, right lower extremity; NRE, nerve root enhancement; FRE, flow-related enhancement; F/U, follow-up.

the capillary permeability of the intradural nerve roots is akin to that of the central nervous system (blood-brain barrier) (4–6). The presence of contrast within the nerve root apparently would imply a breakdown of this capillary barrier. Compression-induced changes in nerve root metabolism, including changes in capillary permeability, have been documented experimentally in laboratory animals (7, 8). We believe that this was the dominant mechanism in our patients with focal nerve root enhancement limited to the compressed segment of the root. However, we propose an alternate mechanism for continuous intradural lumbosacral nerve root enhancement, which, in our experience, does not correlate consistently with nerve root compression or clinical symptoms.

The argument that retrograde enhancement from the nerve root sleeve cephalad toward the conus medullaris also represents blood-nerve-barrier breakdown is open to question for several reasons. First, although it has been proved that compression-induced ischemia of spinal roots causes acute disruption of axonal transport and eventually leads to wallerian degeneration (9), neither antegrade nor retrograde enhancement corresponding to active (wallerian) degeneration of the long tracts of the central nervous system has been conclusively documented in the literature to date. Second, in two

**TABLE 2: Nerve root enhancement without compression**

| Case | Age | Sex | Initial or F/U | Clinical Presentation | Level of NRE | FRE | Symptom Onset–Scan Interval | Additional MR Findings | Symptoms Referable to NRE? |
|---|---|---|---|---|---|---|---|---|---|
| 7 | 33 | F | Initial | LBP→P, RLE | Left S-1 | + | 3 mo | L4-5 desiccation | No |
| | | | Lost to F/U | | | | | | |
| 8 | 39 | M | Initial | LBP→P + N, LLE | Right S-1 | + | 9 y | Left L5-S1 protrusion | No |
| | | | Lost to F/U | | | | | | |
| 9 | 28 | M | Initial | LBP→P + N, RLE | Right S-3 | − | 1 wk | Right L5-S1 etrusion<br>Right S-1 NR compression | No |
| | | | F/U S/P discectomy | Stable (3 mo) | Stable | − | 4 mo | Epidural scar<br>No NR compression | No |
| 10 | 75 | M | Initial | LBP→P + N, LLE | Right S-3 | + | 3 y | Left L4-5 extrusion<br>Mild-mod spinal stenosis L1-2, L4-5 | No |
| | | | F/U S/P discectomy | Improved<br>No radiating pain (6 wk) | Resolved | − | 3 y, 3 mo | Decompressive laminectomies L1-2, L4-5<br>No residual stenosis | |
| 11 | 32 | M | Initial | Nonanatomic sensory changes RLE | Right S-1<br>Filum | +<br>+ | 10 y | No significant disease | No |
| | | | F/U | No change | Right S-1 | + | 10 y, 3 mo | No change | |
| 12 | 67 | M | Initial | RLE P + N | Right L-5<br>Right S-1<br>Left L-5 | +<br>−<br>− | 2 wk | Conus infarct, T-12 vertebral body infarct | Yes<br>Yes<br>No |
| | | | F/U | LBP→N, RLE | Resolved | − | 5 mo | Infarcts decreased in size | |
| 13 | 9 | F | Initial<br>Lost to F/U | N, LLE | Left S-2 | + | 11 mo | Paraspinal ganglioneuroma invading spinal canal T12-L3 | No |

Note.—LBP indicates low back pain; P, pain; LLE, left lower extremity; RLE, right lower extremity; NRE, nerve root enhancement; FRE, flow-related enhancement; F/U, follow-up.

cases in this study, contiguous axial images obtained above the level of the conus failed to demonstrate lumbosacral nerve root enhancement tracking cephalad into the substance of the cord. Rather, it continued proximally beyond the root entry zone, extrinsic to the dorsal or ventral surface of the cord (Fig 3). Finally, invoking blood-nerve barrier breakdown as the cause for lumbosacral nerve root enhancement does not explain the observation of flow-related enhancement noted in a large percentage of cases in this study. Taking all this into account, we believe that the most likely explanation for continuous intradural lumbosacral nerve root enhancement involves intravascular rather than neuronal enhancement. The presence of flow-related enhancement within these vessels would be consistent with slow flow within intradural veins draining caudally from the level of the conus.

Accurate descriptions of the venous anatomy of the conus and cauda equina are scarce in the radiologic literature because these superficial veins are poorly visualized at spinal angiography, lumbar phlebography, and myelography (10–13). The venous drainage of the conus proceeds caudally through dorsal and ventral median veins, which course within or adjacent to

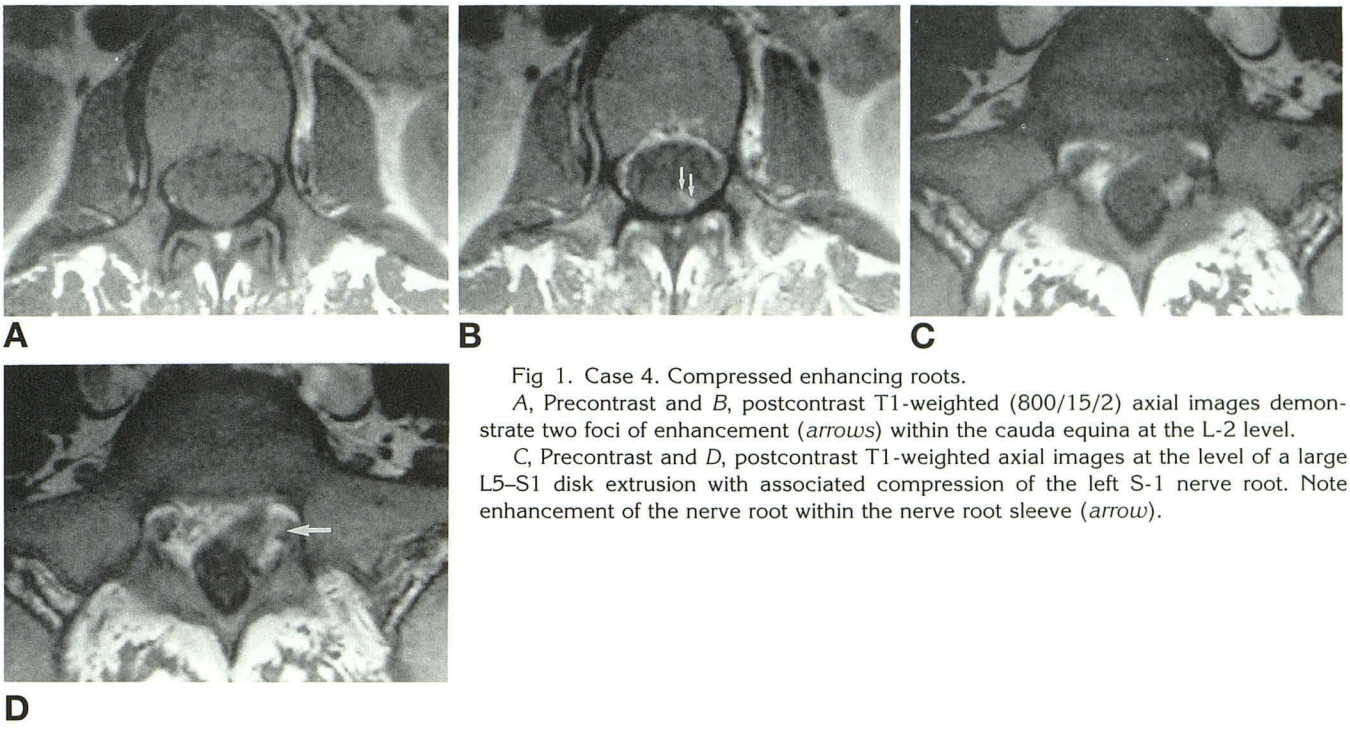

Fig 1. Case 4. Compressed enhancing roots.

*A*, Precontrast and *B*, postcontrast T1-weighted (800/15/2) axial images demonstrate two foci of enhancement (*arrows*) within the cauda equina at the L-2 level.

*C*, Precontrast and *D*, postcontrast T1-weighted axial images at the level of a large L5–S1 disk extrusion with associated compression of the left S-1 nerve root. Note enhancement of the nerve root within the nerve root sleeve (*arrow*).

the dorsal or ventral sulcus of the cord. These veins drain into the epidural venous plexus by way of the great radicular and small radicular veins. The great radicular veins are few in number, large in caliber, and are most commonly located at the lower thoracic or upper lumbar levels (14–18). Each great radicular vein usually accompanies the adjacent nerve root as it exits the dura. In approximately 25% of the 70 specimens examined by Moes and Maillot, the ventral median vein continued its caudal descent beyond its confluence with lower thoracic or upper lumbar great radicular veins to terminate in an accessory great radicular vein, which

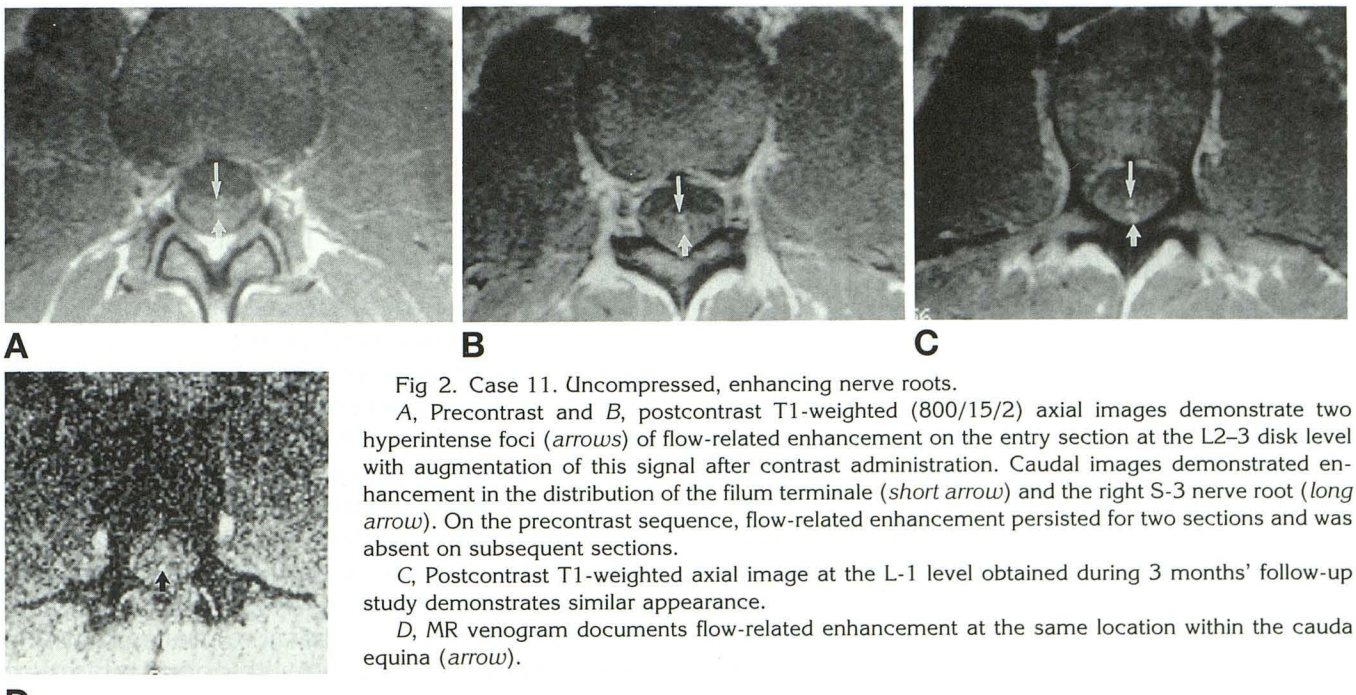

Fig 2. Case 11. Uncompressed, enhancing nerve roots.

*A*, Precontrast and *B*, postcontrast T1-weighted (800/15/2) axial images demonstrate two hyperintense foci (*arrows*) of flow-related enhancement on the entry section at the L2–3 disk level with augmentation of this signal after contrast administration. Caudal images demonstrated enhancement in the distribution of the filum terminale (*short arrow*) and the right S-3 nerve root (*long arrow*). On the precontrast sequence, flow-related enhancement persisted for two sections and was absent on subsequent sections.

*C*, Postcontrast T1-weighted axial image at the L-1 level obtained during 3 months' follow-up study demonstrates similar appearance.

*D*, MR venogram documents flow-related enhancement at the same location within the cauda equina (*arrow*).

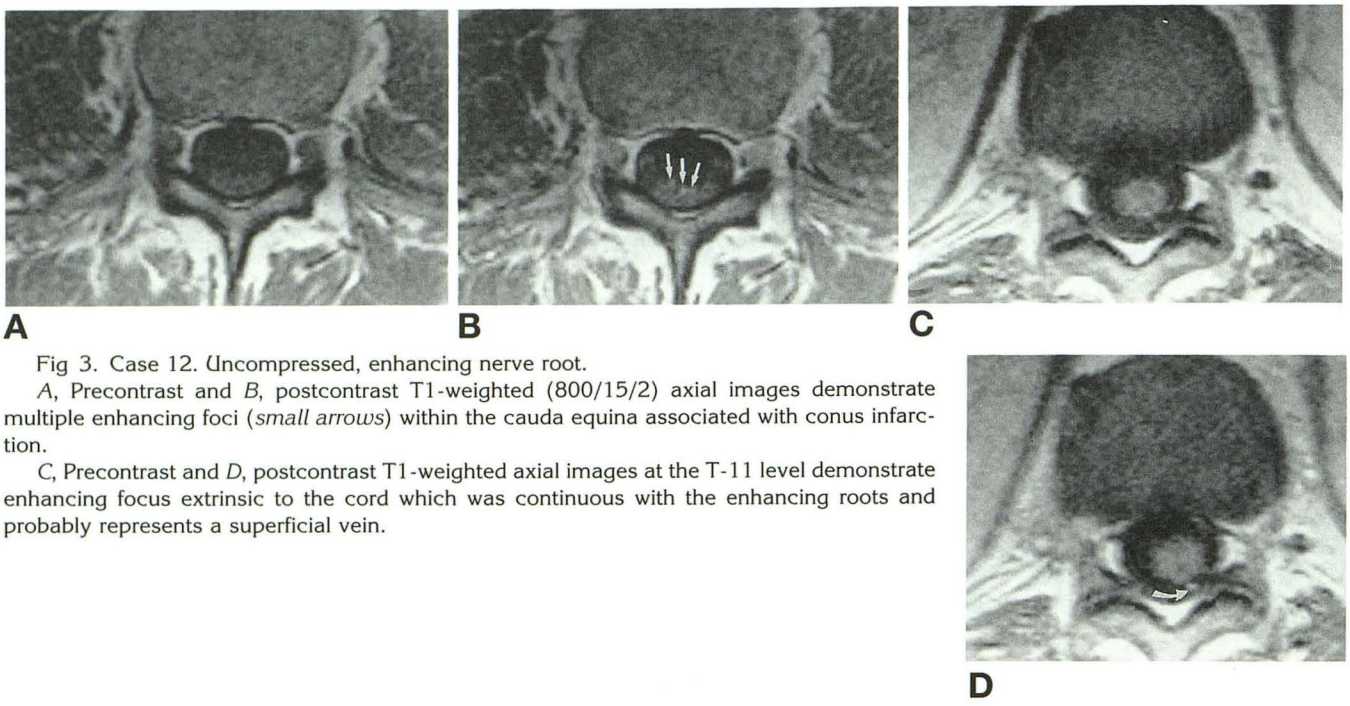


Fig 3. Case 12. Uncompressed, enhancing nerve root.

*A*, Precontrast and *B*, postcontrast T1-weighted (800/15/2) axial images demonstrate multiple enhancing foci (*small arrows*) within the cauda equina associated with conus infarction.

*C*, Precontrast and *D*, postcontrast T1-weighted axial images at the T-11 level demonstrate enhancing focus extrinsic to the cord which was continuous with the enhancing roots and probably represents a superficial vein.

accompanied a distal lumbosacral nerve root or filum terminale (15) (Fig 4). These distal lumbosacral great radicular veins have been described by several other authors (14, 16–18). The caliber of these vessels varies, ranging in diameter from 0.5 to 1.1 mm, with the ventral great radicular veins being dominant at the lower lumbosacral level (14, 15, 19). We dissected five cadaveric spines for the purpose of confirming the presence of these veins and found a large accessory great radicular vein at the S-1 level in one case (Fig 5). Additionally, each nerve root contains within its endoneurium two or three ventral or dorsal small radicular veins ranging in size from 150 to 200 $\mu$m. These smaller vessels are considered by most authorities also to drain caudally into the epidural venous plexus (12, 15, 16, 20).

Given this anatomy, intravascular enhancement theoretically could be encountered within a great radicular vein or within multiple small radicular veins coursing beneath the endoneurium of the nerve root. However, considering the size of these small radicular veins (150 to 200 $\mu$m), it is likely that only veins of the caliber of the great radicular vein would produce flow-related enhancement. The cases of lumbosacral nerve root enhancement in this study observed to have flow-related enhancement most likely represent lumbosacral great radicular veins.

We propose that continuous intradural lumbosacral nerve root enhancement may be encountered as either a physiologic or pathologic phenomenon. Physiologic lumbosacral nerve root enhancement would be expected in patients with variably present distal lumbosacral great radicular veins. The caliber of these vessels approximates that of a normal root such that they could be easily mistaken as a spinal nerve. We believe that the presence of such a vein probably accounts for most of the cases of enhancement in the uncompressed category of this study. The high incidence of flow-related enhancement in this group (67%) lends support to this interpretation. If one excludes the case with conus infarct (case 12), this figure rises to 82%. Two of the four cases in the uncompressed group demonstrated persistent lumbosacral nerve root enhancement on their 3- to 6-month follow-up exams, as would be expected if, as we suggest, this phenomenon represents a normal anatomic variation. This theory does not explain the two remaining cases that demonstrated resolution of enhancement at follow-up. The small conus infarct case (case 12) was noted to have multiple lumbosacral nerve root enhancement, which subsequently resolved. The observation of lumbosacral nerve root enhancement in association with conus infarction has been previously reported (21). Al-

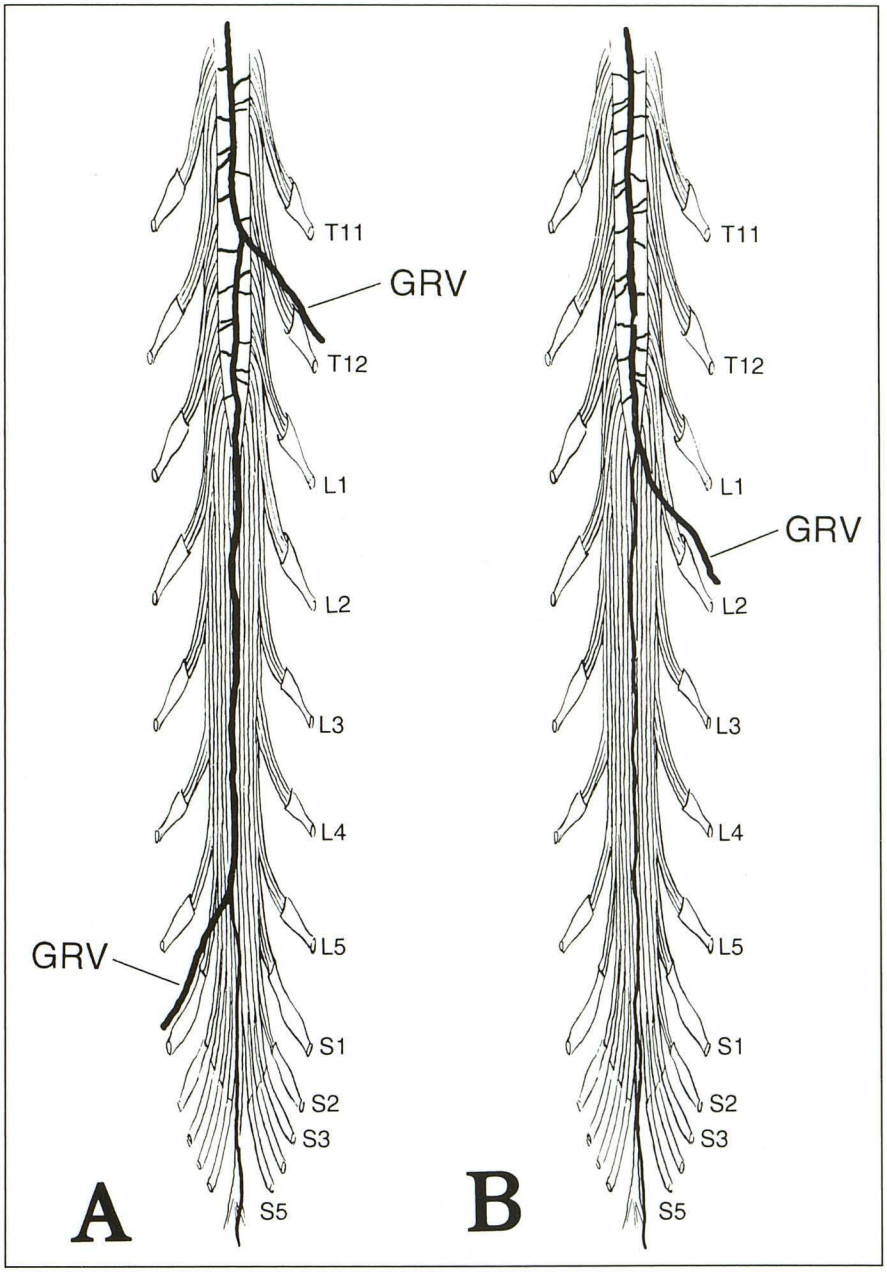


Fig 4. The superficial veins of the ventral surface of the conus and cauda equina.

*A*, The venous anatomy present in one quarter of cadaveric specimens examined by Moes and Maillot (19). Note great radicular veins (*GRV*) accompanying the right S-1 nerve root in addition to the more constant thoracolumbar great radicular veins accompanying T-12 on the *left*.

*B*, Three quarters of cadaveric specimens demonstrated only thoracolumbar great radicular veins, here depicted at L-2 on the *left*. A small vein accompanies the filum terminale distally.

though breakdown of the blood-nerve barrier could be proposed as the mechanism of enhancement in our case, it is noteworthy that the area of conus infarct did not enhance. Alternatively, this transient enhancement may have represented arteriovenous shunting (a transient angiographic phenomenon seen in acute cerebral infarction) into small radicular veins. The remaining case of resolving enhancement in the uncompressed group (case 10) involved a single nerve root (S-3) associated with moderate central spinal canal stenosis at the L1–2 disk level. The patient underwent an L1–2 decompressive laminectomy and had a follow-up study 6 weeks after surgery. It is interesting to speculate that the lumbosacral nerve root enhancement observed initially may have represented collateral venous drainage resulting from impaired epidural venous plexus outflow associated with the stenosis. After decompression, collateral flow through this radicular vessel may have decreased sufficiently to cause resolution of the enhancement.

We propose that pathologic or abnormal lumbosacral nerve root enhancement represents obstruction of the small radicular veins within

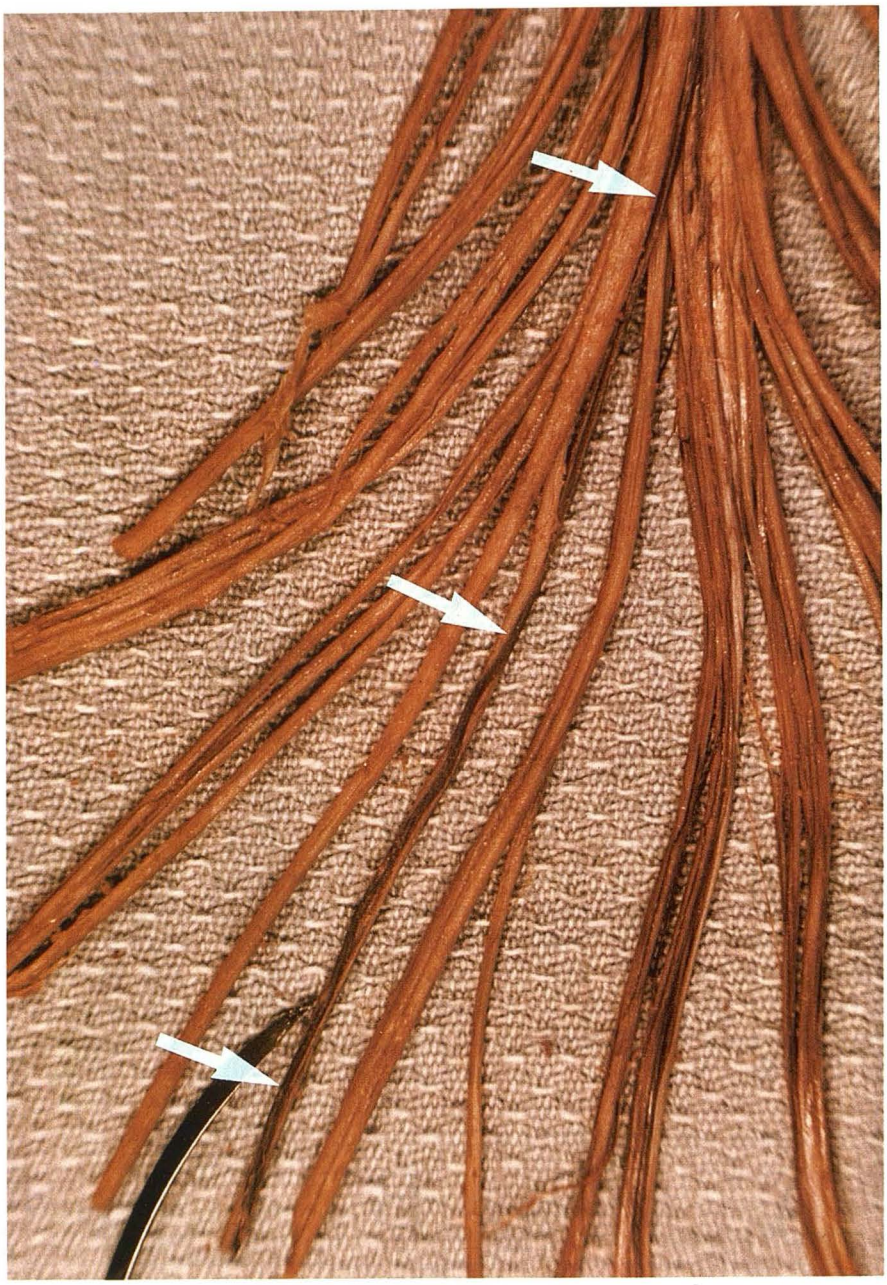

Fig 5. Cadaveric dissection of the ventral surface of the cauda equina reveals a lumbosacral great radicular vein (*arrows*) accompanying the right S-1 nerve root.

the endoneurium of the nerve root. This intravascular enhancement is directly related to the nerve root compression and, as such, would be expected to resolve with spontaneous disk regression or surgical decompression. Postsurgical inflammation probably causes a similar transient obstruction of these veins, resulting in lumbosacral nerve root enhancement as observed by Boden et al (1). Three of four cases of enhancement associated with nerve root compression in this study demonstrated disk regression on follow-up studies obtained 3 to 6 months after conservative therapy. In all three cases, the enhancement resolved. The remaining case showed no regression of the disk herniation with persistent lumbosacral nerve root enhancement on the follow-up study.

The poor overall correlation between the level of lumbosacral nerve root enhancement and the patients' symptoms in this study would be expected if, as we suggest, these enhancing nerve roots are actually radicular vessels and not, as previously suggested, indicative of an extensive radiculitis. The difference between our poor clinical correlation and the excellent correlation obtained by Jinkins in his series of patients not

operated on is probably related to the higher incidence of nerve root compression in his 10 cases of contiguous intradural lumbosacral nerve root enhancement (70%), compared with our 47% incidence (2). In addition, all three of his cases of lumbosacral nerve root enhancement without compression had clinical polyneuropathies, making it more difficult to exclude symptomatic enhancement. We cannot explain the relatively low incidence of continuous intradural lumbosacral nerve root enhancement (5%) in his study. Our incidence of approximately 24% would be more in keeping with anatomic studies if a significant number of cases of lumbosacral nerve root enhancement reflects the presence of a distal lumbosacral great radicular vein.

In conclusion, our results suggest that most, if not all, cases of continuous intradural lumbosacral nerve root enhancement represent intravascular enhancement. We propose that this enhancement may be encountered as either a physiologic or pathologic phenomenon. Physiologic enhancement would be expected, even in the asymptomatic state, in those patients with variably present distal lumbosacral great radicular veins. In such patients, any associated compression of the accompanying nerve root may be incidental, and enhancement would be expected to be a static phenomenon. Pathologic enhancement could be produced by partial obstruction of the small radicular veins found within the endoneurium of all spinal nerves. Such enhancement would be directly related to the presence of nerve root compression and should eventually resolve after spontaneous regression of disk herniation, surgical decompression, or, in the immediate postoperative setting, after the resolution of inflammation.

Because as many as half of all cases of contiguous intradural lumbosacral nerve root enhancement may be clinically irrelevant, this phenomenon is of dubious clinical value. Although the addition of contrast-enhanced sequences occasionally can provide greater anatomic detail in the MR evaluation of the spine with degenerative disk disease not operated on, the use of intravenous contrast for the purpose of detecting lumbosacral nerve root enhancement is not warranted and may be clinically misleading.

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