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MR of Enhancing Nerve Roots in the Unoperated Lumbosacral Spine

J. Randy Jinkins

PURPOSE: To evaluate prospectively the unoperated lumbosacral spine by gadolinium-enhanced MR for evidence of clinically correlative abnormal enhancement of nerve roots. **METHODS:** Two hundred patients were prospectively studied with intravenous gadolinium- (0.1 mmol/kg) enhanced MR. Evidence was sought of intrathecal enhancement of lumbosacral nerve root(s), and the correlation of this enhancement with the clinical syndrome was evaluated. **RESULTS:** Ten patients demonstrated abnormally enhancing lumbosacral nerve root(s) (5%). Of these, seven (70%) were associated with focally protruding disk pathology. The three (30%) remaining patients had isolated enhancement of multiple nerve roots in the absence of significant associated anatomic pathology. Overall, the correlation of radicular enhancement with the presenting clinical syndrome was excellent. **CONCLUSION:** A breakdown in the blood-nerve barrier as observed on gadolinium-enhanced MR serves as a marker for nerve root pathology in the unoperated lumbosacral spine, which may have clinical relevance in certain clinical situations.

Index terms: Spinal cord, magnetic resonance; Contrast media, paramagnetic; Nerves, spinal; Radiculitis

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Magnetic resonance (MR) is of great value in the evaluation of spinal pain syndromes; however, it has been shown that each case must be carefully assessed, because morphologic abnormalities identified on imaging studies may or may not be the actual cause of the clinical syndrome (1, 2). Indeed, frank disk herniations in the lumbosacral spine may be seen in imaging studies in completely asymptomatic individuals (3–6). Therefore, if an imaging marker could be found that revealed actual underlying neural pathology, and if that pathology proved to correlate with the clinical syndrome, then such a marker might prove useful with regard to the identification and confirmation of the specific cause of the signs and symptoms. This report relates the findings of intravenous gadolinium-enhanced MR of the lumbosacral spine in nonoperated subjects presenting with pain syndromes within the distribution of the sciatic neural plexus.

Materials and Methods

A prospective review was undertaken of 200 adult patients presenting with pain syndromes related to the lumbosacral spine and lower extremities. Five-mm thick T1-weighted (500/15/2, TR/TE/excitations) spin-echo images were obtained in the sagittal and axial planes (angled to the lower three lumbar disks) both before and after the administration of intravenous gadolinium (Magnevist (Berlex Laboratories, Wayne, NJ): 0.1 mmol/kg) on a high-field MR unit (1.5 T). Five-mm thick T2-weighted (2500/30-80/1) spin-echo images were also acquired in the sagittal plane before contrast administration. Evidence was sought for anatomic abnormality (ie, disk herniation) as well as for abnormal intrathecal enhancement of lumbosacral nerve roots after intravenous gadolinium administration. Care was taken not to evaluate for pathologic enhancement either within the neural foramina or more than one 5-mm section below the L5-S1 disk space, so as to avoid confusion of pathologic enhancement with normally enhancing lumbar or sacral dorsal root ganglia. Focal enhancement was defined as that observed at one disk interspace and in the immediately adjacent MR sections. Multilevel enhancement was that observed in areas falling outside of the initially identified interspace. The relevant clinical syndrome was assessed by both chart review as well as an on-site MR questionnaire, which included self-indicated pain diagrams

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that were completed by the patient. In a fashion in which the MR data acquisition was blinded from the clinical data collection, these two parameters were subsequently matched to study the degree of correlation of abnormal lumbosacral root enhancement with the clinical syndrome.

Results

Ten patients out of 200 reviewed revealed abnormal enhancement of lumbosacral nerve root(s) (5%) (Table 1). Six of the patients had enhancing nerve roots in association with intervertebral disk herniation. Five disk herniations lateralized to one side and one was midline (Figs. 1 and 2). One additional patient had an asymmetric lateralizing disk bulge associated with ipsilateral neural and perineural enhancement (Fig. 3). This first group with nerve root enhancement that was linked with protrusive disk disease was designated group I (N = 7). Three patients demonstrated nerve root enhancement in the absence of any anatomic spinal derangement (Fig. 4). This second group of patients who manifested idiopathic radicular enhancement was designated group II (N = 3). In three of the 10 patients, the enhancement was focal (30%), and in seven, the pattern of enhancement extended over multiple spinal segments (70%) (Fig. 5). Two patients showed single root enhancement (20%), and eight revealed multiple enhancing radicles (80%) (Fig. 5).

On further inspection, it was noted that the enhancing nerve roots on MR involved multiple radicles in 100% of the patients in idiopathic group II (N = 3). On the other hand, enhancement involving multiple roots was seen in 71.4% of the group I with protrusive disk disease (N = 5). Multilevel neural enhancement was noted in 100% of the idiopathic group II, and in 57.1% of group I (N = 4).

The overall total number of focal posterior disk protrusions (ie, frank disk herniations, focal disk bulges) in this series was 33. Thus, the seven cases of enhancing nerve roots in this series in group I represented an incidence of enhancement in protrusive disk disease of 21.2% (seven of 33). Of seven midline disk herniations occurring in this study, one showed evidence of abnormal intrathecal neural enhancement (14.3%) (Fig. 2). This compared with five cases of lateralizing disk herniation that demonstrated enhancing nerve roots out of a total of 26 disk herniations that lateralized to one side (19.2%). In group II in which there was no protrusive disk derangement identified, the prevalence root enhancement was 1.5% overall (three/200).

The specific correlation of MR evidence of enhancing nerve roots with the clinical syndrome

TABLE 1: Summary of clinicoradiologic findings in 10 subjects with nerve root enhancement in the unoperated LS spine

Case No.	Age/Sex	Clinical Presentation	Dermatome of Clinical Expression	Duration of Presenting Syndrome	LS Root Enhancement: Level	Additional MR Findings	CSF Findings	Clinical Management	Clinical Follow-Up
1	27/M	LBP, LLE RR; numbness; paresthesias	LLE L4-S2	2 yr	mr: focal sr: multilevel	Lft HNP L4-L5		Conservative	No change
2	34/M	LBP, BLE RR; numbness	BLE L1-S2	2 yr	mr: multilevel			Conservative	No change
3	34/M	LBP, LLE RR; weakness; numbness	LLE S1-S2	3 wk	mr: focal sr: multilevel	Lft HNP L5-S1		Diskectomy	Normal
4	35/M	LBP, RLE RR; weakness; numbness	RLE S1-S2	8 mo	mr: focal	Rt HNP L5-S1		Diskectomy	Normal
5	39/M	LBP, LLE RR	LLE S1	2 yr	sr: focal	Lft LDB L5-S1		Conservative	Moderate improvement
6	40/M	LBP, RLE RR; weakness; numbness; paresthesias	RLE L4-S2	1 yr	mr: focal	Lft HNP L4-L5		Conservative	No change
7	45/M	LBP, LLE RR	LLE S1	6 mo	sr: multilevel	ML HNP L5-S1		Conservative	No change
8	46/F	LBP, LLE RR; weakness; paresthesias	LLE L5-S1	5 mo	mr: multilevel	Lft HNP L4-L5		Conservative	No change
9	54/M	BLE RR; weakness; paresthesias	BLE L1-S2	10 yr	sr: multilevel		Glucose: NL, protein: NL, cell count: NL	Conservative	No change
10	57/M	LBP, BLE RR	BLE L1-S2	3 yr	mr: multilevel			Conservative	Progression

Note.—Legend: M, male; F, female; Rt, right; Lft, left; ML, midline; LBP, low back pain; L, lumbar; S, sacral; RLE, right lower extremity; LLE, left lower extremity; BLE, bilateral lower extremity; mr, multiple root; sr, single root; RR, radiating radiculopathy; HNP, herniated nucleus pulposis; LDB, lateralizing disk bulge; NL, normal; CSF, cerebrospinal fluid; LS, lumbosacral.

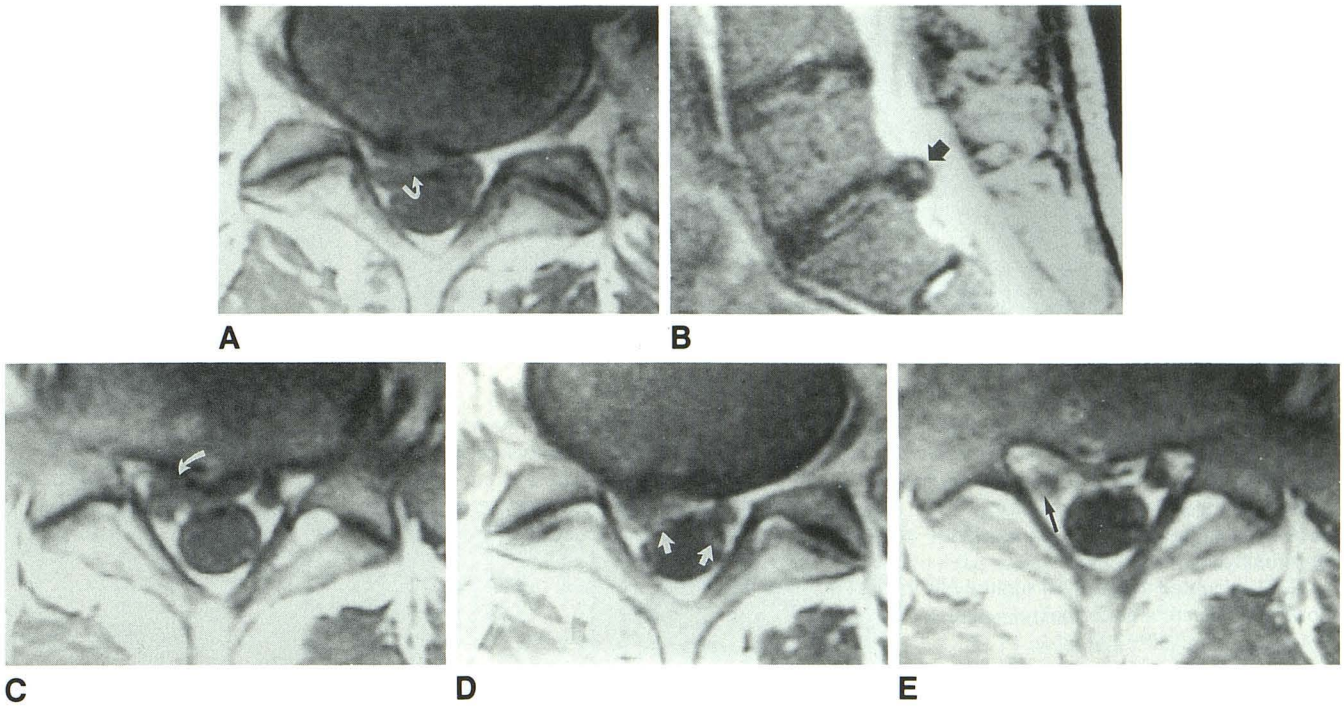


Fig. 1. Case 4. Focal enhancing nerve root associated with ipsilateral disk herniation in subject with right lower extremity radiating radiculopathy and weakness.

A, T1-weighted (600/20/2) axial MR image through the L5-S1 disk demonstrating a lateralized disk herniation (*arrow*).

B, T2-weighted (2000/80/1) right paramedian MR confirming the disk herniation (*arrow*).

C, T1-weighted (600/20/2) axial MR image immediately below the level of the L5-S1 disk showing obliteration of the epidural fat (*arrow*).

D, T1-weighted (600/20/2) axial MR image at the same level as *A* after intravenous gadolinium administration showing multiple enhancing intrathecal nerve roots (*arrows*).

E, T1-weighted (600/20/2) axial MR image at the same level as *C* after intravenous gadolinium administration revealing epidural enhancement. Also identified is focal enhancement of the right S1 nerve root itself within the nerve root sheath (*arrow*).

was excellent. Generally speaking, where multiple enhancing roots were seen on MR, a polyradiculopathy was present in all patients (N = 8). Similarly, single enhancing roots on MR correlated clinically with a monoradiculopathy in all patients in this subset (N = 2). Motor weakness in the appropriate lower extremity(s) was observed in four of the patients in group I (57.1%) and in one of the patients in group II (33.3%). As noted previously, while most instances of enhancing nerve roots were seen in cases of disk herniation, one observation of an enhancing root/sheath was noted in association with a lateralizing disk bulge (Fig. 3). This fit the clinical syndrome that constituted a monoradiculopathy radiating into the appropriate dermatome ipsilateral to the side of the asymmetrically bulging disk. In addition, a case of midline disk herniation showed an enhancing nerve root lateralizing to the side of the clinical radiculopathy (Fig. 2); and one other case which demonstrated unilateral disk herniation but clinically manifested contralateral symptoms, revealed an enhancing root on the same side as the radicular symptomatology (Fig. 6).

In group I, the duration of the clinical lumbosacral syndrome ranged from 3 weeks to 2 years. In group II, the duration of the syndrome ranged from 2 to 10 years. Two of the patients with disk herniation had diskectomies with complete resolution of all signs and symptoms. All of the remaining patients had either moderate improvement, no change, or progression of their clinical syndrome on conservative therapy (Table 1).

Discussion

Laboratory experimentation has shown that there is a definite blood-nerve barrier (BNB) (7–11). This barrier seems to vary somewhat from the cord to the peripheral nerve (10). However, in humans, the intrathecal roots normally have a BNB that for practical imaging purposes ap-

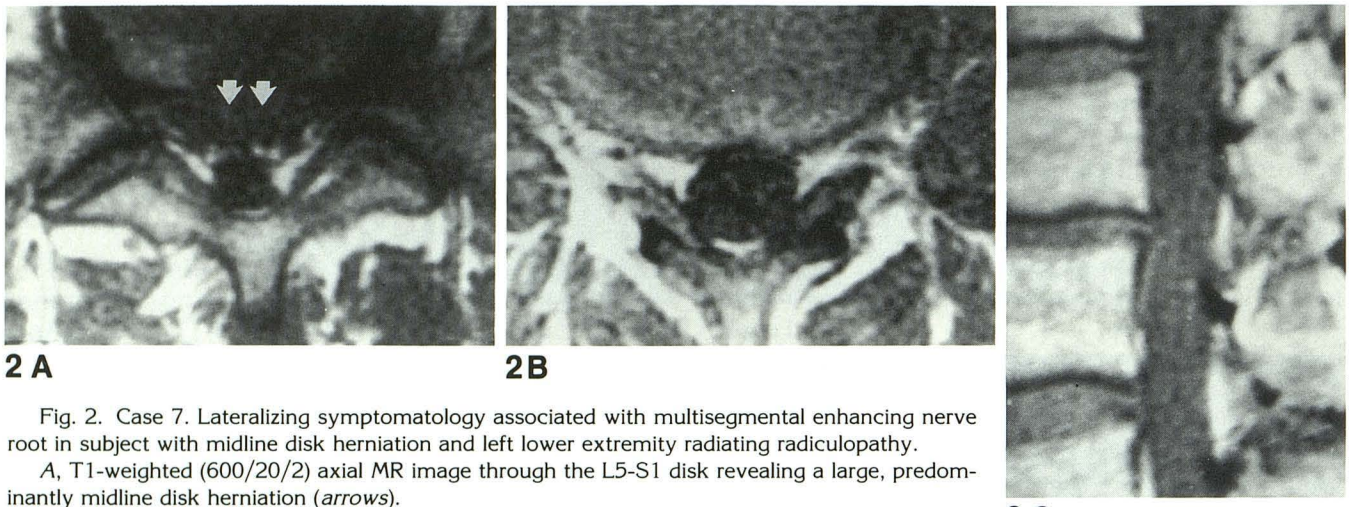


Fig. 2. Case 7. Lateralizing symptomatology associated with multisegmental enhancing nerve root in subject with midline disk herniation and left lower extremity radiating radiculopathy.

A, T1-weighted (600/20/2) axial MR image through the L5-S1 disk revealing a large, predominantly midline disk herniation (*arrows*).

B, T1-weighted (600/20/2) axial MR image at the L3–L4 level demonstrating no discrete abnormality.

C, T1-weighted (600/20/2) sagittal MR image focusing on the region craniad to the L5-S1 level showing no discrete abnormality.

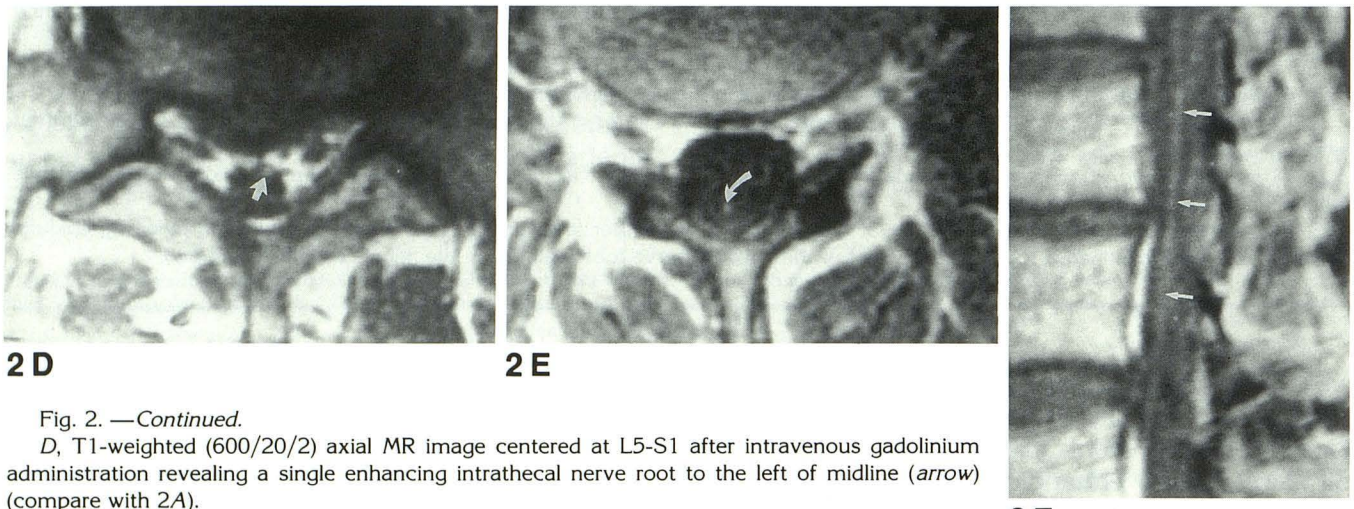


Fig. 2. —*Continued.*

D, T1-weighted (600/20/2) axial MR image centered at L5-S1 after intravenous gadolinium administration revealing a single enhancing intrathecal nerve root to the left of midline (*arrow*) (compare with 2*A*).

E, T1-weighted (600/20/2) axial MR image at the L3-L4 level after intravenous gadolinium administration demonstrating a single root enhancing proximal to the L5-S1 midline disk herniation (*arrow*) (compare with 2*B*).

F, T1-weighted (600/20/2) sagittal MR image acquired above the L5-S1 level after intravenous gadolinium administration revealing faint multilevel nerve root enhancement (*arrows*) (compare with 2*C*).

proaches that of the blood-brain or blood-cord barrier.

Any nonspecific pathologic insult or acquired neural alteration (ie, trauma, ischemia, inflammation, demyelination, axonal degeneration) involving roots or nerves will disrupt this BNB (12–18). In these instances, the injury may allow intravascular water and molecular structures to escape the vasa nervorum and enter the endoneurium. If an intravascular contrast agent (ie, gadolinium) is administered at this time, and the pathologic exit of the agent into the extravascular space of the nerve is in sufficient amounts, then enhancement on imaging studies (ie, MR) may

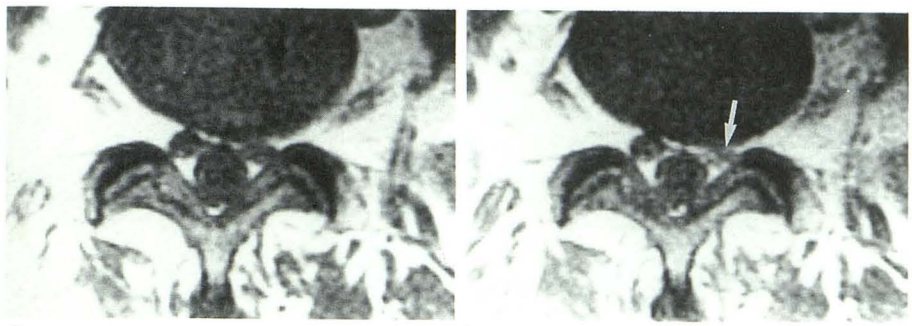


Fig. 3. Case 5. Focal enhancing neural/perineural enhancement associated with lateralized disk bulge in patient with left lower extremity radiating radiculopathy.

A, T1-weighted (600/20/2) axial MR image demonstrating a nonfocal left-sided lateralizing disk bulge.

B, T1-weighted (600/20/2) axial MR image after intravenous gadolinium administration revealing subtle generalized enhancement of the left S1 nerve root and the parent nerve sheath (*arrow*). This results in poor edge discrimination of these structures from the surrounding epidural fat (compare with opposite side and *A*).

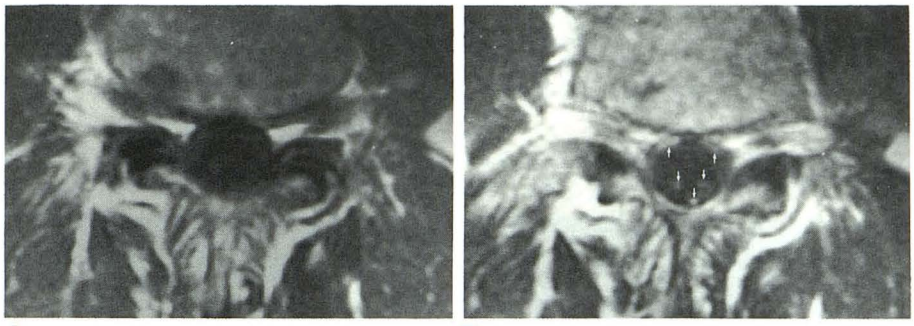


Fig. 4. Case 9. Idiopathic, diffuse multiroot, multisegmental enhancing nerve roots in patient with bilateral lower extremity radiating polyradiculopathy and weakness.

A, T1-weighted (600/20/2) axial MR image demonstrated no discrete abnormality.

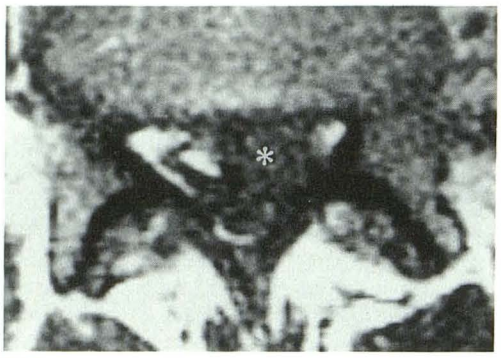
B, T1-weighted (600/20/2) axial MR image after intravenous gadolinium administration revealing multiple enhancing nerve roots (*arrows*).

result. It is postulated that this represents an active *radiculitis* that has previously been directly observed clinically by other researchers both in the presence and in the absence of impinging disk pathology (19–24).

The 5% overall incidence of enhancing nerve roots in the unoperated lumbosacral spine in this study seemed low. However, if the incidence of focal disk protrusion was taken into account (16.5%, N = 33), then the prevalence of pathologic enhancement in the cases of protrusive disk disease in group I (N = 7) was 21.2%. The mechanism of the focal breakdown in the BNB in this situation was almost certainly mechanical. However, some contribution of perineural fibrosis/arachnoiditis adding to the picture of enhancement must also be considered (Fig. 3) (25).

The incidence of idiopathic root enhancement in group II was very low overall (1.5%; N = 3). This small group all had multiple, multilevel enhancing roots (100%). Although of unknown etiology, this enhancement pattern most probably represents a low-grade inflammatory response, and perhaps, therefore, a low-grade active arachnoiditis. Whether due to a potentially identifiable infectious or toxic agent, an autoimmune process, or some other diffuse insult (including neoplastic), the pathologic etiologic factor has not been clinically ascertained in this group of patients at the time of publication.

The general correlation of the cases of enhancing nerve root(s) with the clinical syndrome was excellent. All patients with multiple enhancing roots (N = 8) manifested a polyradiculitis clinically (Fig. 3). The individuals with multiple enhancing roots associated with disk herniation (N = 5) support the theory that a herniation at one level may cause clinical pathology within more than one root (Fig. 5). The patients with singly enhancing roots (N = 2) were both associated with anatomic protrusive disk pathology on MR that visuospatially directly affected the nerve roots. Out of a total of seven subjects with protrusive disk disease, in four the pathology was a disk herniation ipsilateral to the clinical syndrome (Fig. 1). The remaining three cases point out that midline disk herniations, disk herniations contralateral to symptomatology, and even nonherniative lateralizing disk protrusions may be associated with enhancing nerve root(s) that are appropriate to the clinical syndrome (Figs. 2, 3, and 6). As mentioned earlier, some component of local-



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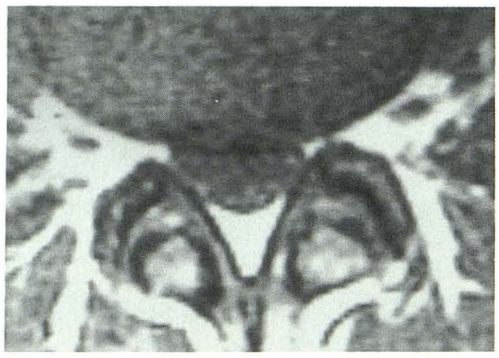

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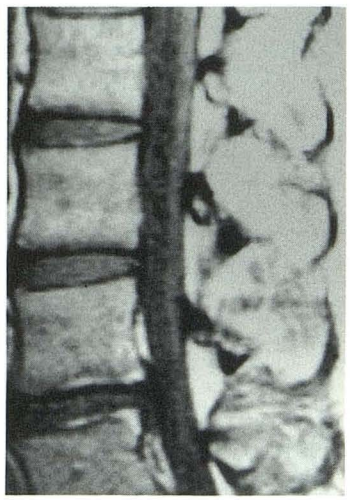


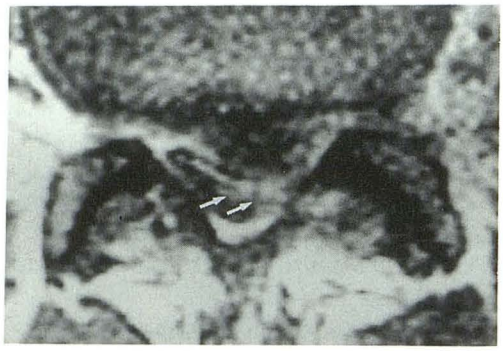
5C

Fig. 5. Case 1. Multiple enhancing roots and multisegmental proximal extension associated with lateralized disk herniation in patient with left lower extremity radiating radiculopathy.

A, T1-weighted (600/20/2) axial MR image at the L4-L5 level showing a large disk herniation on the left (*asterisk*).

B, T1-weighted (600/20/2) axial MR image at the L3–L4 level demonstrating a moderately narrowed canal and a generalized disk bulge.

C, T1-weighted (600/20/2) sagittal MR image focusing on the region craniad to the L5-S1 level.



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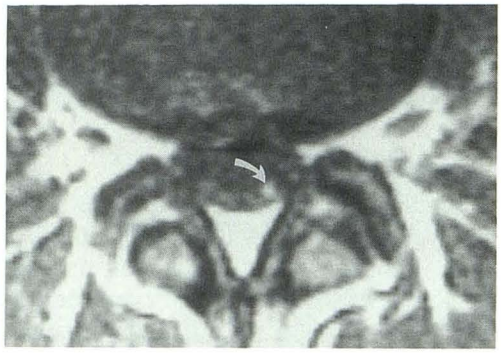

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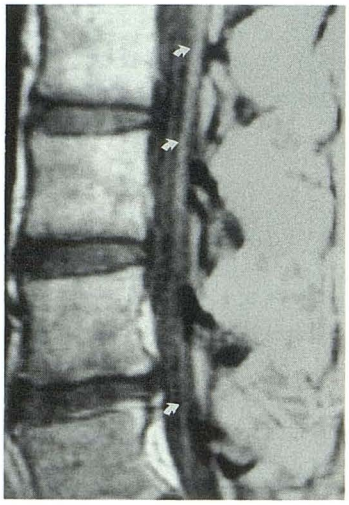

5F

Fig. 5. —Continued.

D, T1-weighted (600/20/2) axial MR image at the L4-L5 level after intravenous gadolinium administration showing multiple enhancing intrathecal nerve roots toward the left side (*arrows*) (compare with 5*A*).

E, T1-weighted (600/20/2) axial MR image at the L3-L4 level after intravenous gadolinium administration demonstrating a single root-enhancing proximal to the left-sided L5-S1 disk herniation (*arrow*) (compare with 5*B*).

F, T1-weighted (600/20/2) sagittal MR image focusing on the region above the L5-S1 level after intravenous gadolinium administration revealing multilevel nerve root enhancement (*arrows*) (compare with 5*C*).

ized arachnoiditis must be considered as a hypothetical contributing cause of neural/perineural enhancement engendered by the trauma of a protruding disk (25, 26).

It should be noted that, although the numbers were small, these cases of enhancing nerve roots nevertheless constituted an important patient group. First, for obvious reasons, the false negative rate for neural enhancement, and thus nerve root pathology, would be 100% without the use of intravenous gadolinium. Second, in none of the cases of idiopathic radicular enhancement (N = 3), nor in the three cases of root enhancement associated with lateralizing disk bulge, midline

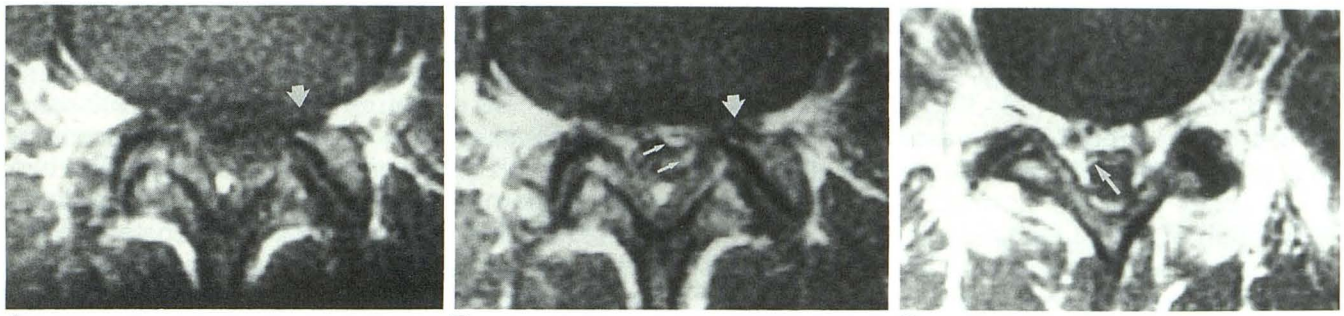

Fig. 6. Case 6. Multiple enhancing nerve roots in subject with disk herniation lateralizing to the left but with contralateral right lower extremity radicular symptomatology.

A, T1-weighted (600/20/2) axial MR image at the L4-L5 level showing a somewhat stenotic spinal canal and a suggestion of a mass in the left neural foramen (*arrow*).

B, T1-weighted (600/20/2) axial MR image at the L4-L5 level after intravenous gadolinium administration revealing multiple enhancing intrathecal nerve roots (*small arrows*). The nonenhancing left-sided disk herniation is also identified (*large arrow*).

C, T1-weighted (600/20/2) axial MR image at the L5-S1 level after intravenous gadolinium administration demonstrating intense enhancement of at least one intrathecal root (*arrow*) to the right of midline.

disk herniation, and disk herniation contralateral to symptoms would the apparent pathology responsible for inciting the clinical syndrome (ie, radicular/periradicular inflammation) have been certain without the enhanced study. This totaled 60% of the cases of enhancing nerve roots in this series. In the other 40%, the enhancing neural radicle(s) was intimately associated with the offending lateralizing frank disk herniation, and, therefore, the diagnosis of the anatomic pathology responsible for provoking the clinical sympatology would have been made correctly even without the use of an enhancing agent.

The mechanism for the production of conscious pain in insulted nerve roots theoretically revolves around a fundamental pathologic change in the axonal membrane. The axon theoretically becomes in itself an ectopic source of neurelectrical impulses. The central nervous system interprets this as pain and paresthesias within the distribution of the insulted axon(s). This is a type of *pathologic pain*, in that it is a pain impulse that is generated within axons that are hypermechanosensitive, it is a pain impulse that is ectopic in origin (the impulse is not initiated within a true pain receptor), and it is a pain impulse that is far larger in proportion and duration than would be anticipated originating from the mechanical perturbation of a normal, uninflamed, or uninjured axon. This same pathologic neuroelectrical hyperactivity might also be expected in idiopathic diffuse arachnoidal inflammation involving single or multiple nerve roots (27). The abnormal enhancement of nerve roots on MR associated with a breakdown in the BNB may serve as a visual marker for such pathology.

It is important to consider why many of the disk herniations did *not* show abnormally enhancing nerve roots. First, it may be that many of these disk herniations did not significantly impinge on a nerve root(s). This could certainly be true of some midline disk herniations, especially those at the L5-S1 disk level that typically has a sizable anterior epidural space that is largely free of spinal nerve roots. This space might harbor even a sizable disk herniation without actual nerve root compression. On the other hand, some of the disk herniations observed on MR not associated with enhancing nerve roots likely represented truly asymptomatic lesions and, therefore, were clinically irrelevant (3–6). However, consideration must also be given to the possibility that the enhanced MR in some instances was falsely negative with regard to its potential to signal the presence of a clinical radiculopathy.

It should be stated hypothetically that enhancement was *not* always a prerequisite for a clinical radiculopathy for several reasons. First, focal neural enhancement was probably temporally related to the insult. Thus, the period of enhancement may overlap but not precisely coincide with the period of symptoms. At a given time after the injury, enhancing root(s) likely represented pathologic change that was over and above that of a more simple insult not linked with a BNB disruption. The findings on MR are partly a function of the degree of injury and the timing of the MR examination. Experimentally, it has been

demonstrated that following a traumatic insult to the spinal nerve root, a BNB breakdown occurred at the site of neural injury and extended for a short distance distally (28–30). This was at least partially due to focal frank vascular damage resulting in intraneurally extravasated contrast media coupled with the normal intraneural distal convection of interstitial fluid/contrast.

Second, it was also shown in laboratory models of nerve crush that the degree and site of BNB breakdown directly accompanied the extensive wallerian degeneration and subsequent axon regeneration associated with some cases of nerve injury (16–18). That this change was not seen to extend proximally in the laboratory experiments may reflect the fact that the injuries being studied were within the spinal nerves. This injury was induced distal to the bipolar sensory cell bodies within the dorsal root ganglia, and since the motor neurons are also proximal to this peripheral nerve crush (ie, within the cord), wallerian degeneration extending proximally would not be expected. The typical areas of disk impingement and neural enhancement in this series, however, were within the spinal *roots*. In this circumstance, the peripheral axonal processes of both motor cells (within the ventral roots caudal to the impingement) and sensory cells (within the dorsal roots cranial to the impingement) would be affected. Thus, perhaps both proximal and distal enhancement on MR can be partly explained on the basis of active *bidirectional* wallerian degeneration. In this way, areas of neural enhancement remote from the actual site of injury may indicate a process associated with axonal degenerative/regenerative phenomena, but *not* necessarily linked with abnormal ectopic axonally generated action potentials and, therefore, clinical pain and dysesthesias. Plausibly, remote neural enhancement related to axonal degeneration may nevertheless be allied with motor axon dysfunction and clinical weakness/reflex dysfunction. The extent of the change observed on enhanced MR should parallel the degree of the primary injury, the secondary changes initiated by the injury (ie, edema, swelling, fibrosis, degeneration, regeneration, etc), and the phase of the process in which MR imaging takes place. Allowances should also be made in interpretation for differences in tissue reaction response that are unique to the individual, and that will affect the clinicoradiologic presentation.

Third, regarding the lack of observed enhancement in some symptomatic patients, consideration must be given for the amount of contrast administered. It is not clear at this time what dosage of gadolinium is ideal (eg, 0.1, 0.2, 0.3 mmol/kg) for the optimal imaging of pathologic enhancement of spinal nerve roots.

As can be seen from these considerations, it would be an error to equate neural enhancement directly with radicular symptomatology. The two are fundamentally different pathophysiologic processes that may, in certain circumstances, be temporally and spatially associated. On these grounds, it is reasonable to assume that the two may also become dissociated in any neural structure, at any given time.

Whether or not patients with enhancing nerve roots will require additional and/or different treatment than those without such enhancement (eg, anti-inflammatory agents with surgery or anti-inflammatory agents alone) remains to be elucidated. An array of possible avenues aimed at reducing the pathologic increase in functional sodium channel density in the axon membrane believed to be responsible for pathologic pain have been considered. These include corticosteroids that may function by directly reducing the functional sodium channel spatial density, axoplasmic transport blockers (ie, colchicine, vinblastine) which prevent the macromolecular sodium channel units from being transported from the neuron cell body to the axonal site of insult, and direct sodium channel blockers (ie, lidocaine) (31–40).

A final comment should be made regarding the discrepancy in statistics between patients with nerve root enhancement who were unoperated and personal observations of patients who were postoperative with signs and symptoms related to the lumbosacral neural plexus. There was approximately a four times higher incidence in the postoperative group than that found in the unoperated patient population studied in the present report. Even if some of the radicles hypothetically enhanced preoperatively, and, therefore, the enhancement seen postoperatively transcended the surgery, the incidence was still greater than expected for undetermined reasons.

Because a 5% prevalence of enhancing nerve roots in the unoperated lumbosacral spine makes it inefficacious to enhance all patients presenting to MR for evaluation of lumbosacral pain syndromes, some recommendations for enhancement gained from the experience with this group of patients may be valuable. Any individual who presents with clinically reliable evidence of a mono- or polyradiculopathy, but who does *not* demonstrate a correlative anatomic abnormality on unenhanced MR (ie, *no* ipsilateral/isolevel disk

herniation, osseous foraminal stenosis, clumped/adhesed nerve roots, etc), or alternatively, any individual who presents with a clinical syndrome that does not match the anatomic abnormality(s) on the unenhanced MR, (ie, contralateral/remote/multilevel disk herniation(s), etc) should be considered for further study. In these patients, intravenous gadolinium-enhanced MR may reveal the true nature and location of the pathologic neural alteration responsible for the clinical syndrome that potentially can thereby dictate a more specific treatment regimen.

In summary, gadolinium-enhanced MR has shown abnormally enhancing intrathecal nerve roots in the lumbosacral spine with an overall incidence of 5%. In the 10 subjects manifesting enhancing root(s), 70% were associated with focally protrusive anatomic abnormalities of the intervertebral disk (ie, frank disk herniation or lateralizing disk bulge); this constituted 21.2% of all focal disk protrusions as judged by MR. In the other 30%, the etiology responsible for the neural enhancement was not ascertained; but as these cases all revealed enhancement of multiple roots at multiple levels, it was felt that they most probably represented idiopathic low-grade inflammation. In none of the cases of nerve root enhancement in this series could the diagnosis of specific radicular pathology be made without the use of intravenous gadolinium. The therapeutic strategies for the treatment of clinically correlative BNB disruption as identified on MR remain to be elucidated.

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