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Extradural inflammation associated with annular tears: demonstration with gadolinium-enhanced lumbar spine MRI

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Abstract Annular tears are manifest on MRI as the high-intensity zone (HIZ) or as annular enhancement. Patients with annular tears may experience low back pain with radiation into the lower limb in the absence of nerve root compression. Inflammation of nerve roots from leak of degenerative nuclear material through full-thickness annular tears is a proposed mechanism for such leg pain. The aim of this study is to illustrate the appearance of extradural enhancement adjacent to annular tears in patients being investigated for low back pain with radiation into the lower limb(s). Sagittal T1- and T2-weighted spin echo and axial T1-weighted spin echo sequences were obtained in eight patients being investigated for low back and leg pain. In all patients, the T1-weighted sequences were repeated following intravenous gadopentetate dimeglumine (Gd-DTPA). Annular tears were identified at 12 sites in eight patients. Extradural inflammation appeared as a region of intermediate signal intensity replacing the fat between the posterior disc margin and the theca, which enhanced following Gd-DTPA. The inflammatory change was always associated with an annular tear, and in four cases directly involved the nerve root. Enhancement of the nerve root was seen in two cases. The findings may be relevant in the diagnosis of chemical radiculopathy secondary to inflammation at the site of an annular leak from a degenerating disc.

Key words MRI · Intervertebral disc · Low back pain · Sciatica · Discography

Introduction

MRI is established as the investigation of choice for assessing patients presenting with low back pain (LBP) and sciatica [3]. Recognised causes of nerve root compression include disc herniation and spinal stenosis. However, many patients presenting with LBP and pain referred to the lower limb(s) show no evidence on imaging studies of nerve root compression [14, 15, 20], suggesting that there are other factors involved in the production of leg pain ('sciatica'). Inflammation of nerve roots (chemical radiculopathy) adjacent to annular tears has been suggested as a cause of sciatica [13], but there is little supporting evidence from imaging studies in humans.

This paper describes a new sign on gadolinium-enhanced MRI that we observed in patients presenting with LBP and leg pain, which may be of relevance to the diagnosis of discogenic low back pain and chemical radiculopathy.

Materials and methods

In a prospective study over a 26-month period we identified eight patients presenting with LBP and pain referred to the leg(s) whose lumbar spine MRI studies demonstrated appearances in the extradural space that mimicked those of postoperative fibrosis, namely effacement of extradural fat by material of intermediate signal intensity (SI) on axial T1-weighted spin echo (WSE) sequences (Fig. 1), which enhances following administration of in-

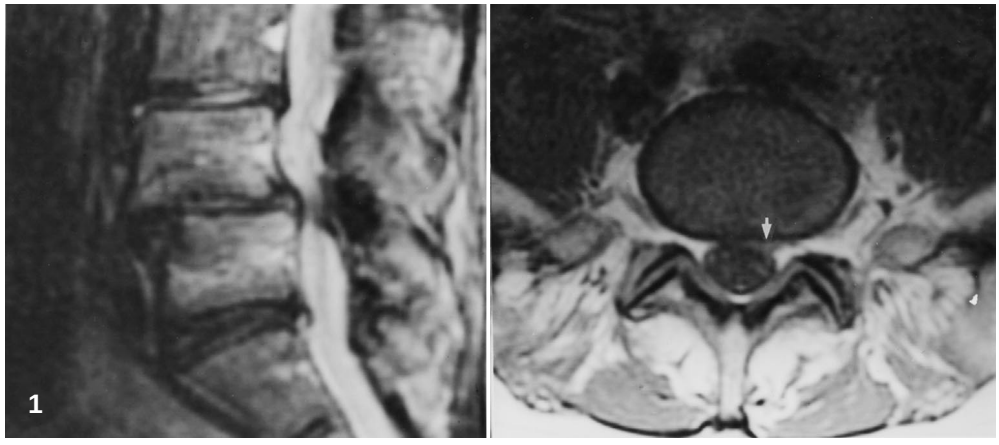

Fig. 1 A, B Case 6. **A** Midline sagittal T2-weighted spin echo (WSE) sequence showing high intensity zones (HIZs) at the L4/5 and L5/S1 levels, with grade I degenerative spondylolisthesis at L4/5. **B** Axial T1 WSE sequence through the L5/S1 disc showing effacement of the extradural fat on the left side between the posterior disc margin and the theca *(arrow)*

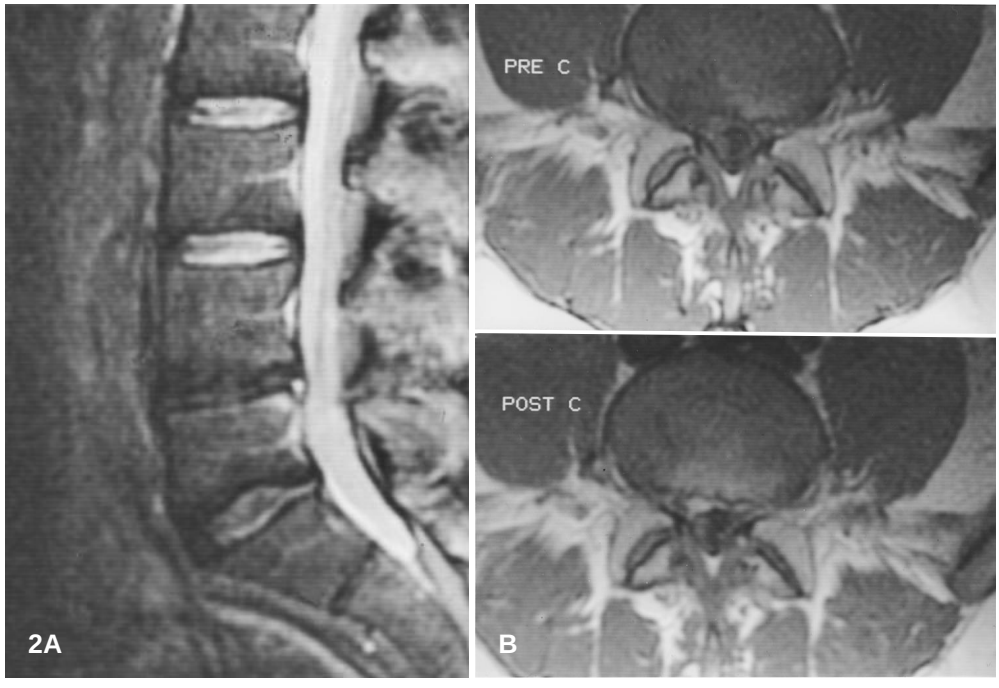

Fig. 2 A, B Case 8. **A** Sagittal T2 WSE sequence to the right of midline showing disc degeneration at L4/5 and L5/S1 with a HIZ in the posterior annulus of the L4/5 disc. **B** Axial T1 WSE sequences at the L4/5 level before *(top)* and after *(bottom)* Gd-DTPA. The extradural fat in the right lateral recess is replaced by tissue of intermediate signal intensity (SI), which enhances uniformly

travenous Gd-DTPA (Figs. 2, 3) [7, 10, 11]. The abnormality typically occurs between the posterior disc margin and the thecal sac at the level of the disc. It may be central or lateral, is always associated with a degenerative disc and occurs adjacent to an annular tear. The latter is manifest either as a high-intensity zone (HIZ) as defined by Aprill and Bogduk [2] or by enhancement in the annulus [15, 17, 18].

Five patients had undergone no previous spinal surgery. Two of the eight patients had undergone previous laminectomy and discectomy and one had undergone previous automated percutaneous lumbar discectomy (APLD). All patients were imaged at 0.5 T using a dedicated lumbar spine surface coil. Sagittal T1 (TR 520, TE 25) and T2 (TR 2000, TE 90) WSE sequences and axial T1 (TR 720, TE 25) WSE sequences were performed in all cases. In all patients, the axial T1 WSE sequences were repeated following intravenous injection of 10 ml Gd-DTPA (Magnevist, Schering, Berlin). In three of these cases, the sagittal T1 WSE sequences were also repeated following Gd-DTPA. Pre- and post-contrast studies were imaged at identical window settings. Three patients were subsequently referred for lumbar discography.

Results

The clinical details and MRI findings are summarized in Table 1. Annular tears manifest by the HIZ were identified at 11 sites in seven patients. In the remaining case, the tear was identified as a region of enhancement in the posterior annulus. In all eight cases, tears were associated with extradural inflammation, which was limited to the side of the referred leg pain in six cases. In four cases, the enhancing tissue directly involved the adjacent nerve root. In two cases, the extradural inflammation was midway between the disc and theca. Enhancement of adjacent nerve roots was identified in two patients, both of whom had not had previous surgery.

Three patients (2, 6, and 7) had undergone discography, which revealed concordant pain reproduction at the

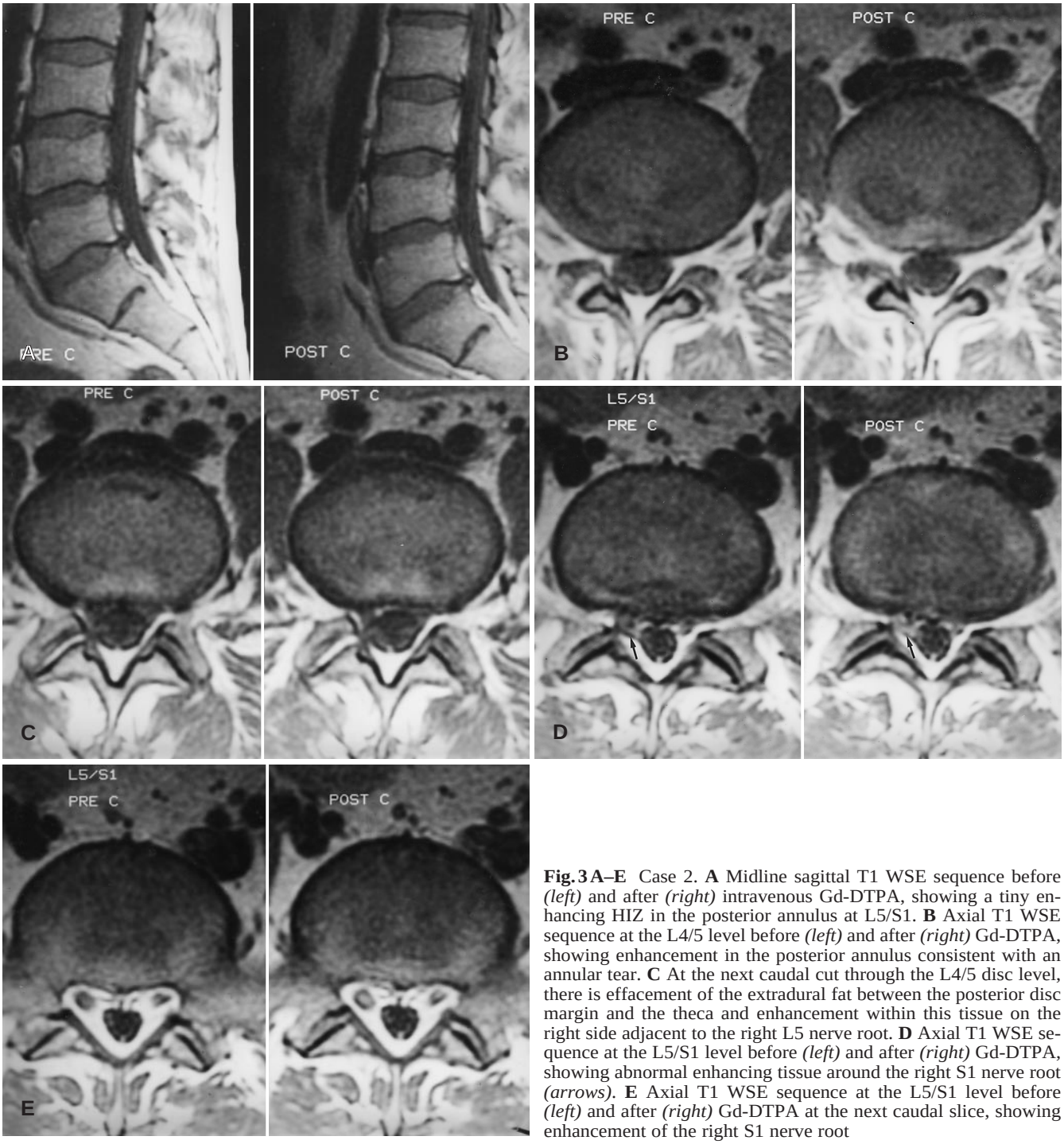

Fig. 3 A–E Case 2. **A** Midline sagittal T1 WSE sequence before *(left)* and after *(right)* intravenous Gd-DTPA, showing a tiny enhancing HIZ in the posterior annulus at L5/S1. **B** Axial T1 WSE sequence at the L4/5 level before *(left)* and after *(right)* Gd-DTPA, showing enhancement in the posterior annulus consistent with an annular tear. **C** At the next caudal cut through the L4/5 disc level, there is effacement of the extradural fat between the posterior disc margin and the theca and enhancement within this tissue on the right side adjacent to the right L5 nerve root. **D** Axial T1 WSE sequence at the L5/S1 level before *(left)* and after *(right)* Gd-DTPA, showing abnormal enhancing tissue around the right S1 nerve root *(arrows)*. **E** Axial T1 WSE sequence at the L5/S1 level before *(left)* and after *(right)* Gd-DTPA at the next caudal slice, showing enhancement of the right S1 nerve root

sites of the annular pathology. The remaining patients were not considered surgical candidates and were not referred for discography. One of these (no. 4) had a very successful response to caudal epidural.

Both patients who had undergone previous laminectomy and discectomy had evidence of postoperative fibrosis involving the adjacent nerve root at the site of surgery. However, they also had evidence of extradural inflammation at sites distant from their surgery but associated with annular tears.

Table 1 Clinical details and imaging findings (*LBP* low back pain, *Lt* left, *Rt* right, *APLD* automated percutaneous lumbar discectomy, *SLR* straight leg raising)

Case no.	Age/sex	History	Neurological examination	MRI findings
1	47/F	LBP, Lt leg pain to ankle. Previous APLD. Benefit from caudal epidural	No neurological deficit	Degenerate L4/5 disc. Central extradural inflammation
2	38/M	LBP, Rt leg pain to foot. Benefit from caudal epidural	Decreased SLR bilaterally. Global decrease in power Rt ankle	Degenerate L3/4, L4/5 and L5/S1discs. Posterior annular tears L4/5 and L5/S1. Extradural inflammation L4/5 (Rt) and L5/S1 (Rt). Enhancement Rt S1 nerve root.
3	36/F	LBP, bilateral leg pain (Lt > Rt). Previous Rt L5/S1discectomy and decompression	Decreased SLR bilaterally. Decreased sensation Rt calf. Decreased Rt ankle jerk	Degenerate L4/5 and L5/S1 discs. Posterior annular tear L4/5. Extradural inflammation L4/5 (Lt). Postoperative scar L5/S1 (Rt) involving Rt S1 nerve root
4	50/F	LBP, bilateral leg pain	Reduced sensation Rt foot. Absent Rt ankle jerk	Degenerate L3/4, L4/5 and L5/S1 discs. Posterior annular tear L4/5. Extradural inflammation L4/5 (Lt). Rt paracentral L5/S1 disc hernia compressing S1 root
5	39/F	LBP, bilateral leg pain	Decreased SLR Lt. Decreased sensation Lt foot. Decreased Lt ankle jerk	All discs degenerate, maximal L4/5 and L5/S1. Posterior annular tear L4/5 and L5/S1. Extradural inflammation L5/S1 (Lt). Enhancement of Lt S1 root
6	55/F	LBP, Lt leg numbness. Bilateral buttock pain	No neurological deficit	Degenerate discs L3/4, L4/5 and L5/S1. Posterior annular tear L4/5 and L5/S1. Extradural inflammation L5/S1 (Lt). Degenerative spondylolisthesis L4/5 with central spinal stenosis
7	30/F	LBP and Lt leg pain. Previous L5/S1 discectomy × 2	No neurological deficit	Degenerate discs L3/4, L4/5 and L5/S1. Posterior annular tears L3/4 and L4/5. Central extradural inflammation L4/5. Postoperative fibrosis L5/S1 involving Lt S1 root
8	49/M	LBP, Rt groin and leg pain	No neurological deficit	Degenerate discs L4/5 and L5/S1. Rt posterior annular tear L4/5. Extradural inflammation Rt L4/5 level involving Rt L5 root

Discussion

Crock [5, 6] promoted the concept of 'internal disc disruption', suggesting that trauma to the intervertebral disc resulted in the production of inflammatory substances within the nucleus pulposus that could have local autoimmune effects causing back pain, and chemical effects on the adjacent nerve roots resulting in leg pain, but typically no neurological deficit. Such clinical symptoms are often referred to as 'discogenic' in nature. Adams et al. [1], in cadaver studies, showed that contrast medium could leak through full-thickness annular tears after intradiscal injection. Such leak of contrast medium with involvement of adjacent nerve roots has been demonstrated at lumbar CT discography and correlates with the reproduction of leg pain [13]. Experimental studies have shown that nucleus pulposus injected into the extradural space can result in nerve root inflammation [12, 16]. The possibility of such a chemical radiculopathy in humans therefore exists but, as yet, there is little supporting evidence in terms of imaging studies. Enhancement of nerve roots on MRI has been identified in patients with lumbar disc herniations [22–24] and following lumbar discectomy [11], although the exact relevance of this is not known.

Annular tears are manifest on MRI as the HIZ, a focus of increased SI in the posterior annulus of a degenerative lumbar intervertebral disc [2]. The HIZ has been shown to have a very high specificity for a concordantly painful radial annular tear at lumbar discography [2, 19, 20], although the sensitivity of the sign is low [19]. Annular tears have been identified as the only abnormality (apart from disc degeneration) in patients presenting with acute lumbar radiculopathy [15].

Extradural fibrosis is a well-recognised finding with acute lumbar disc herniation [15, 17] and is considered to be a useful sign of disc sequestration. Also, a recent study looking at enhanced MRI of the postoperative lumbar spine suggested that postoperative extradural fibrosis was seen only following discectomy for disc herniation and not following nerve root decompression due to lateral recess or foraminal stenosis [25], even if nerve roots had been retracted prior to partial facetectomy. It was suggested that fibrosis occurred as a consequence of the defect in the posterior annulus allowing escape of nuclear material into the extradural space. A similar mechanism may be responsible for the development of the extradural inflammatory tissue demonstrated in the cases presented in this study. In this situation, it is suggested that degener-

ative nuclear material leaks into the extradural space through a full-thickness posterior annular tear. In all of the patients, the inflammatory reaction was present in the anterior extradural space adjacent to a posterior annular tear.

In the patients who had undergone previous spinal surgery, enhancing tissue consistent with extradural fibrosis was seen in two of them. However, they also exhibited extradural enhancement at sites distant from the surgical defect, both in terms of the operated level and, in one case, the side. It was therefore felt that the latter changes could not be attributed to the previous laminectomy. In the patient who had undergone a previous APLD, extradural postoperative fibrosis would not be an expected complication and therefore could not explain the extradural enhancement in this case. The fact that we have observed this sign so infrequently may be due to these patients representing the most severe end of the spectrum of discogenic low back pain. Also, the lack of routine use of gadolinium enhancement for patients who have not had previous surgery may be a factor. Minor effacement of extradural fat on unenhanced T1 WSE sequences may be difficult or impossible to identify, especially adjacent to a bulging disc. Also, it is unlikely that such changes would be easily identifiable on T2-weighted fast spin echo axial sequences without fat suppression, since both inflammatory changes and fat would appear hyperintense. It is possible that the use of gadolinium-enhanced fat-suppressed T1-weighted axial sequences would increase the identification of this sign, as is the case with postoperative fibrosis [8].

Extradural enhancement on post-gadolinium lumbar spine MRI may be seen in a variety of situations. Following surgery for disc herniation, postoperative fibrosis is accurately differentiated from recurrent or residual disc material [7, 10, 11]. Enhancement can also be seen around the margins of herniated disc fragments due to ingrowth of vascularised granulation tissue [15, 17]. Extradural extension of vascularised tumour and extradural abscess will enhance following gadolinium [7, 11]. Engorged extradural venous plexus in patients with multilevel spinal stenosis has also been identified as a cause of enhancement in the anterior extradural space [25], appearing as a bilobed mass at the level of the vertebral body, lying between the vertebra and the posterior longitudinal ligament. This should not be mistaken for an inflammatory mass. Spontaneous extradural haematoma can result in masses within the extradural space adjacent to posterior annular tears [9]. However, the imaging features of these are similar to those of acute disc herniation and are different to the appearances described here for presumed extradural inflammation.

Extradural fibrosis may follow spontaneous resolution of disc herniation. However, we do not feel that this could be the cause for the appearances in the present cases, for a number of reasons. Firstly, resolution of disc herniation in patients treated conservatively is significantly associated with improvement or resolution of symptoms [4, 15]. The patients in the present study were all imaged because of prolonged symptoms that had not responded to conservative treatment. Secondly, it is unusual for disc herniations to resolve completely [4, 15]. There was no evidence in the present cases of disc material in the spinal canal. Finally, studies looking at MRI of resolving disc herniation have not documented the presence of the HIZ on follow-up examinations.

Conclusions

We have described a new sign on enhanced lumbar spine MRI, which we believe represents extradural inflammation adjacent to peripheral radial annular tears. This sign may be relevant in the diagnosis of discogenic low back pain and chemical radiculopathy secondary to inflammation at the site of an annular leak from a degenerating disc. However, further long-term studies are required with larger numbers to determine the prevalence of this finding and to clarify the relationship to clinical features and the possible impact on patient management. Also, we suggest that the apparent rarity of this sign does not justify the routine use of gadolinium in the non-operated lumbar spine.

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