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# Vertebral body MRI related to lumbar fusion results

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**Abstract** The evaluation of continued pain after a technically successful posterolateral lumbar spine fusion is often challenging. Although the intervertebral disc is often a source of low back pain, abnormal endplates may also be a focus of pain, and possibly a source of continued pain after a posterolateral fusion. MRI allows noninvasive evaluation for disc degeneration, as well as for abnormal endplates and adjacent vertebral body marrow. Previous studies have found inflammatory marrow changes, adjacent to abnormal endplates, associated with disc degeneration in low back pain patients. In this study, preoperative MRI scans in 89 posterolateral lumbar fusion patients were reviewed, by an independent radiologist, to determine whether vertebral body marrow changes adjacent to the endplates were related to contined pain. Independent chart review and follow-up telephone interview of all patients at a 4-year follow-up (mean) formed the basis for the clinical results. Vertebral body MRI signals consistent with inflammatory or fatty changes were found in 38% of patients, and always occurred adjacent to a degenerated disc. Inflammatory MRI vertebral body changes were significantly related to continued low back pain at $P = 0.03$. We conclude that posterolateral lumbar fusion has a less predictable result for the subset of degenerative disc patients with abnormal endplates and associated marrow inflammation. More research is needed to determine the biological and biomechanical effects of a posterolateral fusion upon the endplate within the fused segments. If indeed further study supports the hypothesis that abnormal endplates associated with inflammation are a source of pain, then treating the endplates directly by anterior fusion may be a preferred treatment for this subset of degenerative patients.

**Key words** Endplate · MRI · Disc degeneration · Pain · Fusion

## Introduction

Low back pain has a multifactorial etiology that includes irritation of the lumbar spinal nerve roots, paraspinal muscles, facet joint capsules or ligaments; degenerative disease of the intervertebral discs or facet joints; and bony vertebral lesions. Pain due to a degenerated disc is most commonly attributed to mechanical irritation of the innervated anulus fibrosis, especially its posterior and posterolateral peripheral aspects. The effects of a degenerated disc upon the endplate as a source of pain has received less attention.

The diagnosis of pain related to a degenerated disc is based primarily on history and physical examination. MRI and provocative discography can assist the surgeon in the determination of the exact level of a symptomatic intervertebral disc. Pain at the time of provocative discog-

raphy has usually been attributed to the disc anulus, which has been shown to be innervated at its outer layers [4]. Yet, some patients have severe discographically concordant pain and normal disc morphology [24, 27]. For a subgroup of patients, the pain at discography has been related to fissures and erosions of the endplates [6, 13]. Altered signals have also been found adjacent to the endplates of vertebral bodies of degenerative spines, indicating changes in the relative amounts of lipid or hydration [7]. These inflammatory MRI changes adjacent to degenerated discs have been correlated histologically with inflammatory fibrovascular tissue at sites of endplate fissures [26, 33]. A recent study involving MRI scans found a strong correlation between severe relentless low back pain in patients with endplate disruption and adjacent marrow inflammation [33]. Short-term pain relief by intradiscal injection of local anesthetic or cortisone has been reported [9, 10]. Anatomically, this beneficial effect may be due to the fact that the endplate is closer than the peripheral anulus to the injection site. Fissures in the endplates would allow egress of the therapeutic agent to the inflamed adjacent marrow.

Posterolateral segmental lumbar fusion is a common surgical treatment for severe degenerative disc disease and is considered to carry less risk of surgical complications than fusion by an anterior approach. Unfortunately, some patients continue to have significant symptoms despite a posterolateral fusion procedure. A previous study of posterolateral fusion patients with degenerative conditions determined that, in patients with persistent pain, the most common reasons for late reoperation were pseudarthrosis and adjacent segment degeneration [5]. In some cases, a source of residual back pain can not be identified; this may be frustrating and disabling to the patient and a challenge to the treating surgeon. Persistent symptoms are sometimes attributed to presumed residual motion or stresses of the disc. A posterolateral fusion decreases motion and strain on the disc, but the stresses on the endplate are poorly understood.

Some authors advocate an anterior procedure for patients in whom posterolateral fusion surgery has failed; others advocate an anterior or combined anterior and posterior fusion as the index procedure [3, 17, 22]. The choice between an anterior or posterior primary fusion for degenerative disc disease is usually based upon opinion rather than on science. Clinical results could be improved if the surgeon could select preoperatively not only those patients in whom a fusion is likely to succeed, but also those who would benefit most from an anterior vs a posterior procedure. Endplate disruption as a possible source of pain has received only scant attention. In particular, if inflammation secondary to an abnormal endplate is a source of pain, should one perform an anterior fusion? Furthermore, if the endplates are a source of pain, can they be assessed in a noninvasive manner?

The purpose of the present study was to determine whether MRI vertebral body endplate changes had clinical relevance to the results of lumbar fusion patients. Specifically, the possible relationship of preoperative lumbar spine inflammatory or fatty MRI changes to the outcome of posterolateral lumbar fusion surgery, in terms of continued low back pain, was determined.

## Materials and methods

This retrospective study was performed at one of the authors' teaching institutions, the Twin Cities Scoliosis and Spine Center. All charts were reviewed for lumbar or lumbosacral procedures during the 3-year period of December 1987 through December 1990. This identified 156 adult patients who had undergone a primary lumbar fusion procedure for mechanical low back pain related to degenerative spine disease. All patients had their charts and imaging studies reviewed, and these are detailed in another study [5]. They were categorized into four major diagnostic groups: high-grade (> 50% slippage) spondylolisthesis (high slip), low-grade spondylolisthesis and spondylolysis (low slip), degenerative disc disease (DDD), and post-discectomy (post-disc). During the study period, the philosophy of the four attending surgeons at the Twin Cities Scoliosis and Spine Center was to perform posterolateral lumbar fusions for incapacitating mechanical low back pain.

Lumbosacral MRI scans were obtained in 90 patients (58%) at an average of 5.6 months before surgery, and these form the basis for the present study. Of these 90 patients, the primary diagnosis in 22 was spondylolysis and/or low grade spondylolisthesis, and in 4 it was spondylolisthesis of grade III or greater. Degenerative disc disease was the primary diagnosis in 23 patients, and 41 patients had undergone a prior lumbar discectomy (17 had undergone multiple prior discectomies). All 90 patients had been treated with posterolateral lumbar fusions. Posterolateral fusions had been performed from L5 to the sacrum in 26 patients, from L4 to the sacrum in 48 patients, from L3 to the sacrum in 3 patients, and from L2 to the sacrum in 2 patients. "Floating" fusions had been performed from L4 to L5 in six patients, from L3 to L5 in one patient, from L3 to L4 in three patients, and from L2 to L4 in one patient. Of the 90 patients who had undergone a posterolateral fusion, 79 had been instrumented. Eighty-two percent of patients had undergone a decompression at the time of their fusion for lower extremity symptoms.

Chart review of each patient addressed pre- and postoperative symptoms (back pain, thigh pain, and leg pain below the knee to the foot). Clinical and radiographic findings were identified including radicular signs (sensory changes in a reproducible dermatomal distribution, weakness and/or deep tendon reflex changes attributable to a nerve root), intactness of their fusion based on follow-up radiographic studies or subsequent fusion mass exploration surgery, identifiable sources of persistent pain after their fusion procedure, smoking status, and whether disability or litigation was involved regarding a back injury.

Of the 90 fusion patients who underwent preoperative MRI, 88 were personally interviewed to assess their clinical result. This was performed in a standard fashion over the telephone by an independent reviewer (G.R.B.) at a mean follow-up period of 3.9 years. There were 45 men, aged 18–70 years (average 40.3 years) and 45 women, aged 20–79 years (average 40.5 years). Forty-eight percent were involved in work injury compensation or other disability litigation, and 37% were smokers. Patients were categorized as having a significant postoperative reduction in pain if they stated that they were pain free or used only occasional nonsteroidal anti-inflammatory medication.

Each MR image, of which 89 were available for review, was read by one of the authors (K.B.H.), who was unaware of the surgical outcome for the patient. In particular, each level of the spine was analyzed for the relative hydration or fat signal within the ver-

tebral bodies and adjacent to the endplates, based upon T1- (or proton spin density) and T2-weighted images. The results were classified as inflammatory with endplate fissures (decreased signal on T1-weighted images and increased signal on T2-weighted images, Modic type 1) or fatty (increased signal on T1-weighted images and slightly decreased signal on T2-weighted images, Modic type 2)[26]. The patients' clinical results for low back pain were then correlated with the preoperative MRI findings of each patient. Univariate and multivariate $\chi^2$ statistical analyses were performed to determine significant correlations.

## Results

Clinical results, as determined by telephone interview, generally revealed decreased symptoms after lumbar fusion. Patients categorized into the "no pain" result group were either entirely pain free or required only sporadic use of nonsteroidal anti-inflammatory medications at follow-up. Overall symptom change revealed a 52% relief in low back pain and a 65% relief in thigh symptoms. For those patients who underwent a decompression, 78% experienced a resolution of lower leg symptoms, and 48% an improvement in radicular signs. Twelve patients (13%) stated that their pain was worse or no better than before their fusion. For the patients involved with work compensation/litigation the improvement was less, with 33% reporting relief in low back pain, 55% relief in thigh symptoms, 72% resolution of lower leg symptoms, and 47% improvement in radicular signs.

Many of the patients who still had low back pain at follow-up had possible etiologies for their symptoms identified. These included pseudarthrosis, new injury, fracture, and adjacent segment stenosis or disc degeneration. (Leg pain at follow-up was found in all patients who developed arachnoiditis.) Logistic regression analysis found three factors to be independently related to continued low back pain: pseudarthrosis, work compensation/litigation status, and inflammatory MRI vertebral marrow changes adjacent to the endplate of a degenerative disc. Pseudarthrosis, was diagnosed in 12 patients (13%) by plain radiography, tomography, or CT, and was confirmed in 8 of them at exploration/revision surgery. Among the diagnostic groups, pseudarthroses occurred most frequently in the low slip group, involving six patients. Pseudarthrosis was significantly correlated with continued low back pain ($P < 0.001$). The subgroup of patients who had disability claims or were involved in litigation correlated independently with continued low back pain after fusion; that is, even after exclusion of those patients with reasons for possible failed fusion surgery, the correlation with continued low back pain was significant ($P < 0.001$). The third factor that was independently related to continued low back pain was inflammatory MRI vertebral body changes.

Thirty-four of the 89 patients with available MRI scans (38.2%) were found to have vertebral body changes, either Modic type 1 or 2 (Table 1). Of the patients with MRI changes, 76% recalled a specific injury related to their low back pain, 61% were laborers, 12% were clerical, 18% were homemakers, and 9% were professionally employed. Vertebral body MRI changes affected a total of 87 endplates and were always located adjacent to a dehydrated and narrowed intervertebral disc. They occurred in degenerative conditions, with a few patients also having spondylolysis or low-grade, but not high-grade, spondylolisthesis. In the patients with MRI changes, these changes occurred at one endplate in 36% of patients and at multiple levels in 64%. Endplates adjacent to the L4-5 intervertebral disc were most commonly affected by the changes. Table 1 presents the distribution of the vertebral body MRI changes. The distribution of the endplate changes for the two types were similar.

**Table 1** Distribution of MRI vertebral body changes (34/88 patients, values = number of affected endplates, ( ) = %)

| Location | # Type 1 (24 patients) | # Type 2 (24 patients) | Total: Type 1 and/or 2 (34 patients) |
|---|---|---|---|
| Inferior L3 | 0 (0) | 1 (2) | 1 (1) |
| Superior L4 | 4 (9) | 1 (2) | 5 (6) |
| Inferior L4 | 11 (25) | 13 (30) | 24 (28) |
| Superior L5 | 15 (34) | 17 (40) | 32 (38) |
| Inferior L5 | 10 (23) | 9 (21) | 19 (22) |
| Superior S1 | 4 (9) | 2 (5) | 6 (7) |
| Total endplates | 44 (100) | 43 (100) | 87 (100) |

Twenty-four patients had Modic type 1, inflammatory, vertebral body changes at a mean age of 42 years (range 27–70 years). Eighteen of these had undergone a prior procedure: 17 a discectomy and 1 a chymopapain nucleolysis. Thus, the most common diagnostic group with endplate changes was the postdiscectomy group, 75% of whom had endplate changes. Type 1, inflammatory, changes were also identified in three patients in the degenerative disc disease group and three in the low-grade spondylolisthesis group. Most patients (84%) with type 1, inflammatory, vertebral body changes reported an injury related to their low back pain, with most injuries being work related.

Another 24 patients, most of whom had also undergone a prior discectomy, had type 2, fatty, vertebral body changes. They included 13 patients with evidence of both type 1 and type 2 changes (that is 13 patients had multiple endplates involved with some endplates having type 2 changes and other endplates having type 1 changes). The mean age of patients with type 2 changes was 40 years (range 27–74 years). Most of the patients with pain and type 2 changes also had type 1 changes present, that is, they had a mixed pattern that typically had active inflammatory changes on the periphery of the fatty changes.

Some patients had preoperative discography performed at the disc level correlating with the MRI changes. Provocative discography was performed at five discs with corresponding type 1, inflammatory, vertebral body changes,

and at five discs with corresponding type 2, fatty, vertebral body changes. In all cases discography produced concordant pain.

Out of the 24 fusion patients with type 1, inflammatory, MRI changes, 19 (79%) continued to suffer pain. The association of inflammatory vertebral body changes with continued back pain for all 24 patients was significant at $P = 0.03$ (Pearson's $\chi^2$). Eliminating other possible identifiable causes of continued low back pain (pseudarthrosis and arachnoiditis), the relationship of continued low back pain to type 1, inflammatory, vertebral body changes remained significant at $P = 0.03$ (Pearson's $\chi^2$). Of those patients with type 1 changes, 37% stated that they were worse or no better than before their fusion surgery. Type 2, fatty changes, were not significantly associated with continued low back pain ($P = 0.30$). A statistically significant relationship between smoking or compensation/litigation and vertebral body changes was not found.

## Discussion

In this study, we found that a correlation existed between certain vertebral body MRI changes and continued back pain after a technically successful posterolateral fusion. Specifically, persistent low back pain was significantly related to inflammatory, Modic type 1, changes. The study covered a mixed adult population of degenerative of spine patients. This included both patients with degenerative disc disease and patients with spondylolisthesis, with the common factor that their low back pain was thought to be of discogenic origin. Overall, patients generally improved with respect to pain, with 90% reporting improvement in their symptoms and 52% having an excellent result and requiring only sporadic nonsteroidal anti-inflammatory medication. As expected, results for patients who developed a pseudarthrosis or were involved with compensation/disability/litigation were not as successful, and this finding is consistent with the experience of others [5, 12, 21, 31, 36]. In correlating patient diagnosis, continued pain, and preoperative MRI scans, we identified another factor that was independently related to a less successful clinical result. Patients with disc degeneration and type 1, inflammatory, MRI changes of the adjacent vertebral marrow were more commonly found to have continued low back pain after their fusion procedure. This seemed to be related to disc degeneration and not to instability/spondylolisthesis as has been suggested by others [33]. Of the patients with disc degeneration and no spondylolisthesis, 19 had type 1 MRI changes, of which 15 (79%) had continued pain.

In the evaluation of persistent back pain after a lumbar fusion, the surgeon should consider pseudarthrosis and adjacent segment degeneration. Other identifiable causes of pain after a fusion included arachnoiditis, fracture due to subsequent trauma, sacroilitis, prominent hardware, infection, and, rarely pathological lesions [5]. In the current study of 90 patients, possible causes of continued low back pain at follow-up were identified in many. All 12 patients with a pseudarthrosis suffered continued pain. Four of five patients with only a unilateral fusion suffered pain; one can question whether some of these patients, none of whom had their fusion explored, had a pseudarthrosis. The notion that residual motion or strain of the intervertebral disc may be a source of continued pain after a posterolateral fusion has not been proven, nor is the mechanism by which pain is then produced fully understood. The hypothesis that pain originates from the disc has strong support and is generally accepted in the orthopedic community. However, discogenic pain due to residual motion is not well understood and one cannot exclude other possible sources, such as incompetent endplates with associated marrow inflammation, in the etiology of mechanical back pain.

In the present study, most patients with inflammatory vertebral body changes on MRI had continued low back pain without another identifiable source of pain. This finding suggests that MRI vertebral body changes may have clinical relevance. MRI vertebral body changes adjacent to degenerated discs have been found to occur in 20% of one study population of consecutive patients having lumbar spine MRI [26], and in 50% of another study population in which all the patients had a diagnosis of degenerative disc disease [7]. In both of these studies, type 2, fatty, changes were more common. Recently, a study found that inflammatory changes were clinically significant such that 73% of patients had severe low back pain [24]. The current study of surgical patients revealed that for the entire group, 38.2% had vertebral body changes on preoperative MRI, with an equal distribution between type 1 and 2. Type 1 vertebral body changes were identified in 24 of the 89 MRI scans (27%) and 19 (79%) of these 24 patients had continued low back pain. However, it must be noted that of these 19 patients, 3 developed a pseudarthrosis and 1, with continued low back and leg pain, developed both pseudarthrosis and arachnoiditis. Further support for the hypothesis that abnormal endplates are related to pain comes from a study of postoperative MRI scans. In this study, it was noted that type 1, inflammatory, vertebral body changes were related to continued low back pain [19]. Three patients in the present study with type 1 changes, all of whom had continued low back pain, had hardware removal at a subsequent procedure and underwent postoperative MRI examinations. This revealed persistent type 1 changes at 1 year follow-up in one patient. A second patient was found to have persistent type 1 changes, but with partial replacement by type 2 changes at 3 years, resolving to normal marrow signal at 5 years after surgery. In the third patient, decreased type 1 changes were noted at the original site, but new type 1 changes at another level were identified at 2 years, followed by the resolution of all changes at 4 years after

surgery. It seems that MRI vertebral body inflammatory and fatty changes are slow dynamic phenomena that may resolve over a period of years. The natural history of these changes is only poorly understood and requires longitudinal studies of patients with serial MRI and clinical correlation of low back pain presence and resolution.

The etiology of the MRI vertebral body changes is not well understood; however, the biochemical changes are probably related to biomechanical changes of the endplates. Similar changes about the knee in patients with a history of trauma [34] have been termed "bone bruises". In a previous study, biopsy of the vertebral bodies adjacent to the endplates in six patients, three with type 1 and three with type 2 changes, revealed that type 1 changes correlated histologically to inflammatory changes and all were also found to have endplate fissuring [26]. These findings were subsequently confirmed by another study with histological examination in ten patients who underwent anterior fusion for severe pain [33]. One can speculate that the MRI vertebral body type 1 inflammatory changes may be due to vertebral endplate discontinuities, which may then allow inflammatory mediators from the disc [15, 16, 23, 25, 28, 32] to act upon the adjacent vertebral body.

The hypothesis that low back pain can originate from the inflammation associated with abnormal endplates of degenerative discs has support from previous studies. Anatomical studies in cadavers have shown that degenerated discs were associated with microfractures of the endplates and the endplate changes always preceded histological changes in the nucleus and anulus of the disc [30]. Biomechanical studies have demonstrated endplate deformation during discography [11]. Abnormalities of the endplates and adjacent marrow may stimulate elements of the sympathetic nervous system [1, 2, 14]. Clinically, direct endplate manipulation and discographic loading of disrupted endplates has been associated with concordant pain symptoms [6, 13, 18, 29]. In the present study, all patients with MRI vertebral body changes and evidence of endplate disruptions who also had discography had concordant pain at the corresponding level. However, there was insufficient information to differentiate between the degree of discography pain severity in patients with type 1 and those with type 2 MRI changes, and one could not exclude the anulus as the source of pain. Endplate discontinuities have been found in the cadavers of heavy laborers [35], which fits with the data showing that most of the patients in this study with vertebral body inflammatory changes were laborers. Moreover, most of these patients were able to describe a specific back injury. This suggests that laborers may suffer injuries that result in endplate injury.

Although the present study suggests a relationship between endplate disruption and low back pain, it does not indicate causality. Additional study at a basic science level is needed to determine how pain may be produced by abnormal endplates: particularly, the relationship of histological vertebral marrow inflammation, endplate disruptions, and biochemical analysis of inflammatory mediators, proteolytic enzymes, and noxious neurotransmitters. Recently, animal studies on baboons found that 12 of 21 animals had MRI vertebral body changes at the endplates associated with disc degeneration [20]. Such an animal model may help in the further understanding of disc degeneration, endplate disruptions, and adjacent vertebral body inflammatory changes. Biomechanical studies of in vivo disc and endplate loading before and after a fusion at the corresponding intervertebral level would contribute to an understanding of what degree fusion alters motion and disc/endplate loading. Our preliminary studies on in vitro spine motion segments have found that a simulated posterolateral fusion did not significantly decrease endplate stresses [8]. If an in vivo posterolateral fusion greatly reduces intervertebral motion, but inconsistently reduces axial disc/endplate loading, then a significant reduction in pain may not be altogether unexpected for the case of an abnormal endplate.

If further research indicates that abnormal endplates and associated inflammatory marrow changes do indeed cause pain, then should one not direct treatment at this source of pain? Whether to fuse posteriorly, anteriorly or both may be a difficult decision. Interbody fusion may directly treat offending painful endplates, and we are implementing a prospective study to determine the effectiveness of interbody fusion for this group of patients. If the results of this study are confirmed by others, then MRI, a noninvasive diagnostic technique, may greatly assist in the process of deciding when to perform interbody fusion, by identifying whether or not endplate pathology exists.

## Conclusions

1. MRI vertebral body changes, fatty and/or inflammatory, may occur in more than one-third of patients with highly symptomatic lumbar degenerative disc disease.

2. When present, inflammatory vertebral body changes, Modic type 1, are related to a less predictable result after a posterolateral lumbar fusion.

3. The question of whether inflammatory vertebral body changes adjacent to a degenerative disc are related to an abnormal and painful endplate requires further study. If they are, then MRI would be a useful noninvasive study in the diagnosis of symptomatic endplates.

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