

MRI of the Lumbar Spine: A Sports Medicine Approach

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INTRODUCTION

MRI of the spine has shown significant advancement over the last 5 years. This is in part explained by improvements in technology, including improved coil technology, improved imaging sequences leading to more rapid scanning and increased resolution of spinal anatomy. The more significant advance, however, has been in our increasing ability to be more sensitive and specific in diagnosing spine pathology. Just as treatment of spinal injuries and degeneration has evolved with a basis in conservative sports medicine therapy, spinal imaging is taking the same course. First and foremost, there must be an understanding of the basic anatomy, including intradiscal anatomy. The imager must have an understanding of the mechanisms of spinal injury and the subsequent degenerative changes that can be seen by MRI. Recognition of the body's inflammatory and reparative response demonstrated on MRI images should lead to specificity of diagnosis.

DISC DEGENERATION AND HERNIATION

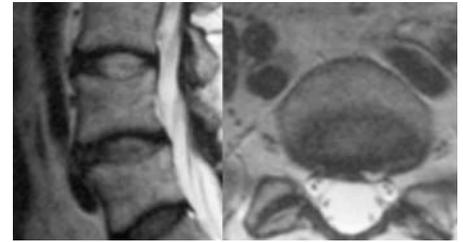
When using MRI to study the degeneration of the spine, it is useful to think in term of Kirkaldy-Willis' model of the degenerative cascade. In that model, the earliest stage of the degenerative process involves tears of the annulus fibrosus, beginning first with small concentric outer annular tears and progressing to radial tears. All annular tears that communicate with the nucleus can be demonstrated by discography, and all discs that show hypointensity on MRI ("black discs") will be shown to have annular tears. A subset of annular tears has been described that are hyper intense on heavily T2 weighted MRI sequences. This subset of annular tears has been called a high intensity zone (HIZ) [4,6,7]. This finding on MRI has been shown to have an 86% positive predictive value for predicting concordant pain at discography, although the finding is still somewhat controversial [1,4,6,7,8,9, 27,41] In a recent study, Weishaupt et al. did not find the HIZ to be valuable when comparing MRI to discography [42]. They were able to show only a 56% PPV and a sensitivity of 27%. They concluded that that the HIZ could not be used to identify a painful disc. The anatomic and biochemical basis for this

finding as a marker for pain rests in the fact that with outer annular tears as the disc attempts to repair itself neovascularization extends into the tear, dragging nociceptive pain fibers along. There are two pieces of evidence that the bright signal represents neovascularization and not just fluid which would also be bright on a T2 sequence. First, most HIZs enhance following the intravenous administration of gadolinium, which requires the presence of vascularization. Second, an HIZ has been biopsied, and granulation tissue was observed [19,21]. While originally HIZs were described only in the midline of the posterior annulus, and not differentiated as to type, experience has taught us that they may occur anywhere in the outer annulus, and also may be subdivided into three types [2,3,12]. A radial tear extends linearly from the nucleus to the outer annulus; a transverse tear is a focal tear at the insertion of the annulus to the adjacent vertebral margin (most frequently inferiorly), and a concentric tear, which extends in a curvilinear fashion transversely along the outer annular fibers.

In the original work by Yu, et al., the transverse tear was felt to be most associated with pain on discography, and we are currently evaluating whether this distinction holds in comparing MRI to provocative pressure monitored discography.

While most HIZs enhance following intravenous gadolinium, this does not help one differentiate between painful and non-painful HIZs [10]. While some observers feel that the HIZ is a marker for internal disc disruption and "discogenic" pain, the jury is still out on this question.

With the coalescence of concentric annular fissures a portal is created for the extension of nuclear material beyond its normal confines beneath the inner annulus. Any focal abnormal annular contour measuring

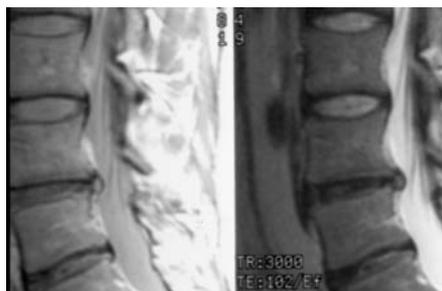


Small transverse HIZ

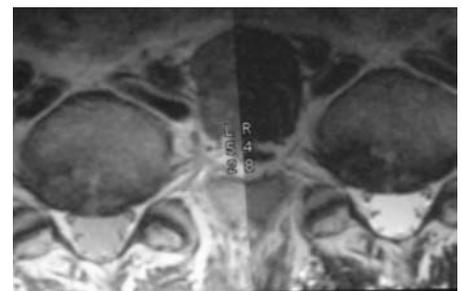
at least 3 millimeters will be shown at autopsy to have nuclear material underneath it. One of the great problems in the reporting of spinal MRIs has been the the lack of standardized terminology. Such terms as "discal bulge", "prolapse", and "bulging disc" should be discarded to be replaced by specific terms to describe specific pathoanatomy that can now be demonstrated with high resolution MRI. The term "annular bulge" should be used when as a result of disc dessiccation and loss of height the annulus concentrically extends beyond the vertebral margin. The term **protrusion** should be reserved for a focal or broad based smoothly marginated extension of the annulus beyond the vertebral margin where one of the following findings is clearly evident: the outer annulus/posterior longitudinal ligament complex is intact; or the base of the abnormal annular morphology is broader than the apex.

An **extrusion** on the other hand, should have the opposite findings: the outer annulus/posterior longitudinal ligament complex is clearly disrupted, and/or the base is narrower than the apex. It should be noted that because the PLL is narrower between the discs and broadens out at the disc, that an extruded disc fragment may migrate cranially or caudally and remain in front of the PLL.

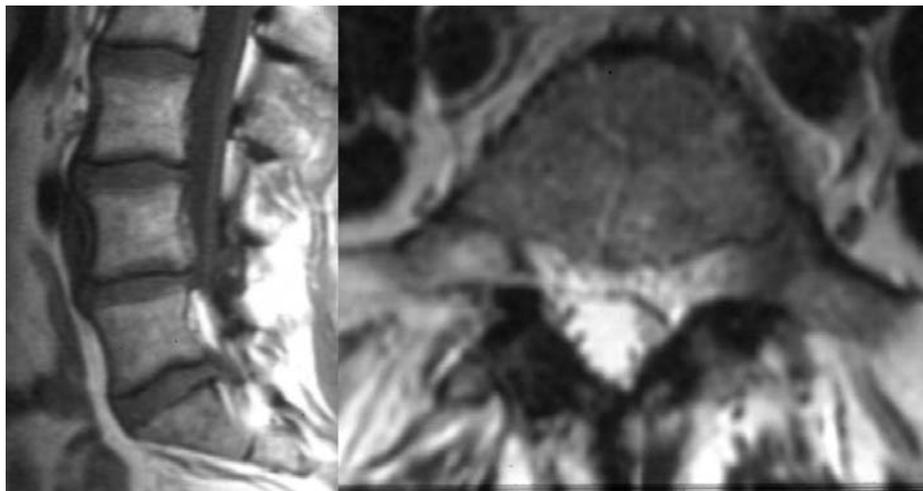
Finally, a **sequestration** refers to a disc fragment that has broken off from the parent nucleus and is free in the epidural space.



Protrusion (sagittal)



Protrusion (axial)



Extrusion (sag)

Swollen L5 nerve root

Such a fragment is often surrounded by granulation tissue or hematoma, either or both of which may contribute significantly to the epidural mass. The term **herniation** should only be used when the more precise terminology is unclear, such as when interpreting a CT scan, myelogram, or low resolution MRI. The term "herniated nucleus pulposus" is never appropriate, since a herniation may contain a number of tissues, including the nucleus, the cartilaginous endplate, the annulus, and even a rim of

vertebral cortex.

The differentiation of different anatomic types of herniation is more than an academic exercise. A number of studies have shown that people without back pain or radiculopathy may have abnormal MRIs [20,31]. However, there is also evidence that extrusions are in fact specific and not found in asymptomatic populations [11,22,36, 37,39]. There is also evidence of interobserver and intraobserver reliability in making the differentiation by MRI [20,38,40].



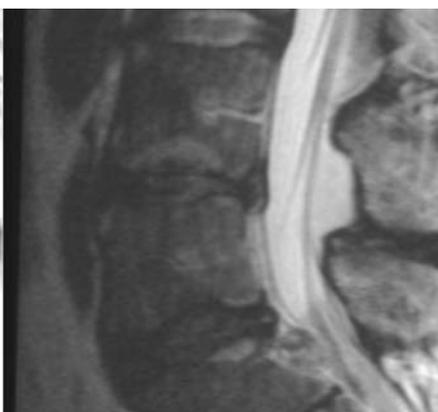
Acute Post "Limbus" Herniation

SECONDARY SIGNS OF INFLAMMATION FROM DISC EXTRUSIONS

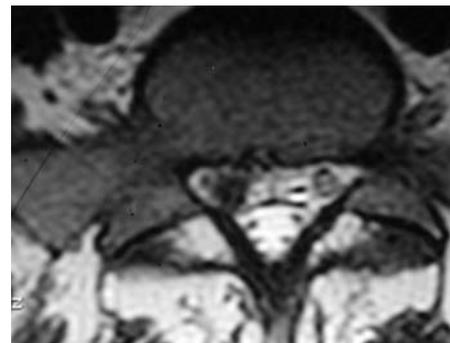
A disc fragment extruded into the epidural space or foramen is treated by the body as a "foreign substance", and the response is that of secondary inflammation. Many investigators believe it is this inflammatory reaction that is the cause of pain, rather than the mass effect. MRI may visualize the inflammatory response. The epidural fat is often indurated and therefore shows increased signal on MRI [21]. A cap of



Extrusion + granulation (T1)



Extrusion + granulation (T2)



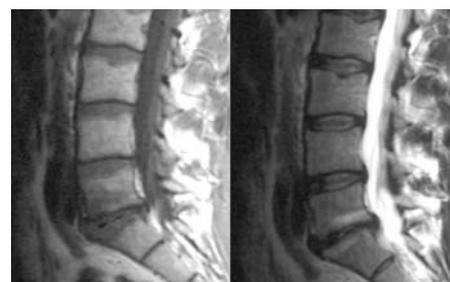
Desiccated Fragment

granulation tissue may develop and contribute to the mass effect [17,24,26,39]. This surrounding mass will enhance after the administration of intravenous gadolinium. It is also brighter on T2 weighted images, a finding that may have prognostic significance since some investigators have shown that disc extrusions that are bright will more likely resolve than those that are "black" [17]. The bright disc extrusions contain more granulation tissue, and the dark extrusions are desiccated fragments, unlikely to be resorbed. It should also be noted that many of the "bright" disc extrusions may in fact be spontaneous epidural hematomas adjacent to a small protrusion [16]. These masses will almost certainly disappear within four to six weeks.

It is also possible, using high resolution MRI, to demonstrate either pre or post compressive swelling of an involved nerve root. This can be seen both in and out of the thecal sac, and can be used as further specific evidence for directed single nerve root block or localized epidural injection. While some of these nerve roots may enhance after the injection of intravenous gadolinium, this has not been shown to be of additional help in localizing the symptomatic nerve root [29].

OSSEOUS INFLAMMATORY CHANGES ADJACENT TO DISC DEGENERATION

Bone marrow adjacent to a degenerating or degenerated disc may show abnormal signal intensity. Instead of the preponderant



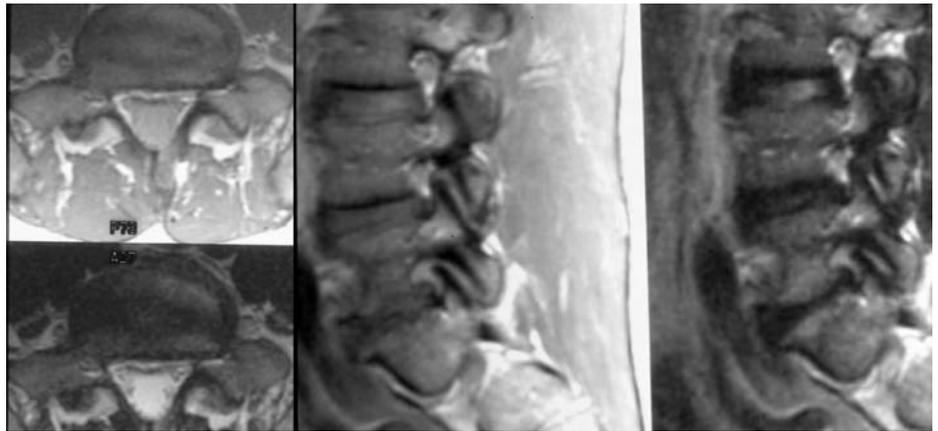
Modic Type 1 (T1) Modic Type 1 (T2)

fat signal seen in adults, the marrow may be bright on a T2 weighted sequence and decreased in signal on a T1 weighted sequence. Modic first described this MRI finding, and it is called Modic Type 1 change. The finding is due to granulation tissue (not just edema), and therefore much as the granulation tissue around a disc extrusion will enhance after intravenous contrast.

If the signal is dark on T2 and bright on T1 (typical of complete fatty replacement), then this is termed Modic Type 2. There is evidence that Type 1 change is specific for discal pain and perhaps instability [15,18,28,30]. A recent study indicated that moderate end plate changes of both Type 1 and Type 2 may show a high correlation with discogenic pain [42]. Similar inflammatory marrow changes may be seen adjacent to Schmorl's nodes [26,32,33,34]. It may be that there is a progression from Type 1 to Type 2 as the spine restabilizes, or in the case of spinal fusions, the fusion becomes solid. It is also possible for Type 2 to revert to Type 1 with interval destabilization.

SPINAL STENOSIS

Lumbar spinal central stenosis may be divided into developmental and acquired. Almost all central stenosis is due to developmental stenosis with short pedicles and relatively large facets; or degenerative spondylolisthesis. Developmental central stenosis alone is unlikely symptomatic, but lessens the "reserve" necessary to protect against central stenosis when the disc and posterior elements degenerate. As the facet joints degenerate there is a decrease in the interfacet angle and often flattening of the posterior thecal sac from the noncompliant posterior epidural fat pad. Absolute measurements of the diameter or cross sectional area of the central canal are of less importance than the observation of the effect on the thecal sac and nerve roots. We



Up-Down Foraminal Stenosis with swollen L5 nerve root

categorize stenosis by quarters: <25% cross sectional loss – mild; 25-50% – moderate; 50-75% – moderately severe; and >75% severe.

The anatomic area from the exit of the nerve root from the thecal sac until the entrance zone of the foramen is termed the lateral recess. In reality, lateral recess stenosis is almost always a result of facet hypertrophy, and the area narrowed is therefore the "subarticular gutter" which is the most cranial portion of the lateral recess.

Foraminal or lateral stenosis is almost always a result of loss of disc height and posterolateral osseous ridging secondary to annular bulging. These findings narrow the foramen in an "up-down" or "cranio-caudal" fashion, entrapping the exiting nerve root between the posterolateral ridge and the undersurface of the pedicle. Less commonly isolated overgrowth of enlarged facet joints and facet capsular hypertrophy results in isolated "front-back" or "ventro-dorsal" foraminal stenosis. A combination of both types of lateral stenosis would be termed concentric foraminal narrowing. As with disc extrusions, pre or post compressive nerve root swelling may be seen with central and foraminal stenosis.

SUMMARY

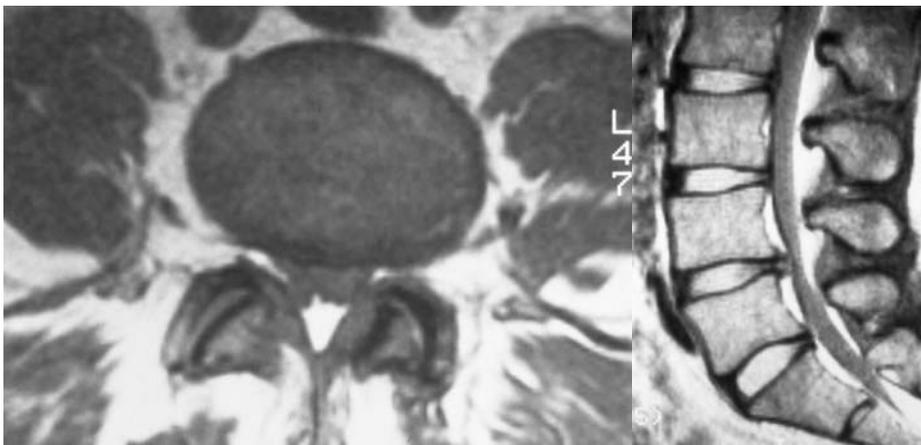
The knowledge of normal and intradiscal anatomy demonstrated by MRI has grown exponentially over the last few years. While the significance of some findings is clear cut, especially in the evaluation of radiculopathy, in the study of discogenic pain, controversies still exist. As with all imaging studies, the comparison of MRI findings with the clinical evaluation is always essential.

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Imaging of the Spine

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Developmental Central Stenosis

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