Degenerative Disk Disease I

Techniques for the MRI evaluation of the lumbar spine differ from those implemented in the cervical and thoracic regions. For axial imaging in the cervical region, GRE T2W techniques are obtained due to potential problems from CSF pulsation and the small size of cervical spinal structures warranting thin slice imaging (2–3 mm). In distinction, slice thicknesses of 3 to 4 mm are acceptable in imaging of the lumbar spine where less prominent pulsation artifact also favors the acquisition of FSE T2WI. A thick coronal saturation slab is also routinely placed over the prevertebral tissues to eliminate artifacts from the aorta and vena cava, as well as abdominal motion. FSE has other advantages over GRE, including—due to its additional 180-degree refocusing pulse—diminished artifacts arising from differences in tissue susceptibility. Such artifacts play a role clinically, not only in postoperative patients wherein ferromagnetic implants may limit the diagnostic utility of GRE sequences, but also with respect to evaluation of spinal canal and neuroforaminal narrowing. With the latter, susceptibility effects from bone may exaggerate canal or foraminal narrowing depending upon the selection of imaging parameters. Tissue contrast with FSE T1WI of the lumbar spine is derived from differences in SI between high SI epidural fat versus the low SI thecal sac contents and intervertebral disk. This contrast is lost somewhat on FSE T2WI due to the preservation of hyperintense fat signal on such sequences.

The MRI appearance of the intervertebral disk changes with age. The intervertebral disk of a neonate is of moderate and high SI on T1WI and T2WI, respectively, the latter due to cartilaginous ground substance. The rim of the disk is of low SI as cartilage in this region is denser (i.e., containing less ground substance). With time, such cartilage comes to comprise the outer annulus and equator, resulting in a similar low SI. The collagen of the inner annulus remains of high SI on T2WI, isointense to the nucleus pulposus. Such SI is related to the presence of mucopolysaccharides with strong, fixed negative charges that attract free water into the disk. With normal aging, various factors may contribute to loss of such molecules and thus of intradiscal free water. The annulus may, for example, tear as illustrated in Figs. 46.1A,B. Here, sagittal (A) FSE T2WI demonstrates a disk bulge with a radial annular tear that has allowed high SI fluid and mucoid material (white arrow) to fill the resulting gap. (B) Axial T2WI illustrate mild resulting narrowing of the central spinal canal from the disk bulge, along with the aforementioned high SI (white arrow) material along the posterior disk margin. This hyperintense region typically enhances on contrast-enhanced T1WI. Concentric tears run parallel to the vertically oriented collagenous fibers of the disk, while transverse tears, resulting from the disruption of the attachment (i.e., Sharpey fibers) of the annulus to the cartilaginous vertebral body end plate, run perpendicular to the peripheral collagenous fibers. Nociceptive neural receptors are present within the outer annulus, and thus annular tears, even if an isolated finding, constitute a potential cause of back pain. Although some loss of disk SI occurs normally with age, desiccation is the hallmark of degenerative disk disease. Disk desiccation occurs due to proteoglycan loss within the nucleus pulposus resulting in a diminished ability of that structure to attract water.

Fig. 46.1 (A,B)
A relatively normal L2–L3 disk is illustrated in Figs. 46.2A,B: (A) sagittal T1WI demonstrate preservation of intervertebral disk height, whereas (B) FSE T2WI illustrate a relatively normal hyperintense nucleus pulposus surrounded by the low SI annulus. Note as well, the normal low signal intensity intranuclear cleft. In distinction, the L3–L4 intervertebral disk demonstrates marked loss of height and SI, the latter evident by the uniformly low SI on (B) FSE T2WI. Severely degenerated disks may contain foci of gas, contents suggested in this case by the linear hypointensity within the disk on (A) T1WI. The lack of mobile protons within these likely nitrogen-containing gas pockets results in low SI on both T1WI and T2WI. Figures 46.2A and 46.2B also illustrate mild end plate degenerative changes at L3–L4 (see Chapter 47) as well as a grade 3 anterolisthesis of L5 on S1. Grading of anterolisthesis is based upon the degree of anterior displacement of the superior vertebral body. Grade 1 lesions consist of displacement less than 25% of vertebral body length, grade 2 lesions of displacement between 25 and 50%, and grade 3 lesions of displacement greater than 50%. The anterolisthesis in Figs. 46.2AB was associated with bilateral pars interarticularis defects—a finding better visualized on CT and present bilaterally in nearly all patients with grade 2 or 3 listhesis. Pars defects are likely related to prior trauma, although a congenital etiology has also been suggested in the past. In the absence of pars defects, spondylolisthesis is typically accompanied by marked bilateral facet arthropathy. The L5–S1 intervertebral disk in Figs. 46.2AB rests superior to the posterior portion of S1 despite the anteriorly displaced L5 vertebrae. This portion of the disk does not extend significantly beyond the posterior border of S1 but appears to result in AP narrowing of the central spinal canal—a phenomenon known as a pseudo bulge. An anterolisthesis will also lead to elongated neural foramina in the AP dimension, narrowing them in the craniocaudal dimension. Although axial imaging is typically obtained with slices parallel to the intervertebral disk—to facilitate evaluation of disk protrusions and associated canal stenoses—nonangled imaging planes (with acquisition of a continuous slice block) may aid in visualization of pars interarticularis defects.