47. Degenerative Disk Disease II

Spondylolisthesis, described in the last section, is but one cause of central canal stenosis. The sagittal FSE T2WI of Figure 47.1 A illustrates a normally hydrated intervertebral disk at L5-S1, with desiccated disks at all other levels. Despite this, no significant disk bulges or protrusions are present, although the canal is, nevertheless, of small (< 11.5 mm) AP diameter. Congenitally shortened pedicles—the etiology of this finding—is evident in the axial T2WI of Figure 47.1 C. A congenitally narrowed canal will amplify the severity of any degenerative findings that might develop. The canal in Figure 47.1 B, D is also narrowed. Here, a (B) FSE T2WI demonstrates some loss of disk SI at all visualized levels except S1-2. Mild loss of vertebral body height at L5-S1 suggests loss of discal SI secondary to degenerative changes rather than normal aging. A prominent disk bulge is also present at L4-5, which together with ligamentum flavum hypertrophy at this level results in
moderate to severe central canal stenosis—a finding best appreciated on the axial T2WI of Figure 47.1 D. Other structures surrounding the spinal canal can similarly lead to canal stenosis. Degenerative changes of the spine are present in Figure 47.2 including either disk bulges or disk-osteoophyte complexes at all visualized lumbar levels as well as disk space height loss at L3-4 and L4-5. Modic type 2 degenerative changes (with the signal intensity of fat) are present at L4-5. Proliferation of the epidural fat is, however, in this case the most salient contributor to spinal canal stenosis, particularly at the L5-S1 level. Epidural lipomatosis occurs most frequently in the lower thoracic and lumbar spine and is often associated with exogenous steroid administration. A subacute epidural hematoma may exhibit similar SI but is not suppressed on STIR or FS imaging.

Fig. 47.2

Degenerative changes of the intervertebral disk do not occur in isolation and often result in discogenic sclerosis of the adjacent vertebral body endplates. Endplate degeneration involves the L5-S1 vertebral body endplates in Figure 47.1 B. Here, FSE T2WI demonstrate abnormal endplate hyperintensity. Such changes are consistent with either Modic type 1 or type 2 degenerative disease. Type 1 changes consist of edema-like SI (with low SI on T1WI) involving the endplates. Type 2 changes are the most common, being felt to represent a temporal progression from Type 1 changes and corresponding to fatty infiltration, thus exhibiting high SI on both T1 and FSE T2WI. Type 3 changes (which are uncommon) are associated with development of sclerotic bone, manifesting as low SI on both T1 and T2WI. Distinction amongst the different types of endplate disease is significant as Modic type 1 changes appear similar to findings in infectious diskitis. Endplate enhancement is seen with both entities, further complicating the diagnosis. Disk SI tends to be low in degenerative disease, secondary to desiccation, while the disk in infection is marked by high SI fluid/edema. In addition, normal or degenerated intervertebral disks do not enhance or enhance only peripherally, while discal enhancement in diskitis may be diffuse.
Strain induced by Sharpey’s fibers upon the vertebral disk endplate may result in development of osteophytes that can narrow the central canal or alternatively the neural foramina, the latter illustrated in Figure 47.3. In the lumbar spine, high SI epidural fat outlining the low SI nerve roots allows for accurate evaluation of neuroforaminal stenosis on sagittal T1WI. This appearance is illustrated in Figure 47.3 A at the level superior to the white arrow. The caliber of the neural foramen at this level is not, however, normal as the normal foramina should have the appearance of a key-hole. In this case, as is typical (for early degenerative disease), the inferior portion of the foramina, consisting of veins and fat, is narrowed while the superior portion which contains neural tissue is spared. At the subjacent level, a disk osteophyte complex—with low signal intensity—is present, compromising the foramen and obliterating the fat surrounding the nerve root (white arrow). (B) Axial images confirm severe narrowing of the neural foramina together with mild facet osteoarthritis. The facet joint is the articulation between the inferior and superior articular processes of vertically adjacent vertebrae and is normally lubricated by synovial fluid, although the increased fluid (with high SI) seen in Figure 47.3 B is abnormal and has been shown to correlate with joint instability. As shown, the ligamentum flavum (with low SI) comprises the joint’s anterior/medial border. Degenerative changes of the facets consist of joint space narrowing, cartilaginous obliteration, as well as osseous erosions and osteophytosis. In contrast to facet arthropathy, facet synovitis is marked by hyperintense signal, best visualized on FS or STIR T2WI. Both sterile and infectious synovitis enhance, rendering their distinction difficult on MR. Arthropathy of the superior facets in particular may narrow the lateral recess—the region bordered by the superior articular facet posteriorly, the pedicle laterally, and the posterior surface of the vertebral body anteriorly—resulting in compression of the nerve root prior to its entrance into the neural foramen.