Degenerative Disk Disease II

Spondylolisthesis is but one cause of central canal stenosis. The sagittal FSE T2WI of Fig. 47.1A 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 Fig. 47.1C. A congenitally narrow canal will amplify the severity of any degenerative findings that might develop. The canal in Figs. 47.1B,D is also narrowed. Here, a (B) FSE T2WI demonstrates loss of disk SI at all visualized levels except S1–S2. 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–L5, 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 Fig. 47.1D. Other structures surrounding the spinal canal can similarly lead to canal stenosis. Degenerative changes of the spine are present in Fig. 47.2 including disk bulges or disk-osteophyte complexes at all visualized lumbar levels as well as disk space height loss at L3–L4 and L4–L5. Modic type 2 degenerative changes (with the signal intensity of fat) are present at L4–L5. 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.

Degenerative changes of the intervertebral disk do not occur in isolation and often result in discogenic sclerosis of the adjacent vertebral body end plates. End plate degeneration involves the L5–S1 vertebral body end plates in Fig. 47.1B. Here, FSE T2WI demonstrates abnormal end plate hyperintensity. Such changes are consistent with Modic type 1 or type 2 degenerative disease. Type 1 changes consist of edema-like SI (low SI on T1WI) at the end plates, believed to correlate with increased vascularity. Type 2 changes occur chronically and represent fatty infiltration, exhibiting high SI on both T1 and FSE T2WI. Type 3 changes are associated with development of sclerotic bone, manifesting as low SI on both T1 and T2WI. Distinction among the different types of end plate disease is significant as Modic type 1 changes appear similar to findings in infectious diskitis. End plate enhancement is seen with both entities, further complicating the diagnosis. Disk SI tends to be low in degenerative disease, secondary to desiccation; the disk in infection is marked by high SI fluid/edema. In addition, normal or degenerated intervertebral disks do not

![Fig. 47.1 (A–D)](image-url)
Strain induced by Sharpey fibers upon the vertebral disk end plate may result in development of osteophytes that can narrow the central canal or neural foramina, the latter illustrated in Fig. 47.3A,B. 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 Fig. 47.3A 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 keyhole. In this case, as is typical, 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—isointense to the vertebral body—is present, compromising the foramen and obliterating the fat surrounding the nerve root (white arrow). (B) Axial images confirm severe narrowing of the right neural foramina and illustrate as well normal facet joint anatomy. The facet joint is the articulation between the inferior and superior articular processes of vertically adjacent vertebrae and can be normally filled with high SI synovial fluid as in Fig. 47.3B. As shown, the ligamentum flavum comprises the joint’s anteromedial 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 on FS or STIR T2WI. Both sterile and infectious synovitis enhance, rendering their distinction difficult on MRI. Arthropathy of the superior facets in particular may narrow the lateral recess—the region between the anteromedial superior facet and the posterior border of the vertebrae—resulting in compression of the nerve root prior to its entrance into the neural foramen.