38. Canal Compromise

The fact that compression fractures can be the result of metastatic involvement or benign osteoporosis was mentioned in Chapter 35 and is demonstrated in the pre-contrast T1WI of Figure 38.1 A. In this image, the SI of the T9 and T11 vertebral bodies is markedly higher than that of the normal T6, 7, and 12 vertebral bodies. In fact, vertebral bodies T8-11 were irradiated to treat the metastatic tumor within T10. Within the first week of irradiation, edematous marrow changes can lead to the appearance of high and low SI on T2 and T1WI, respectively. However just as ionizing radiation destroys metabolically active neoplastic lesions, so too does it kill active hematopoietic marrow cells within bone. Thus, one week following radiation therapy, compensatory hypertrophy of the fatty elements of marrow begins to increase the SI on T1WI, eventually resulting in hyperintensity of these vertebral bodies when compared to normal. Structures irradiated with low doses of radiation may eventually return to a normal SI appearance. Unfortunately, radiation therapy in this case was not successful, and recurrence of metastatic disease within the T10 vertebral is seen. Related metastatic collapse has resulted in anterior compression of the spinal canal. As with any suspected compression, axial images should be examined to more accurately assess canal compromise. Finally, loss of anterior T8 vertebral body height suggests an additional compression fracture. With chronic fractures, the preservation of marrow signal intensity permits the diagnosis of an osteoporotic fracture, however acutely fractures due to osteoporosis and malignancy are difficult to distinguish on MR since edema-like SI changes are present in both. In this instance, a band of low SI edema is seen within T8 on

Fig. 38.1
Figure 38.1 A—a fracture favored to be osteoporotic due to the lack of a discrete metastatic foci. In Figure 38.1 B, involvement by metastatic disease has led to the expansion of the posterior elements of T7 with resultant compression of the cord. For reasons described in Chapter 35, the non-FS FSE T2WI shown fails to demonstrate the metastatic foci that were present on this slice in T3, 4, and 7 on the FSE T1 and FS T2WI (images not presented). Not all lesions compressing the cord, of course, are metastatic (i.e. primary bone tumors like osteochondromas) or even neoplastic (i.e. epidural abscesses or osteoporotic compression fractures). An arachnoid cyst, as demonstrated in Figure 38.1 C, is another potential culprit. The SI of these lesions is characteristically that of CSF. As such they may only be detectable by noting spinal cord compression or nerve root displacement. Subtle distinction of the arachnoid cyst from normal CSF SI may be made due to differences in fluid mobility inside (asterisk) and outside of the cyst. As with any lesion impinging the cord, the degree of compression is best evaluated on axial images. The T2WI in Figure 38.1 D demonstrates marked compression of the normal low SI cord (displaced anteriorly) by the hyperintense arachnoid cyst.

In multiple myeloma, involved vertebral bodies may appear normal on MR, or diffusely or focally infiltrated (with variable canal compromise). The latter is the case in Figure 38.2 A, in which small low SI lesions are present, which are somewhat subtle on FSE T1WI. With
the administration of contrast and saturation of fat SI, however (Fig. 38.2 B), these lesions become much more obvious due to their enhancement. In distinction, osteoblastic metastases, often found in prostate and breast cancer, appear as extremely low SI on T1 and, as opposed to their lytic counterparts, also on T2WI. Figure 38.2 C demonstrates a very low SI blastic metastasis (asterisk) amongst other, less hypointense lytic lesions on a sagittal T1WI.