

## 50. Disorders of Marrow

As discussed in the previous chapter, the MR appearance of multiple myeloma in the vertebral column is variable, ranging from a normal imaging appearance to one indicating focal, multi-focal, or diffuse involvement. Within the myeloma spectrum also is the diagnosis of plasmacytoma which refers to plasma cell proliferation within only a single focus and likely represents an early stage of multiple myeloma. The diffuse pattern involvement in multiple myeloma is illustrated in the sagittal T1WI of Figure 50.1 A. Note that in this case the homogenous appearance of the vertebral bodies may be misleading—the lack of focal lesions hindering the detection of abnormality. Thus, it is essential when evaluating sagittal T1WI of the spine to compare the SI of the vertebral bodies to that of the intervertebral disks. Normally, fat content within the vertebral bodies will render them hyperintense to the disks on T1WI. In the present case, however, the vertebral bodies and intervertebral disks are essentially isointense, raising concern for diffuse marrow pathology. Differential considerations for this appearance include marrow replacement, as in myeloma or metastatic disease, as well as marrow reconversion to a hematopoietic dominance. Lymphoma is another cause of diffuse marrow replacement and is illustrated in Figure 50.1 B. Note that this appearance is indistinguishable from that of myeloma (Figure 50.1 A) and that of acute lymphocytic leukemia (Figure 50.1 C). Proliferation of other cell line precursors, such as red blood cells in polycythemia rubra vera, may lead to a similar

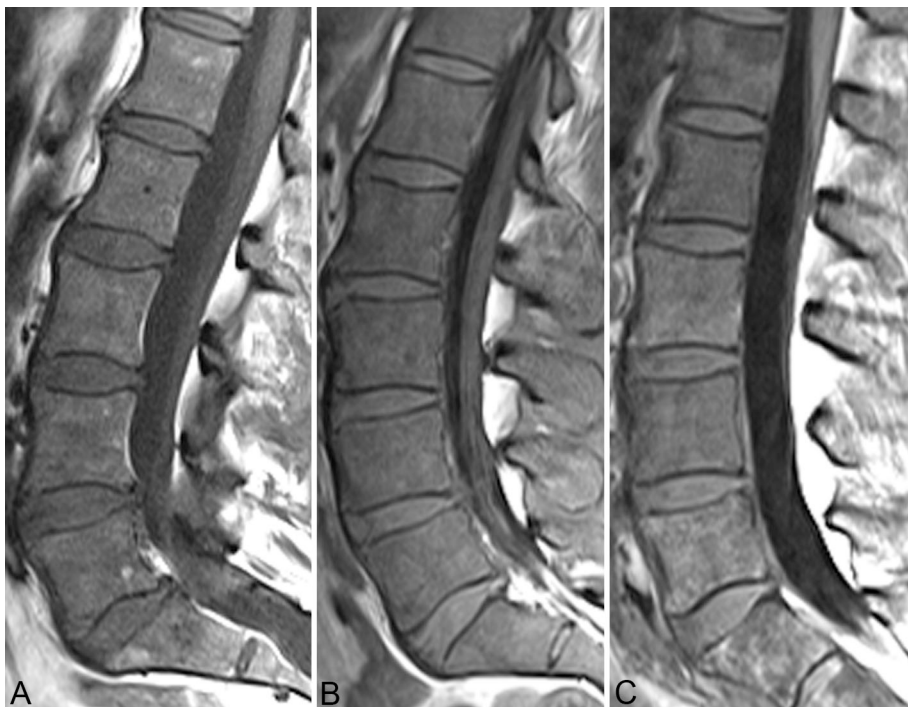


Fig. 50.1

appearance. Analogously treatment with chemotherapeutic agents designed to stimulate cell-line precursors may stimulate growth of hematopoietic elements in marrow, leading to a predominance of hematopoietic marrow and diffuse low SI on T1WI. Examples of such agents include granulocyte and erythrocyte stimulating factors. In the late stages of polycythemia rubra vera, myelofibrosis may occur resulting in fibrous replacement of marrow, yielding a low SI appearance on T1 and T2WI.

In the absence of a supportive history, other causes for diffuse vertebral marrow hypointensity on T1WI must be considered. In particular, interpretation of marrow SI must be made within the context of the patient's age. At birth, the percentage of hematologically active marrow is much greater than that of a normal adult. Thus, diffuse low SI marrow on T1WI is normally seen in children, with the SI increasing with age. As this conversion to fat-predominant marrow progresses, hematopoietic marrow is completely replaced within the appendicular skeleton. Within the vertebral column and axial skeleton, however, the latter persists but in lesser percentage than fatty marrow. Due to this persistence, reconversion of yellow to red marrow is more likely to occur in the axial skeleton. This may at times be a normal finding secondary to chronic hypoxemia in endurance athletes, obese smokers, and in the setting of heart failure. Such reconversion is thought to be mediated by erythropoietin. In distinction to malignant conditions replacing the marrow, like multiple myeloma and metastatic disease, foci of enhancement are not seen with physiologic marrow reconversion, although hematopoietic marrow may normally exhibit faint enhancement. Ancillary findings such as associated compression fractures and paravertebral tumor extension favor a diagnosis of neoplastic marrow replacement. Chronic anemias like sickle cell (see Chapter 96) and thalassemia are also frequent causes of diffuse marrow hypointensity on T1WI. In sickle cell, additional findings include H-shaped vertebral bodies due to infarctions leading to central vertebral body collapse with peripheral height maintenance. Characteristics of an acute infarct include the presence of irregular areas of marrow edema manifesting as high SI on T2WI. Serpentine enhancement is also characteristic, as opposed to the enhancement with osteomyelitis—which may also occur in sickle cell—which is typically more rounded or diffuse. Secondary findings of bone infarct and infection are less common in thalassemia than in sickle cell, but in both conditions frequently administered blood transfusions may lead to an accumulation of iron within tissue including the vertebrae. Associated susceptibility effects lead to a markedly diminished SI on FSE and GRE T2WI. In diseases such as thalassemia, hereditary spherocytosis, and myelosclerosis extramedullary hematopoiesis may also occur as a compensatory response to insufficient marrow red blood cell production. Favored sites include the spleen, liver, and lymph nodes. Thoracic involvement is rarer but may be

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appreciated on imaging as a paraspinal mass resulting from extrusion of proliferating marrow from vertebral bodies to a subperiosteal location. This will appear on MR, along with diffuse low SI vertebral marrow on T1WI, as multiple, smoothly marginated, paraspinal masses without bone erosions. In thalassemia in particular, these masses may grow large enough to compress the spinal canal. These masses typically demonstrate isointensity to vertebral body marrow on T1 and T2WI, and may enhance to varying degrees, confusing the overall appearance with one of multiple myeloma, metastatic disease, or lymphoma.