MRI of Spinal Bone Marrow: Part 2, T1-Weighted Imaging-Based Differential Diagnosis

Christopher J. Hanrahan
Lubdha M. Shah

OBJECTIVE. The purpose of this article is to review the structure of bone marrow and the differential diagnosis of bone marrow pathology on the basis of T1-weighted MRI patterns.

CONCLUSION. Bone marrow is an organ that is evaluated routinely during MRI of the spine, particularly lumbar spine evaluation. Thus, it is one of the most commonly performed MRI examinations. T1-weighted MRI is a fundamental sequence in evaluating spinal marrow, and an understanding of T1-weighted MR signal abnormalities is important for the practicing radiologist.

MRI of the spine is a commonly used diagnostic tool in patients with back pain and requires an understanding of not only the findings of common degenerative changes but also of the normal appearance of bone marrow and common benign lesions.

Because the majority of people will experience back pain at some point in their lives, it is not surprising that back pain results in 2.8% of all physician visits [1]. Most patients are treated conservatively without need for imaging; however, patients with persistent back pain, radiculopathy, or a history of cancer routinely undergo MRI [2]. Although the frequency of malignancy or infection is very low in the primary care population (< 1%) [2], distinguishing normal spinal marrow from pathology on MRI is essential to avoid missing pathology or misinterpreting normal changes, either of which may result in unnecessary additional testing. MRI can detect early bone marrow deposits because it is the only clinical imaging technique that allows direct visualization of bone marrow with high spatial resolution.

Bone Marrow Structure and Function

The bone marrow is a surprisingly large organ that is responsible for normal hematopoiesis and accounts for approximately 5% of body weight in an adult human [3, 4]. The cellular structure of bone marrow is complex, containing stem cells responsible for the production of erythrocytes, granulocytes, monocytes, lymphocytes, and platelets [4]. The supportive cellular environment, including macrophages, adipocytes, osteoblasts, osteoclasts, and adventitial reticular cells, provides the nutrients and cytokines that allow the proliferation, differentiation, and maturation of the hematopoietic cells [4]. A rich vascular supply of venous sinuses supplies nutrients and provides ready access to release blood cells into circulation [3, 4].

Macroscopic Appearance of Marrow

The bone marrow microenvironment provides the components that constitute the macroscopic bone marrow, which gives rise to the terms red marrow and yellow marrow. Red marrow is more cellular, containing the hematopoietic stem cells and blood cell progenitors that give rise to the peripheral blood, whereas yellow marrow contains more fat and is less cellular [3]. Red marrow contains approximately 40% fat, whereas yellow marrow contains 80% fat [3]. This difference is important for understanding the normal and abnormal appearance of marrow on T1-weighted MRI.

Differential Diagnosis of Bone Marrow Pathology

Differential signal intensity (SI) can be used to characterize bone marrow cellular content, which is useful in differentiating pathology. Whereas a routine nonselective iliac crest bone marrow biopsy provides important cellular and structural information, imaging can provide a noninvasive and more...
Hanrahan and Shah

A global picture of the bone marrow cellular composition [3, 5]. A quantitative assessment of particular cell types is not possible using MRI; however, the relative proportion of active and nonactive cellular components can be determined. The relative SI of lesions can raise suspicion for malignant cells. That is, bone marrow infiltration or replacement with malignant cells tends to produce focal or diffuse areas of T1-weighted signal that are equal to or lower than muscle [3]. Because red marrow contains intermixed fat, it typically has T1-weighted SI that is higher in intensity than muscle. However, in cases of profound red marrow reconversion, red marrow may be difficult to differentiate from malignancy [3]. Additional descriptive considerations of the lesions include the distribution (e.g., diffuse vs infiltrative, focal, or multifocal), location (e.g., body, endplate, or posterior elements), and morphology (e.g., discrete border vs aggressive margin). MRI may serve to guide biopsy of areas of abnormal SI [5]. The remaining sections will discuss the differential diagnosis of abnormal marrow signal within the spinal bone marrow on T1-weighted imaging (Table 1).

**Focal T1-Weighted Signal Increase**
T1-hyperintense bone marrow lesions are usually benign. The location and character of the focal fatty lesion will help determine the cause. Differential considerations for focal T1 hyperintensity include normal variant, focal fatty marrow, solitary hemangioma, lipoma, Paget disease, bone marrow hemorrhage, melanoma, and Modic type 2 discogenic degenerative endplate changes and other postinflammatory focal marrow atrophy [6].

**Solitary Hemangioma**
Vertebral hemangiomas are relatively common, occurring in 11% of patients in a large autopsy series [7]. The majority of hemangiomas in the spine have the classic appearance of coarsened trabeculae that have a corduroy pattern on sagittal CT or radiography and a polka-dot appearance on axial CT [8]. These are characterized by increased T1- and T2-weighted SI corresponding to the increased fat content [8].

**Degenerative Disk Disease**
Fatty degeneration of the marrow occurs in 16–23% of patients evaluated for disk disease [6, 9]. On MRI, Modic type 2 discogenic degenerative endplate changes exhibit hyperintense T1-weighted SI, isointense to hyperintense T2-weighted SI, and hypointense STIR SI. Type 1 change, in which there is destruction and fissuring of the endplate, progresses to type 2 changes with healing of subchondral bone [10]. Ongoing inflammatory processes in some type 2 changes are thought to be the causes of the conversion of yellow to red marrow, resulting in mixed type 1/2 Modic change [9, 11].

### TABLE 1: Differential Diagnosis of T1 Signal Changes in Spinal Marrow

<table>
<thead>
<tr>
<th>T1 Signal Pattern</th>
<th>Location</th>
<th>Potential Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal T1 signal increase</td>
<td>Any bone marrow</td>
<td>Normal variant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Focal fatty marrow</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Solitary hemangioma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Degenerative disk disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Paget disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Melanoma metastasis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bone marrow hemorrhage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lipoma</td>
</tr>
<tr>
<td>Diffuse or multifocal increase in T1 signal</td>
<td>Any bone marrow</td>
<td>Prior radiation treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Osteoporosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Multiple hemangiomas</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Spondyloarthropathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anorexia nervosa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic malnutrition</td>
</tr>
<tr>
<td>Focal T1 signal decrease</td>
<td>Endplate centered</td>
<td>Degenerative endplate changes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Osteomyelitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amyloid</td>
</tr>
<tr>
<td></td>
<td>Primarily in vertebral body</td>
<td>Atypical hemangioma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Malignancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrous dysplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metastasis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myeloma</td>
</tr>
<tr>
<td></td>
<td>Centered in posterior elements</td>
<td>Primary bone tumor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fracture</td>
</tr>
<tr>
<td>Diffuse or multifocal decrease in T1 signal</td>
<td>Any bone marrow</td>
<td>Hematopoietic hyperplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neoplasm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal osteodystrophy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sarcoidosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Spondyloarthropathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myelofibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mastocytosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hemosiderosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gaucher disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gout</td>
</tr>
</tbody>
</table>
**Paget Disease**

Paget disease (osteitis deformans) affects 3.0–3.7% of the population over 40 years of age and increases with age [12, 13]. The spine is the second most commonly affected site after the pelvis and comprises 30–75% of cases. Paget disease is characterized by a disturbance in bone remodeling due to an increase in osteoblastic and osteoclastic activity, which results in trabecular disorganization, fatty marrow, and vertebral body expansion. Three phases are seen: lytic, mixed, and blastic. Marrow SI changes vary with the stage of disease [14]. Low T1-weighted SI and mild high T2-weighted SI are seen in the mixed hyper-vascular phase. The vertebral body may show a thickened cortex, which can result in a picture-frame appearance on radiographs [15]. The blastic or sclerotic phase shows low T1-weighted and T2-weighted SI because of increased trabecular thickness, sclerosis, and marrow fibrosis. This can give the ivory vertebrae manifestation on radiographs [15]. In the fatty transformation later stage, there is hyperintensity on both T1-weighted and T2-weighted images. This fat SI can be helpful in guiding treatment; if there is fat SI in the presence of osteolyis in a pagetic vertebral body, malignant transformation is considered less likely and the patient may be treated conservatively [16].

**Melanoma Metastasis**

Although T1-hyperintense lesions are typically benign, correlation with the appearance on other MR sequences and imaging modalities as well as with clinical history may suggest an alternative diagnosis. Melanoma metastases appear as well-circumscribed T1-hyperintense lesions because of the melanin or hemorrhage [17] (Fig. 1). The T1 hyperintensity may be due to the paramagnetic effects of melanin [18]. These lesions show T2 shortening; however, the correlation between melanin content and T2 shortening is weaker than its association with T1 shortening [17]. Contrast-enhanced T1-weighted imaging with fat saturation may increase the conspicuity of melanin content and T2 shortening is weaker than its association with T1 shortening [17]. These lesions show T2 hyperintensity because of the melanin ground marrow signal.

**Bone Marrow Hemorrhage**

Vertebral marrow hemorrhage may present as areas of T1-weighted hyperintensity. In the setting of fracture, initially the edema and hemorrhage appear as T1-weighted hypointensity. As the edema resolves and blood products begin maturing, there is increased T1-weighted hyperintensity because of methemoglobin, resulting in variable T1-weighted signal with regions of T1-weighted hyperintensity mixed with foci of T1 hypointensity [19]. In addition, evolving bone marrow necrosis may show T1-weighted and T2-weighted hyperintensity, which is attributed to blood and proteinaceous debris within hyperemic marrow [20].

**MRI of Spinal Bone Marrow**

**Lipoma**

Lipomas are rare T1-weighted hyperintense lesions within vertebral bodies, with a prevalence of less than 0.1–2.5% of primary bone tumors [21]. Spinal lipomas represent 4% of intraosseous lipomas. Various stages of involution of intraosseous lipomas have been described: lipocytes forming solid tumor; mixture of lipocytes, fat necrosis, and focal calcification; and reactive bone formation admixed with necrotic adipose tissue [22]. The lesions show signal intensities similar to fat on all MR sequences and would be expected to be hypointense on fat-suppressed images.

**Diffuse or Multifocal Increase in T1-Weighted Signal**

Diffusely increased T1-weighted hyperintensity indicates decreased cellularity of bone marrow. The differential diagnosis includes normal variant, irradiated marrow, osteoporosis, heterogeneous fatty marrow, and multiple hemangiomas. Rare diagnoses that may present with diffuse T1-weighted hyperintensity include anorexia and aplastic anemia.

**Prior Radiation Treatment**

Irradiated bone marrow undergoes characteristic time-dependent changes that ultimately result in increased T1-weighted signal with a sharp demarcation corresponding to the radiation port (Fig. 2). Radiation destroys the sinusoidal vasculature, with hematopoietic marrow being replaced by fatty marrow and results in hypocellular bone marrow [23]. At doses above 36 Gy, fatty replacement is permanent, with little chance of hematopoietic recovery. Below doses of 30 Gy, these changes are likely reversible and generally occur within 12–24 months [24, 25].

**Osteoporosis**

On MRI, osteoporosis can have a heterogeneous appearance because of decreased cellular marrow components and increased fat content [26, 27]. T1-weighted images show heterogeneously hypointense signal intensity, but the T2-weighted signal can be variable. Focal fatty marrow usually has rounded lesions that coalesce to involve the entire vertebral body. Because of the round morphology, differentiating multiple focal fatty marrow sites from osteoporosis or multifocal hemangiomas may sometimes be difficult. In these cases, comparison with radiography, CT, or dual-energy x-ray absorptiometry can help to elucidate the correct diagnosis.

**Multiple Hemangiomas**

Vertebral body hemangiomas are benign usually incidental lesions. Hemangiomas can rarely be aggressive with epidural involvement and cord compression [8, 28]. Multiple hemangiomas are seen as multiple T1-weighted hyperintense lesions in the vertebral bodies or posterior elements. Fast spin-echo T2-weighted MRI can be helpful in confirming the diagnosis because these lesions are most often T2-hypointense. Depending on the balance of fat and vascular elements, they may or may not be hypointense on STIR images.

**Spondyloarthropathy**

Focal superior endplate T1-weighted hyperintensity, usually (but not exclusively) involving the anterior corners of the endplate, may be observed in the postinflammatory stage of ankylosing spondylitis because of fatty infiltration (Fig. 3). These are termed Romanus lesions and are a delayed manifestation involving the edges of the vertebral endplates secondary to enthesitis of the anterior or posterior longitudinal ligamentous complexes [29]. Later vertical bone outgrowths, or syndesmophytes, form between adjacent vertebrae and are the end stage of ankylosing spondylitis.

**Anorexia Nervosa**

Marrow changes in patients with anorexia nervosa are attributable to early osteoporosis and premature conversion of red marrow to yellow marrow [30]. Other studies have shown a waterlike SI pattern in the marrow spaces [31], which has been termed “serous atrophy.” This appearance has been attributed to the gelatinous transformation of the bone marrow, which is characterized by fat cell atrophy, loss of hematopoietic cells, and deposition of extracellular gelatinous substances, indicating increased severity of the disease [31] (Fig. 4). This waterlike signal intensity is a sign of a generalized severe illness and also can be visualized in patients with alcoholism, malignancies, chronic heart failure, and HIV/AIDS [32].
Focal T1-Weighted Signal Decrease

Focal low T1-weighted signal within spinal bone marrow provides a diagnostic dilemma because there are many causes of localized low T1-weighted SI. Therefore, it is useful to consider the location of the signal abnormality, including signal abnormality centered or confined to the endplate primarily in the vertebral body or centered in the posterior elements.

Endplate Centered

Degenerative endplate changes—Degenerative disk disease has been known to affect adjacent endplate bone marrow since the correlation of degenerative changes with histology by Modic et al. [6]. Generally, the degenerative endplate changes are characteristic and unlikely to be confused with other pathology. Low T1-weighted signal in types 1 and 3 endplate change is due to edema and fibrovascular change and fibrotic change, respectively [6]. Characteristic dorsal vertebral corner defects are sometimes visualized with disk herniations and should not be confused with other pathology [33]. When the signal changes are early and more focal, they could be confused with metastatic disease or other pathology.

Osteomyelitis—Although the majority of cases of discitis-osteomyelitis will present with characteristic findings, early or atypical discitis-osteomyelitis could present with nonspecific findings. The typical imaging presentation of pyogenic discitis-osteomyelitis is low T1-weighted signal involving the vertebral body endplates and increased T2-weighted signal in the disk [34]. Granulomatous discitis-osteomyelitis can spare the disk space, but there is usually paraspinal soft-tissue involvement [34]. Occasionally in early discitis-osteomyelitis or in atypical cases, the imaging presentation may be that of a single vertebral body with low T1-weighted signal that could mimic a metastatic lesion or myeloproliferative disorder [34].

Amyloid—Rarely, focal decreased T1-weighted signal can be caused by amyloid deposition [35], which occurs in long-standing renal dialysis patients, who are usually elderly [35]. The manifestations include low T1-weighted signal usually subjacent to the endplates within the lower cervical spine in the majority of cases [35]. Less commonly, there is involvement of the thoracic or lumbar spine [36]. Typically, the regional endplates show both low T1-weighted and T2-weighted signal; however, in some cases, the T2-weighted signal can be intermediate to high and those cases may be indistinguishable from discitis-osteomyelitis [36].

Primarily in the Vertebral Body

Hemangioma—Most hemangiomas present with the characteristic high T1-weighted and T2-weighted signal; however, some hemangiomas may appear low or intermediate in SI on T1-weighted images [37, 38]. This SI on T1-weighted MRI corresponds to less fat, which may indicate a more vascular and potentially a more aggressive hemangioma [38]. These can be confused with malignant lesions, but fortunately CT often reveals the characteristic coarsened trabeculae unless the lesion is small [39].

Fracture—Fractures of vertebral bodies can present with focal decreased T1-weighted signal, usually in the acute setting [19, 40]. Fractures can occasionally be confused with an acute Schmorl node or pathologic compression fracture. A Schmorl node cannot present with the typical undulated endplate appearance, and follow-up MRI or CT can be helpful to distinguish the abnormality [41]. Distinguishing whether a compression fracture is an insufficiency or pathologic fracture may pose a dilemma [19, 40]. Often, specific MRI characteristics can help differentiate benign from malignant fractures. Findings of a convex posterior vertebral body border, abnormal SI involving one or both pedicles, epidural or focal paraspinal mass, or additional spinal metastases are suggestive of a pathologic fracture [40]. Findings of a low T1-weighted SI band with other areas of normal marrow, retropulsion, or multiple compression fractures are indicative of a benign fracture [40]. In- and out-of-phase imaging and diffusion-weighted imaging may also be helpful in problematic cases [42–44], although the usefulness of diffusion-weighted imaging remains controversial [43].

Malignancy—Focal areas of abnormal signal may represent neoplasm. The most common neoplastic processes that involve the spine are metastatic disease, lymphoma, and plasma cell dyscrasia, either solitary plasmacytoma or multiple myeloma [45]. Neoplastic foci within the spine produce signal changes on T1-weighted images reflecting increased cellularity of the neoplastic lesions with infiltration or replacement of fat [3]. The majority of neoplastic cases can be differentiated from red and yellow marrow by signal that is lower than the adjacent disk or muscle [3, 5]. Difficulty may be encountered with intermediate T1-weighted vertebral body hemangiomas, which have little intralysosomal fat and may be distinguished from metastatic disease by CT [46]. In addition to the most common lesions discussed previously, primary tumors of the spine can produce low T1-weighted signal within the vertebral body. More often than not, primary bone tumors of the vertebral body present with paravertebral or epidural extension (Fig. 5). Exceptions to this do occur, and biopsy of a bone marrow lesion or imaging follow-up may be warranted.

Fibrous dysplasia—Fibrous dysplasia is a rare benign fibroosseous lesion of bone that can manifest in multiple locations in bone (polyostotic) or a single location (monostotic) [47]. Fibrous dysplasia is extremely uncommon in the spine; most cases have been associated with polyostotic fibrous dysplasia, but it can manifest as the monostotic form [48] (Fig. 6). Typically, fibrous dysplasia has low T1-weighted SI and variable T2-weighted SI [47, 49]. Enhancement varies depending on the histologic composition of any given lesion [49]. Despite its rarity, fibrous dysplasia should be included on the list of bone marrow abnormalities that cause decreased T1-weighted signal.

Centered in the Posterior Elements

T1-weighted signal abnormality within the posterior elements can be the result of metastasis, myeloma, lymphoma, fracture, or primary bone tumor. In our experience at a center for multiple myeloma treatment, we frequently see myeloma lesions in the posterior elements. Without any other bone marrow involvement, a solitary T1-weighted signal abnormality in the posterior elements is more likely caused by a primary bone tumor. If a primary tumor is suspected, a lesion appears aggressive, or extensive signal abnormality is present, CT can be helpful in further characterizing a lesion, especially for detection of internal matrix [45].

Diffuse or Multifocal Decrease in T1-Weighted Signal

Diffuse or multifocal decreased T1-weighted signal is caused by replacement of fatty marrow, which can be due to cellular tissue or edema. Diffuse benign processes include hematopoietic marrow hyperplasia and hemosiderin deposition. Systemic inflammatory processes, such as sarcoidosis, gout, or spondyloarthropathy, can also have extensive osseous involvement. Neoplastic

Hanrahan and Shah

1312 AJR:197, December 2011
cell infiltration resulting in diffuse spinal T1-weighted hypointensity can be seen with hematologic malignancies.

**Hematopoietic Hyperplasia**

When the hematopoietic capacity of the existing red marrow is exceeded, fatty marrow reconverts to red marrow. The pattern of reconversion is the reverse of that of physiologic marrow conversion, from axial to appendicular. Hematopoietic hyperplasia can result in diffusely T1-weighted hypointense signal in the axial skeleton (Fig. 7), but the SI is usually slightly hyperintense relative to muscle on STIR and fat-suppressed T2-weighted images. It is often seen with chronic anemia, such as sickle cell disease, thalassemia, and hereditary spherocytosis [50] (Fig. 8). These diseases result in the expansion of red marrow leading to the widening of medullary spaces and thinning of cortical bone. The vertebral bodies can develop a biconcave deformity due to the cortical thinning and softening of bone, producing the characteristic fish vertebrae or in the case of infarction in sickle cell disease, H-shaped vertebrae [51, 52]. Similarly, red marrow conversion may occur in circumstances in which there is an increased oxygen requirement, such as in endurance athletes [53].

Chronic illnesses, heavy smoking, and obesity tax hematopoietic reserves and are associated with marrow hyperplasia [54]. In anemia of chronic illness, the release of iron from macrophages is impaired such that there is abnormally increased bone marrow iron [55], which manifests as decreased T1-weighted SI. HIV-positive patients may also show similar MR findings [56]. In addition, many cancer patients may be treated with agents to stimulate hematopoietic elements, such as granulocyte-colony-stimulating factor and erythropoietin, as an adjunct to chemotherapy and radiation, which can result in hyperplasia of red marrow [57].

**Neoplasia**

Diffuse T1 hypointensity of spinal marrow may be the result of neoplastic cell infiltration. Typically, neoplastic processes have lower T1-weighted signal and have hyperintense STIR and fat-suppressed T2-weighted signal relative to the adjacent disk and paramedian muscles (Fig. 9). Pathologic processes show avid enhancement, but this may be difficult to appreciate when it is a diffuse process. In these cases, comparison of unenhanced and gadolinium-enhanced bone marrow SI may be helpful. Most patients over 40 years old with diffuse marrow infiltration show a greater than 35% SI increase, whereas normal marrow shows a 35% or lower SI increase [58].

Spinal involvement and appearance vary with malignancies. In leukemia, bone marrow infiltration is often a component of systemic disease (Fig. 10). Low T1-weighted leukemic infiltrates are particularly apparent in yellow marrow but can be difficult to differentiate in patients with predominantly red marrow [59]. With lymphoma, 30% of patients have skeletal involvement, with the long bones being affected more than the spine [59]. Lymphoma may manifest with extension into the epidural space from the vertebral body. Metastases can present with diffuse or multifocal T1-weighted hypointensities. Metastases show cortical destruction more often than hematopoietic malignancies [60].

**Renal Osteodystrophy**

Renal osteodystrophy broadly applies to all pathologic features of bone in patients with renal failure. Abnormal retention of phosphate by the kidneys results in hyperphosphatemia, which in turn causes hypercalcemia and consequently hyperparathyroidism [61]. Therefore, any one of these disorders may produce renal osteodystrophy, in which the spine is osteopenic and exhibits heterogeneous T1-weighted signal. There is central demineralization with sclerosis of the margins. Low T1-weighted and T2-weighted signal along the endplates may give the characteristic rugger jersey appearance [62].

**Sarcoidosis**

Sarcoidosis is a multisystem disorder characterized by noncaseating epithelial granulomas, with osseous involvement seen in 1–13% of patients [63] (Figs. 11A–11C). Sclerotic lesions of the spine are uncommon but can mimic blastic metastatic disease [64] (Fig. 11D). Osseous involvement usually occurs at the initial manifestation but has been reported to appear many years after resolution of thoracic sarcoidosis [64]. MRI usually shows multifocal enhancing vertebral body lesions that are hypointense on T1-weighted images and hyperintense on T2-weighted images.

**Spondyloarthropathy**

Inflammatory lesions of spondyloarthropathy may present with focal areas of low T1-weighted signal in the acute stage. These acute lesions are low on T1-weighted images and high on T2-weighted and STIR images and have corresponding bone erosions on CT [65]. These are not pathognomonic for ankylosing spondylitis because the inflammatory lesions can also be seen with other spondyloarthropathies and the rare SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, and osteitis) [66].

**Myelofibrosis**

Myelofibrosis is characterized by the replacement of normal marrow by fibrotic tissue, which can result in anemia and extra medullary hematopoiesis. It is associated with chemotherapy or radiation therapy for lymphoma, leukemia, multiple myeloma, and metastatic disease. Occasionally, it is a primary disorder. The characteristic findings on MRI are very low T1-weighted and T2-weighted signal in the marrow and slightly hyperintense signal relative to muscle on fat-suppressed images.

**Mastocytosis**

In systemic mastocytosis, there is an abnormal proliferation of mast cells in the skin, bone marrow, spleen, liver, and lymph nodes. Other hematologic disorders, such as myeloproliferative and myelodysplastic syndromes, or lymphoreticular malignancies can coexist [67]. Mast cells can synthesize a variety of cytokines that may affect the skeletal system, increasing bone resorption and leading to osteoporosis [68]. Spinal marrow involvement may be homogeneous or heterogeneous and appear as T1-weighted hypointensity [69].

**Hemosiderosis**

Hemosiderosis can result in hypointense marrow signal on all MR sequences because of the magnetic susceptibility of hemosiderin. Breakdown of RBCs in hemolytic anemia can lead to hemosiderin accumulation, as can chronic blood transfusions [70]. A clue to the diagnosis will be hypointensity of the liver and spleen.

**Gaucher Disease**

Congenital diseases, such as Gaucher disease, may present with hypointense T1-weighted marrow signal (Fig. 12). This autosomal recessive disorder results in the accumulation of glucocerebrosides within histiocytes because of decreased levels of the enzyme glucocerebrosidase. The marrow disease begins in the axial skeleton after the distribution of reconverted marrow [71]. The marrow fat is re-
placed by infiltration of Gaucher cells, which manifest as T1-weighted and T2-weighted hypointensity. In some cases, the STIR sequences reveal hyperintense inclusions, which may indicate acute bone crisis, occult fracture, infection, or bone infarction [72].

**Gout**

Spinal involvement by gout is rare, with relatively few reports in the literature. Nevertheless, it should be kept in the differential diagnosis for areas of low T1-weighted signal. Gout has a variable MRI appearance and is most often manifested as areas of low T1-weighted and variable T2-weighted signal intensity, with homogeneous or peripheral gadolinium enhancement [73–76]. Gout can occur in the vertebral bodies, which sometimes mimics discitis-osteomyelitis. It can also involve the facet joints or odontoid process or occur within paraspinal soft tissues [73–75, 77–81]. Fortunately, most spinal gout cases occur in patients with known gouty arthropathy, which has a male predominance [73, 82]. In rare cases, gout can coexist with discitis-osteomyelitis [75]. CT may be helpful in identifying calcifications [74, 83], but biopsy may be necessary to establish the diagnosis.

**Acknowledgments**

The authors thank B. J. Manaster and Kent Sanders for helpful reviews of the manuscript.

**References**

35. Jung HS, Jee WH, McCauley TR, Ha KY, Choi KH. Discrimination of metastatic from acute os-
MRI of Spinal Bone Marrow


41. Wu HT, Morrison WB, Schweitzer ME. Edema
tous Schmorl’s nodes on thoracolumbar MR im-
aging: characteristic patterns and changes over time. Skeletal Radiol 2006; 35:212–219

42. Baur A, Stäbler, Brüning R, et al. Diffusion-
weighted MR imaging of bone marrow: differen-

43. Castillo M. Diffusion-weighted imaging of the

44. Disler DG, McCauley TR, Ratner LM, Kesack
CD, Cooper JA. In-phase and out-of-phase MR imaging of bone marrow: prediction of neoplasia
based on the detection of coexistent fat and water. AJR 1997; 169:1439–1447

nosis of imaging of solitary tumors of the spine: what

46. Zajick DC Jr, Morrison WB, Schweitzer ME, Parellada JA, Carrino JA. Benign and malignant
processes: normal values and differentiation with

47. Kranzfeld MJ, Moser RP, Gilkey FW. Fibrous

stotic fibrous dysplasia of the spine: a report of sev-

49. Jee WH, Choi KH, Choe BY, Park JM, Shinn KS. Fibrous dysplasia: MR imaging characteristics
with radiopathologic correlation. AJR 1996; 167:1523–1527

50. Stabler A, Doma AB, Baur A, Kruger A, Reiser
MF. Reactive bone marrow changes in infectious
spondylodiscitis: quantitative assessment with MR im-

51. Resnick DL. Fish vertebrae. Arthritis Rheum
1982; 25:1073–1077

52. Lonergan GJ, Cline DB, Abbondanzo SL. Sickle

topoietic bone marrow hyperplasia: correlation of
spinal MR findings, hematologic parameters, and
bone mineral density in endurance athletes. Radi-
ology 1996; 188:503–508

54. Poulton TB, Murphy WD, Duerr JL, Chapek CC,
Feiglin DH. Bone marrow reconversion in adults
who are smokers: MR Imaging findings. AJR 1993; 161:1217–1221

55. Wyatt SH, Fishman EK. CT/MRI of musculoskel-
etal complications of AIDS. Skeletal Radiol 1995;
24:481–488

findings in musculoskeletal complications of
AIDS. RadioGraphics 2004; 24:1029–1049

57. Altehoefer C, Bertz H, Ghanem NA, Langer M.
Extent and time course of morphological changes
of bone marrow induced by granulocyte-colony
stimulating factor as assessed by magnetic reso-

Reiser M. MRI gadolinium enhancement of bone
marrow: age-related changes in normals and in
diffuse neoplastic infiltration. Skeletal Radiol
1997; 26:414–418

59. Parker BR. Leukemia and lymphoma in child-

60. Kim EE, Jackson EF. Molecular imaging in on-
cology: PET, MRI, and MRS. New York, NY:
Springer, 1999;290

61. Parfitt AM. The hyperparathyroidism of chronic
1977; 52:3–9

Arthritis Rheum 1981; 24:1191–1194

63. Jelinek JS, Mark AS, Barth WF. Sclerotic lesions
of the cervical spine in sarcoidosis. Skeletal Radiol
1998; 27:702–704

64. Packer CD, Mileti L. Vertebral sarcoidosis mimicking lytic osseous metastases: development
16 years after apparent resolution of thoracic sar-

65. Hermann KG, Althoff CE, Schneider U, et al. Spi-
nal changes in patients with spondyloarthritis: com-
parison of MR imaging and radiographic ap-
ppearances. Radiographics 2005; 25:559–569;
discussion, 569–570

66. Laredo JD, Vuillem-Bodaghi V, Bourny N, Cot-
ton A, Parlier-Cuau C. SAPHO syndrome: MR
appearance of vertebral involvement. Radiology
2007; 242:825–831

67. Parker RI. Hematologic aspects of systemic mas-
14:557–568

68. Delling G, Ritzel H, Werner M. Histological char-
acteristics and prevalence of secondary osteoros-
sis in systemic mastocytosis: a retrospective
analysis of 158 cases [in German]. Pathologe
2001; 22:132–140

69. Roca M, Mota J, Girald P, Garcia Erce JA. Sys-
temic mastocytosis: MRI of bone marrow in-

70. Levin TL, Sheth SS, Hurlet A, et al. MR marrow
signs of iron overload in transfusion-dependent
patients with sickle cell disease. Pediatr Radiol
1995; 25:614–619

71. Hermann G, Shapiro RS, Abdelwahab IF,
Grabowski G. MR imaging in adults with Gauch-
er disease type I: evaluation of marrow involve-
ment and disease activity. Skeletal Radiol 1993;
22:247–251

72. Maas M, Hollak CE, Akkerman EM, Aerts JM,
Stoker I, Den Heeten GJ. Quantification of skele-
tal involvement in adults with type I Gaucher’s
disease: fat fraction measured by Dixon quantita-
tive chemical shift imaging as a valid parameter.
AJR 2002; 179:961–965

73. Pfister AK, Schlarb CA, O’Neal JF. Vertebral ero-
sion, paraplegia, and spinal gout. AJR 1998;
171:1430–1431

74. Hsu CY, Shih TT, Huang KM, Chen PQ, Sheu JJ,
Li YW. Tophaceous gout of the spine: MR imag-

75. Tsai CH, Chen YJ, Hsu HC, Chen HT. Bacteremia
coxistising with tophaceous gout of the spine mimicking spondyloarthritis: a case report. Spine
2009; 34:E106–E109

76. Duprez TP, Malghem J, Vande Berg BC, Noel
HM, Munting EA, Muldauek BE. Gout in the cer-
vical spine: MR pattern mimicking diskoverte-
bre donation. AJR 1996; 17:151–153

77. Hausch R, Wilkerson M, Singh E, Reves C, Har-
rington T. Tophaceous gout of the thoracic spine
presenting as back pain and fever. J Clin Rheuma-
tol 1999; 5:335–341

78. Hou LC, Hsu AR, Veeravagu A, Boakye M. Spi-
nal gout in a renal transplant patient: a case report
discussion, 73

79. Mahmud T, Basu D, Dyson PH. Crystal arthrop-
y of the lumbar spine: a series of six cases and a
2005; 87:513–517

80. Popovich T, Carpenter JS, Rai AT, Carson LV,
Williams HJ, Marano GD. Spinal cord compres-
sion by tophaceous gout with fluorodeoxyglucose-
 positon-emission tomographic/MR fusion imag-
ing. AJR 2006; 27:1201–1203

81. Vinsent AL, Cockerill EM. Involvement of the
spine in gout: a case report. Radiology 1972;
103:311–312

82. King JC, Nicholas C. Gouty arthropathy of the
lumbar spine: a case report and review of the lit-

83. Gerster JC, Landry M, Dufresne L, Mewely JY.
Imaging of tophaceous gout: computed tomogra-
phy provides specific images compared with mag-
A

Hanrahan and Shah

Fig. 1—48-year-old man with metastatic melanoma. Sagittal T1-weighted MR image reveals hyperintense metastatic melanoma deposit in midthoracic vertebral body (thick arrow). There is intramedullary hyperintense metastatic focus (thin arrow) with associated mild cord expansion and hypointensity, which is compatible with edema. Extensive paraspinal-mediasinal metastatic lymphadenopathy (arrowhead) is also seen.

B

Fig. 2—Fatty marrow due to radiation therapy in 53-year-old man with colon adenocarcinoma. Sagittal T1-weighted MR image shows diffuse fatty marrow replacement of thoracic spine as sequela of radiation therapy.

Fig. 3—Ankylosing spondylitis in 35-year-old woman with 2-year diagnosis of ankylosing spondylitis who was treated with etanercept (Enbrel, Amgen) for 2 months before MRI. A and B, Sagittal T1-weighted (A) and STIR (B) images show high signal on T1-weighted and low signal on STIR image involving vertebral body corners, corresponding to chronic Romanus lesions (arrowheads).
Fig. 4—Malnutrition in 43-year-old man with gelatinous transformation (serous atrophy) of bone marrow secondary to severe malnutrition.
A, Sagittal T1-weighted MR image depicts hypointense signal in corresponding regions. Height and cortical integrity are preserved.
B, Sagittal STIR image shows ill-defined hyperintensity in lumbar vertebral bodies, particularly adjacent to basivertebral plexus.
C, Contrast-enhanced sagittal T1-weighted MR image with fat saturation exhibits amorphous enhancement in regions of fluid signal intensity noted on STIR and T1-weighted images.

Fig. 5—Ewing sarcoma in 30-year-old woman who presented with left-sided radiculopathy.
A, Sagittal T1-weighted image shows low signal intensity of nearly entire L5 vertebral body bone marrow, with epidural extension.
B, Axial gadolinium-enhanced T1-weighted image with fat saturation depicts large epidural component of mass (arrow), extent of extramedullary spread into psoas muscle, and encasement of exiting left L5 nerve root (arrowhead).
Fig. 6—Fibrous dysplasia in 28-year-old woman with 3-year history of lower back pain, progressively worse in recent months. Biopsy of L2 lesion revealed fibrous dysplasia.
A, Sagittal T1-weighted MR image shows discrete rounded hypointense lesion in L2 vertebral body (arrow). There is no cortical destruction, fracture, or soft-tissue mass.
B, Enhanced T1-weighted MR image with fat saturation depicts mild homogeneous enhancement of well-circumscribed lesion in L2 vertebral body (arrow).

Fig. 7—Bone marrow hyperplasia in 35-year-old woman with end-stage renal failure, chronic anemia, and erythropoietin treatment.
A and B, Sagittal T1-weighted (A) and STIR (B) images depict very low T1-weighted signal in bone marrow, with disks brighter than vertebral marrow and low STIR signal intensity, corresponding to red marrow hyperplasia.
MRI of Spinal Bone Marrow

Fig. 8—17-year-old girl with thalassemia with extramedullary hematopoiesis in patient with diffuse red marrow replacement of spinal bone marrow. A and B, Sagittal thoracic spine T1-weighted (A) and T2-weighted (B) MR images show decreased fat in marrow on basis of decreased T1-weighted signal intensity and low T2-weighted signal (arrowheads). Extensive epidural extramedullary hematopoiesis is also present (arrows).

Fig. 9—65-year-old man with Waldenstrom macroglobulinemia. Sagittal T1-weighted image through thoracic spine shows reversal of normal spinal marrow appearance on T1-weighted images with signal in vertebral body marrow (arrowhead) that is lower in signal intensity than adjacent disk (arrow).

Fig. 10—34-year-old man with recurrent diffuse leukemic infiltrate of bone marrow. A and B, Sagittal cervical spine T1-weighted (A) and T2-weighted (B) MR images show reversal of marrow signal intensity with disks (arrows, A) brighter than vertebral body marrow (arrowheads, A) on T1-weighted image. Diffuse very low T2-weighted signal is present in vertebral marrow on T2-weighted image (arrow, B).
Fig. 11—Sarcoidosis. A–C, 66-year-old man with systemic sarcoidosis. Sagittal T1-weighted (A), T2-weighted (B), and enhanced T1-weighted MR images (C) demonstrate hypointense lesions in the T8, T11, T12, and L1 vertebral bodies (arrows) that enhance with gadolinium and correspond to the mixed lytic-sclerotic lesions of osseous sarcoidosis. (Fig. 11 continues on next page)
MRI of Spinal Bone Marrow

Fig. 11 (continued)—
Sarcoidosis.
D, 45-year-old woman with sarcoidosis. Heterogeneous bone marrow fat is present on this T1-weighted image with multiple lesions apparent in L4 and L5 with low-intensity rim (arrowheads). These corresponded to sclerotic lesions on CT performed 6 months before. Normal bone was noted on CT performed 1 year before and also on lumbar spine MRI performed 5.5 years before this MRI.

Fig. 12—Gaucher disease in 35-year-old man. Sagittal T1-weighted MR image reveals diffuse heterogeneous hypointensity due to marrow infiltration of vertebral bodies as well as posterior elements (arrow). Central focal endplate depressions in mid thoracic spine are due to bone infarcts (arrowhead).

FOR YOUR INFORMATION
The reader’s attention is directed to part 1 accompanying this article, titled “MRI of Spinal Bone Marrow: Part 1, Techniques and Normal Age-Related Appearances,” which begins on page 1298.

FOR YOUR INFORMATION
The Self-Assessment Module accompanying this article can be accessed via www.ajronline.org at the article link labeled “CME/SAM.”

The American Roentgen Ray Society is pleased to present these Self-Assessment Modules (SAMs) as part of its commitment to lifelong learning for radiologists. Each SAM is composed of two journal articles along with questions, solutions, and references, which can be found online. Read each article, then answer the accompanying questions and review the solutions online. After submitting your responses, you’ll receive immediate feedback and benchmarking data to enable you to assess your results against your peers.

Continuing medical education (CME) and SAM credits are available in each issue of the AJR and are free to ARRS members. Not a member? Call 1-866-940-2777 (from the U.S. or Canada) or 703-729-3353 to speak to an ARRS membership specialist and begin enjoying the benefits of ARRS membership today!
This article has been cited by:

1. Thomas Van Den Berghe, Koenraad L. Verstraete, Frédéric E. Lecouvet, Maryse Lejoly, Julie Dutoit. 2022. Review of diffusion-weighted imaging and dynamic contrast-enhanced MRI for multiple myeloma and its precursors (monoclonal gammopathy of undetermined significance and smouldering myeloma). *Skeletal Radiology* 51:1, 101-122. [Crossref]

2. Eo-Jin Hwang, Sanghee Kim, Joon-Yong Jung. 2022. Fully automated segmentation of lumbar bone marrow in sagittal, high-resolution T1-weighted magnetic resonance images using 2D U-NET. *Computers in Biology and Medicine* 140, 105105. [Crossref]


8. Kheng Song Leow, Keynes T. A. Low, Wilfred C. G. Peh. Magnetic Resonance Imaging of Spinal Infection 51-69. [Crossref]


12. Shivani Gogi, Veena Madireddy, Vanaja Bulpapuram, Vijaya Kumari Mudunuri. 2020. MRI in Bone Marrow Imaging - A Prospective Study in a Tertiary Hospital. *Journal of Evidence Based Medicine and Healthcare* 7:34, 1722-1729. [Crossref]

13. Zhao Wei, Ya-Jun Ma, Hyungseok Jang, Wenhui Yang, Jiang Du. 2020. To measure T1 of short T2 species using an inversion recovery prepared three-dimensional ultrashort echo time (3D IR-UTE) method: A phantom study. *Journal of Magnetic Resonance* 314, 106725. [Crossref]

14. Megha D. Patel, James Brian, Nancy A. Chauvin. 2020. Pearls and Pitfalls in Imaging Bone Marrow in Pediatric Patients. *Seminars in Ultrasound, CT and MRI* . [Crossref]


17. Xiaojuan Li, Ann V. Schwartz. 2020. MRI Assessment of Bone Marrow Composition in Osteoporosis. *Current Osteoporosis Reports* 18:1, 57-66. [Crossref]

18. Divya G, Ankamma Rao D. 2020. MRI Signal Intensity Changes of Vertebral Bone Marrow. *Journal of Evolution of Medical and Dental Sciences* 9:04, 227-230. [Crossref]

19. Nancy M. Major, Mark W. Anderson, Clyde A. Helms, Phoebe A. Kaplan, Robert Dussault. Marrow 23-52. [Crossref]

21. Terrell E. Jones, Aaron J. Wyse, Sarah E. Gibson. 2019. Hematolymphoid neoplasms are common in bone marrow biopsies performed for non-specific, diffuse marrow signal alterations on magnetic resonance imaging. *Annals of Diagnostic Pathology* 40, 7-12. [Crossref]

22. Yu. V. Nazinkina. 2019. BONE MARROW LESIONS: DIAGNOSIS WITHOUT BIOPSY. *Diagnostic radiology and radiotherapy* ;1, 19-25. [Crossref]


25. Jiyo S. Athertya, Gurunathan Saravana Kumar, Jayaraj Govindaraj. 2019. Detection of Modic changes in MR images of spine using local binary patterns. *Biocybernetics and Biomedical Engineering* 39:1, 17-29. [Crossref]


28. Davide Caramella, Fabio Chiesa. Essentials of MR Image Interpretation 317-350. [Crossref]


33. Joohee Lee, Yeon Hwa Yoo, Sarah Lee, Hak Sun Kim, Sungjun Kim. 2018. Gelatinous Transformation of Bone Marrow Mimicking Malignant Marrow-Replacing Lesion on Magnetic Resonance Imaging in a Patient without Underlying Devastating Disease. *Investigative Magnetic Resonance Imaging* 22:1, 50. [Crossref]


37. Hye Jin Yoo, Sung Hwan Hong, Dong Hyun Kim, Ja-Young Choi, Hee Dong Chae, Bo Mi Jeong, Heung Sik Kang. 2017. Measurement of fat content in vertebral marrow using a modified dixon sequence to differentiate benign from malignant processes. *Journal of Magnetic Resonance Imaging* 45:5, 1534-1544. [Crossref]

38. Antoine Feydy. 2017. Imagerie des métastases osseuses : quels examens effectuer ?. *Revue du Rhumatisme Monographies* 84:2, 121-129. [Crossref]

39. Patrick Rock, Aneta Kecler-Pietrzyk. Diffuse T1 bone marrow signal loss . [Crossref]
42. Red to Yellow Marrow Conversion 762-763. [Crossref]
43. Mostafa El-Feky, Mohammed Khader.O.Thabet. Focal fatty deposits in spinal bone marrow . [Crossref]
44. J.C. Vilanova, A. Luna. 2016. Infiltración de la médula ósea, mieloma múltiple y enfermedad metastásica. Radiología 58, 81-93. [Crossref]
46. Lymphoma 411. [Crossref]
47. Robert D. Boutin, Lawrence M. White, Tal Laor, Damon J. Spitz, Robert R. Lopez-Ben, Kathryn J. Stevens, Miriam A. Bredella. 2015. MRI findings of serous atrophy of bone marrow and associated complications. European Radiology 25:9, 2771-2778. [Crossref]
48. J. Martel Villagrán, Á. Bueno Horcajadas, E. Pérez Fernández, S. Martín Martin. 2015. Accuracy of magnetic resonance imaging in differentiating between benign and malignant vertebral lesions: Role of Diffusion-weighted imaging, in-phase/opposed-phase imaging and apparent diffusion coefficient. Radiología (English Edition) 57:2, 142-149. [Crossref]
49. J. Martel Villagrán, Á. Bueno Horcajadas, E. Pérez Fernández, S. Martín Martin. 2015. Precisión de la resonancia magnética en la diferenciación entre lesión vertebral maligna y benigna: papel de las secuencias de difusión, del cociente fuera de fase/en fase y de los valores del coeficiente de difusión aparente. Radiología 57:2, 142-149. [Crossref]
50. Ahmed Hamimi, Farid Kassab, Ghaith Kazkaz. 2015. Osteoporotic or malignant vertebral fracture? This is the question. What can we do about it?. The Egyptian Journal of Radiology and Nuclear Medicine 46:1, 97-103. [Crossref]
51. Simona Gaudino, Matia Martucci, Raffaella Colantonio, Emilio Lozupone, Emiliano Visconti, Antonio Leone, Cesare Colosimo. 2015. A systematic approach to vertebral hemangioma. Skeletal Radiology 44:1, 25-36. [Crossref]
52. Paul Babyn, Jennifer Stimec. Bone Marrow 873-901. [Crossref]
53. Mihrə S. Təljanov, Kevin Hoover. Musculoskeletal System 145-183. [Crossref]
54. Lymphoma 746-759. [Crossref]
55. Hyperplastic Vertebral Marrow 956-959. [Crossref]
56. Jeremy Jones, Hom Pant. Osteoporosis . [Crossref]
57. Dalia Z. Zidan, Hesham A. Elghazaly. 2014. Can unenhanced multiparametric MRI substitute gadolinium–enhanced MRI in the characterization of vertebral marrow infiltrative lesions?. The Egyptian Journal of Radiology and Nuclear Medicine . [Crossref]
58. A. Feydy, F. Paycha, S. Wong–Hee–Kam. Traitements des métastases osseuses 243-264. [Crossref]
60. Bhasker Koppula, Justin Kaptuch, Christopher J. Hanrahan. 2013. Imaging of Multiple Myeloma: Usefulness of MRI and PET/CT. Seminars in Ultrasound, CT and MRI 34:6, 566-577. [Crossref]
63. Alan Victor Krauthamer, Sasan Partovi, John Lyo. Neoplastic Disease of the Spine 141-231. [Crossref]
64. Bruno C. Vande Berg, P. Omoumi, C. Galant, N. Michoux, F. E. Lecouvet. MR Imaging of the Normal Bone Marrow and Normal Variants 21-46. [Crossref]