Bone Marrow Signal Alteration in the Spine and Sacrum

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Bone marrow signal abnormality in the spine and sacrum is a common, sometimes unexpected finding on MRI, and it can be a source of diagnostic dilemma to radiologists who interpret these examinations. The myriad causes of bone marrow signal alteration include variants of normal, marrow reconversion, tumor (myeloproliferative disorders, metastatic, or primary), radiation, fracture, degenerative change, infection, inflammatory arthritis, and osteonecrosis. A pattern-based diagnostic approach to bone marrow signal abnormalities in the axial skeleton frequently helps to generate a concise list of diagnostic possibilities, thereby potentially minimizing the need for biopsy. The three main components involved in assessing the pattern of perceived signal abnormality on MRI are its signal characteristics, distribution, and morphology. Much information can be gained by evaluating the signal characteristics of a lesion on the different pulse sequences generally used (described in the next section). The distribution of bone marrow signal abnormalities can be categorized as diffuse or infiltrative, focal, or multifocal (Table 1). In the spine, it is important to consider the distribution of the lesion, whether it is localized within the vertebral body, extends into the pedicles or posterior elements, or is confined to the endplates. The morphology of the lesion should be assessed in terms of its borders, whether it is discrete and well circumscribed, ill-defined, or aggressive and whether there is destruction of the cortical margin or merely outward bulging and expansion. This pattern-based approach must be combined with the routine diagnostic workup of bone marrow signal abnormalities seen elsewhere in the appendicular skeleton, including comparison with prior MRI (if present), complementary imaging (if needed), and clinical chemistry and bone biopsy (where appropriate).

Technique of Bone Marrow Imaging

MRI is ideal for imaging bone marrow because of its superior ability to produce high-resolution images with exquisite soft-tissue contrast. T1-weighted imaging without fat suppression is one of the most important sequences for distinguishing between normal and abnormal marrow. Normal bone marrow is composed of both fatty and hematopoietic elements. Although fatty marrow contains more fat cells than hematopoietic marrow, both types of marrow appear hyperintense relative to skeletal muscle on T1-weighted imaging because they contain a higher proportion of fat cells relative to skeletal muscle. Abnormal marrow is isointense or hypointense to skeletal muscle on T1-weighted imaging because the replacement of fatty marrow elements by the pathologic process causes loss of the normal fat signal (Fig. 1).

Both STIR and T2 fast spin-echo-weighted fat-suppression images are extremely sensitive for detecting fluid. These sequences show subtle bone marrow edema as hyperintense relative to the background of suppressed fat. STIR imaging causes loss of fat signal based on the relaxation properties of fat protons. T2-weighted imaging is often combined with frequency-selective fat saturation to detect marrow abnormality because the high signal intensity of fluid and edema becomes more conspicuous against a low-signal background of suppressed fat. Although both sequences are sensitive for the detection of marrow edema, the STIR sequence tends to produce more homogeneous fat suppression than T2 fast spin-echo because STIR is less sensitive to magnetic field inhomogeneity and magnetic susceptibility.

T2 fast spin-echo imaging without fat suppression is not primarily used for assessing bone marrow abnormality because both fat and fluid appear bright on this sequence. Thus, a pathologic marrow lesion could potentially be overlooked against the background of normal bright fatty marrow.
Bone Marrow Signal Alteration in the Spine and Sacrum

TABLE 1: Causes of Bone Marrow Signal Alteration in the Spine and Sacrum

Diffuse
- Normal variant
- Marrow reconversion or normal hematopoiesis
- Myeloproliferative disorders
  - Multiple myeloma
  - Leukemia
  - Polycythemia vera
  - Myelofibrosis

Focal
- Tumor
  - Plasmacytoma (focal form of myeloma)
  - Solitary metastasis
  - Lymphoma
  - Primary tumor
    - Chordoma
    - Giant cell tumor
    - Aneurysmal bone cyst
    - Hemangioma
- Radiation
- Fracture
- Degenerative change (endplates)
- Infection
  - Diskitis and osteomyelitis
  - Bacterial
  - Mycobacterial
- Inflammatory arthritis
  - Ankylosing spondylitis
  - Psoriatic arthritis
  - Reactive arthritis
  - Inflammatory bowel disease
  - Rheumatoid arthritis
  - Gout
- Osteonecrosis

Multifocal
- Multifocal form of myeloma
- Multiple metastases

T1-weighted imaging with frequency-selective fat suppression also can be used to confirm the presence of fat within a mass and to increase the conspicuity of enhancement after gadolinium administration.

Chemical-shift, or opposed-phase, MRI is a useful technique to distinguish neoplastic from nonneoplastic lesions. Chemical-shift imaging is based on the fact that protons in fat and water precess at different frequencies. When fat and water are present in a single voxel, their signals cancel on out-of-phase images. Benign lesions generally coexist within normal fat-containing marrow and therefore show signal dropout on out-of-phase images. On the other hand, because most neoplasms destroy and replace normal fat and hematopoietic elements, these entities maintain their signal and do not show signal dropout on opposed-phase sequences (Fig. 2).

Diffusion Imaging

Diffusion-weighted imaging (DWI) is based on the principle of mobility of water protons. The apparent diffusion coefficient (ADC) is a quantification of this motion in tissues. The
random, free motion of water protons in normal tissues results in relatively high ADC values (high signal on an ADC map). Densely packed cells within a tumor, or cytotoxic edema of cells (as in an acute cerebral infarct), restricts the motion of free water protons, thus decreasing the ADC (low signal on an ADC map).

Fig. 1—Sagittal T1-weighted images of lumbar spine in different patients.
A, Normal fatty marrow. In this 66-year-old man, marrow signal is hyperintense to that of skeletal muscle seen between and dorsal to spinous processes.
B, Normal hematopoietic marrow. In 23-year-old man, marrow signal is hypointense to fatty marrow seen in A, but it is hyperintense to skeletal muscle.
C, Myelofibrosis. In this 85-year-old woman, there is diffusely decreased signal throughout marrow secondary to marrow replacement with fibrosis and loss of fatty elements. Bone marrow is hypointense to skeletal muscle.

Fig. 2—Carcinoid metastasis (arrows) in lower thoracic vertebral body in 64-year-old man.
A, Axial T2-weighted MRI shows focal, round, hyperintense vertebral body lesion.
B, Axial in-phase T1-weighted chemical-shift image of abdomen shows isointense to hyperintense vertebral body lesion.
C, On axial out-of-phase chemical-shift image, lesion does not lose signal because of tumor replacement of marrow and lack of microscopic fat.
Bone Marrow Signal Alteration in the Spine and Sacrum

DWI has been widely used in brain MRI, particularly in the setting of acute cerebral infarct and tumor. More recently, DWI has been applied to bone marrow imaging, showing promise in differentiating benign from malignant vertebral fractures.

MRI Appearance of Normal Bone Marrow

Bone marrow is composed of three main elements: yellow, or fatty, marrow; red, or hematopoietic, marrow; and trabecular, or cancellous, bone (Table 2). Fatty marrow is composed mainly of fat cells with few hematopoietically active cells. Hematopoietic marrow produces red cells, white cells, and platelets. Trabecular bone forms the architectural framework for the fatty and hematopoietic marrow. In the spine, normal fatty marrow on T1-weighted imaging appears hyperintense relative to the disks and skeletal muscle. On fluid-sensitive sequences, normal fatty marrow in the vertebral bodies appears relatively hypo intense with respect to normally hydrated disks. Hematopoietic marrow is intermediate in signal on T1- and T2-weighted images and shows relatively lower T1 signal compared with fatty marrow. On STIR and fat-suppressed T2-weighted images, hematopoietic marrow shows intermediate signal that is more intense than fatty marrow and subcutaneous fat and similar in signal to muscle. Of note, trabecular bone appears as a low-signal reticulated structure on the background of fat on all pulse sequences.

Normal Distribution of Hematopoietic Bone Marrow

It is important to recognize the normal distribution of hematopoietic and fatty marrow, which varies as a function of age. At birth, all but the ossified epiphyses are filled with hematopoietic marrow. Conversion of hematopoietic marrow to fatty marrow in long bones follows a well-defined pattern, beginning in the distal extremities and progressing proximally. Conversion occurs first in the epiphyses and apophyses, followed by the diaphyses, distal metaphyses, and proximal metaphyses (Fig. 3). The adult pattern, in which only the axial skeleton (pelvis, vertebrae, sternum, clavicles, ribs, and skull) and proximal shafts of the femurs and humeri contain hematopoietic marrow, is completed by the age of 25 years.

Marrow Reconversion

Marrow reconversion refers to the process whereby mature fatty marrow is replaced by hematopoietic marrow when the existing marrow can no longer meet the need for hematopoi- esis. Demand for increased hematopoi- esis occurs in anemias, such as sickle cell anemia and thalassemia; marrow-stimulating medications; marrow replacement disorders; at high alti- tudes; in smokers; and in obese patients. Hyperplasia of hematopoietic marrow occurs in the reverse sequence of normal marrow maturation, commencing proximally in regions that are composed of predominantly red marrow and progressing distally to areas of fatty marrow. Therefore, hematopoietic marrow hyperplasia initially affects the axial skeleton, followed by the appendicular skeleton. The MRI appearance of marrow reconversion or hyperplasia is identical in signal to that of normal hematopoietic marrow.

Tumors

The many causes of tumor in the spine include myeloproliferative disorders, leukemia, metastases, lymphoma, and primary tumors of bone. Whereas myeloproliferative disorders are caused by an abnormal proliferation of cells that normally originate within the marrow, metastases and lymphoma cause marrow replacement with cells not normally occurring within it.

Myeloproliferative Disorders

Myeloproliferative disorders encompass a broad category of diseases caused by abnormal growth of hematopoietic marrow elements, such as plasma cells (multiple myeloma), white
A, T1-weighted coronal image of pelvis in 17-year-old patient shows normal distribution of hematopoietic marrow in iliac bones and proximal femurs. There has been normal fatty conversion in proximal femoral epiphyses and apophyses.

B, T1-weighted sagittal image of lumbar spine in 23-year-old patient shows relatively low-signal hematopoietic marrow throughout all vertebral bodies. Note that hematopoietic marrow is hyperintense or isointense with respect to muscle in both images.

cells (leukemia), red cells (polycythemia vera), and collagen and fibrous tissue (myelofibrosis). Although the MRI appearance of myeloproliferative disorders is variable, there is usually diffuse bone marrow signal abnormality that is caused either by the presence of abnormally proliferating cellular elements or by the combination of abnormally proliferating cells and marrow reconversion because of increased demand for hematopoiesis.

Multiple myeloma—Multiple myeloma is the most common primary neoplasm of bone, and up to 90% of patients with multiple myeloma develop myelomatous skeletal lesions. The incidence in the United States is about three per 100,000 people annually. The median age at diagnosis is 65 years; fewer than 10% present younger than age 40 years.

Multiple myeloma is caused by a monoclonal neoplastic proliferation of plasma cells. Because plasma cells are most abundant in hematopoietic marrow, there is a predilection for multiple myeloma to involve the axial skeleton. The spine is most frequently involved (49%), followed by the skull (35%), pelvis (34%), ribs (33%), and the humeri and femurs. Myelomatous cells cause excessive osteolysis because of secretion of such osteoclast-activating factors as interleukins and tumor necrosis factor. An imbalance of increased osteoclast activity leads to excessive bone resorption. On conventional radiographs, this process causes the classic appearance of osteopenia with punched-out lesions.

Multiple myeloma can produce a variety of MRI patterns, including focal (plasmacytoma), diffuse homogeneous, and stippled or multifocal infiltration of the marrow. A plasmacytoma is a focal proliferation of malignant plasma cells. It is thought to occur early in the disease before diffuse bone marrow involvement.

Plasmacytommas show low T1 and variable or high T2 signal with varying degrees of enhancement after gadolinium administration (Fig. 4). Plasmacytommas tend to occur more frequently in the thoracic spine but can occur anywhere in the spine or in the appendicular skeleton. These lesions are lytic and expansile, replacing marrow and initially leaving the outer cortex intact until there is cortical breakthrough later in the disease process. Sclerotic plasmacytommas have also been described.

The diffuse pattern of multiple myeloma is similar to other marrow proliferative entities, showing widespread infiltrative marrow replacement with confluent low T1 and high T2 and STIR signal. The stippled pattern of multiple myeloma is characterized by numerous punctate and rounded foci of low T1 and high T2 and STIR signal peppered throughout the marrow (Fig. 5). There can be multiple manifestations of myeloma in the same patient.
Bone Marrow Signal Alteration in the Spine and Sacrum

Fig. 4—Focal sacral plasmacytoma in 44-year-old woman who presented with left lower extremity weakness.

A and B, Coronal T1-weighted (A) and STIR (B) images of sacrum show destructive mass in left side of sacrum that is T1 hypointense and STIR hyperintense. Mass has eroded through cortex and extends into presacral space, left sacroiliac joint, and left S1 and S2 neural foramina, engulfing sacral plexus.

C, Axial contrast-enhanced T1-weighted frequency-selective fat-suppression image shows avid enhancement of mass. This case is unusual in that younger patient was affected with particularly aggressive form of disease.

Fig. 5—Stippled pattern of multiple myeloma in 62-year-old man.

A and B, Sagittal T1-weighted (A), and T2-weighted (B) images of lumbar spine show numerous punctate, T1 hypointense, and relatively T2 hyperintense lesions peppered throughout marrow. Several lumbar and lower thoracic compression fractures (arrows, A) are common manifestation of multiple myeloma. Infiltration of posterior elements is also seen (arrow, B).

Leukemia—Leukemia is caused by abnormal white cell proliferation within the marrow with subsequent replacement of normal marrow elements. There is usually a diffuse infiltrative pattern of marrow involvement with hypointense T1 signal lower than skeletal muscle and increased signal intensity on T2-weighted and STIR images because of proliferation of blast cells and likely compensatory increased hematopoiesis. As with myeloma, there may be patchy involvement of the marrow (Fig. 6). It may be difficult to distinguish leukemia from diffuse myeloma on the basis of MRI findings alone.
Long et al.

Fig. 6—Leukemia in 72-year-old woman. 
A and B, Sagittal T1-weighted (A) and T2-weighted (B) images of lumbar spine show diffuse as well as patchy heterogeneous infiltration of marrow with low T1 and isointense T2 signal. Example of diffuse marrow infiltration is seen in L2 vertebral body (arrow). 
C and D, Axial T1-weighted (C) and T2-weighted (D) images of sacrum show similar pattern of diffuse patchy marrow infiltration with focal area of spared normal fatty marrow in right iliac bone (arrowheads).

Myelofibrosis—Myelofibrosis can be caused by primary collagen fibrosis of the marrow or by reactive fibrosis after other myeloproliferative disorders. The highly structured matrix of collagen in fibrosis prevents protons from resonating and producing significant signal. Therefore, myelofibrosis appears on MRI as diffuse low signal intensity of the bone marrow on all pulse sequences (Fig. 7). This pattern is different from metastases, lymphoma, and primary tumors of the spine and sacrum, which generally produce focal or multifocal signal abnormalities.

Fig. 7—Myelofibrosis in 85-year-old woman. 
A and B, Sagittal T1-weighted (A) and T2-weighted (B) images of thoracic and lumbar spine show diffusely low signal (compared with muscle) due to marrow fibrosis. 
C, Coronal localizer image shows diffusely low signal of spine and sacral marrow along with splenomegaly, which often develops in patients with myelofibrosis due to extramedullary hematopoiesis.
Bone Marrow Signal Alteration in the Spine and Sacrum

Metastases

Metastases are the most common malignant tumors affecting the skeleton. They frequently involve bones containing hematopoietic marrow, such as the vertebra, pelvis, proximal femurs, and ribs. Metastases in the axial skeleton can be solitary or multiple. When multiple, they typically appear as focal discrete lesions, rather than as the diffusely infiltrative pattern seen with the myeloproliferative disorders. Most often centered in the medullary canal, metastases are destructive and can be expansile. They usually show T1 hypointensity and T2 and STIR hyperintensity. In the spine, pedicle involvement is common. Sclerotic metastases may show low signal intensity on all sequences (Fig. 8), but there may be associated surrounding T2 and STIR hyperintense edema, giving lesions a target-like appearance.

Lymphoma

Lymphoma accounts for 7% of bone malignancies, with 95% occurring secondary to the deposition of cells from an extraskeletal site. Lymphomatous lesions can be focal or multiple and have a distribution and morphology similar to metastatic carcinoma (Fig. 9). On MRI, lymphoma is T1 hypointense relative to skeletal muscle. The tumor has variable T2 intensity, at times appearing hypointense because of its highly cellular nature. Lymphoma typically does not cause cortical destruction, instead permeating through the cortex and into surrounding soft tissues. Widespread lymphoma occasionally can cause homogeneous and diffuse marrow signal abnormality.

Primary Tumors of the Spine and Sacrum

Primary tumors of bone are rare, accounting for only 0.2% of all neoplasms. They are usually solitary, unlike metastatic disease, myeloma, and lymphoma, which are generally multifocal.

Chordoma—Chordoma accounts for 1–4% of all primary malignant bone tumors. Discounting lymphoproliferative disorders, chordoma is the most common primary malignant tumor of the spine and sacrum in adults. It develops from notochordal remnants and typically arises in the midline. About 60% are sacral, 25% sphenoccipital, 10% cervical, and 5% thoracolumbar. Chordoma is a lobulated tumor composed of cells rich in mucin and glycogen (physaliphorous cells). Although slow-growing and with little metastatic potential, chordoma is locally invasive.

On MRI, a chordoma classically appears as a destructive, lobulated mass that is isointense or hypointense on T1-weighted imaging and hyperintense on T2 fast spin-echo and STIR images (due to its high mucin content). Chordomas show heterogeneous contrast enhancement (Fig. 10).
A, Axial T2-weighted MR image shows multilobulated mass (arrow) arising from coccyx and extending into presacral region, causing mild anterior displacement of rectum. Mass contains low signal internal septations.

B, Sagittal T2-weighted MR image shows similar findings, with mass arising from coccygeal segments (arrow).

C, Axial contrast-enhanced T1-weighted frequency-selective fat-suppression MR image shows enhancement of internal septations within mass (arrow).

Giant cell tumor—Giant cell tumor (GCT) is a benign, locally aggressive tumor that represents about 4–5% of all primary bone tumors. GCT typically occurs in the epiphyses of long bones in patients 20–40 years old. About 5% affect the flat bones, most commonly the sacrum. Less common sites in the axial skeleton (in decreasing order of frequency) are the
Bone Marrow Signal Alteration in the Spine and Sacrum

Fig. 11—Giant cell tumor of clivus. (Courtesy of G. Moonis, Boston, MA)
A, Sagittal T1-weighted MR image shows large expansile lesion that arises from clivus and extends anteriorly. Lesion is largely isointense to bone but hyperintense to muscle.
B and C, Axial (B) and coronal (C) T1-weighted contrast-enhanced images of skull base show clival mass of intermediate signal intensity. Large, destructive, avidly enhancing lesion does not contain mineralized internal matrix.

thoracic, cervical, and lumbar spine. Spinal GCT most commonly involves the vertebral body, with the posterior elements frequently affected in advanced lesions.

On MRI, a giant cell tumor usually appears as an expansile, osteolytic lesion that does not contain a mineralized matrix (Fig. 11). In the sacrum, the tumor is usually large and associated with destruction of the sacral foramen. GCT typically shows hypointense to intermediate signal intensity on T1-weighted imaging and hypointense signal intensity on T2-weighted imaging (similar to the spinal cord). The hypointense T2 signal, which appears to be caused by collagen or hemosiderin content, is helpful in narrowing the differential diagnosis because most other spinal neoplasms (metastases, myeloma, lymphoma, and chordoma) show high signal intensity on long-TR images. Cystic areas are commonly seen in GCT.

Aneurysmal bone cyst—An aneurysmal bone cyst (ABC) is an expansile lesion containing blood-filled spaces separated by connective tissue septa. About 80% of ABCs occur by age 20, and many are thought to be due to previous trauma. Although usually primary, in about 30% of cases they complicate other benign or malignant tumors, such as GCT, chondroblastoma, or osteoblastoma. In the spine, ABC involves the posterior elements.

On conventional radiographs, ABCs are radiolucent lesions in the posterior elements with expansion and marked thinning of the cortex. On MRI (Fig. 12), they are typically hyperintense on T2-weighted imaging and isointense or hypointense on T1-weighted imaging. Fluid–fluid levels may occur, but they are not pathognomonic of ABC (also seen in telangiectatic osteosarcoma).

Hemangioma—Hemangiomas are slow-growing, benign neoplasms that are commonly found in vertebral bodies. They consist of thin-walled, blood-filled vessels interspersed among
the bone trabeculae and contain a variable amount of fat. Typical hemangiomas are round lesions in the vertebral body with high signal on T1- and T2-weighted images and low signal on STIR images because of the presence of fat within the lesion (Fig. 13). They may have a stippled or speckled appearance on axial MRI, similar to the polka-dot appearance described on CT because of preserved low-signal-intensity trabeculae coursing through the mass.

Atypical hemangiomas that contain only a small or microscopic amount of fat are often difficult to distinguish from malignant lesions on conventional T1- and T2-weighted images. These atypical hemangiomas may be characterized with chemical-shift imaging because they lose signal intensity on out-of-phase images due to the presence of microscopic fat (Fig. 14).

**Radiation**

Myelomatous lesions, metastases, and other tumors are often treated with radiation for palliation or in conjunction with chemotherapy or surgery. In addition, the axial skeleton may be included in the radiation field for targeted therapy of extraskeletal disease (e.g., pelvic soft-tissue malignancy). Hematopoietic marrow elements are preferentially destroyed by radiation. By 6 weeks after therapy, the bone marrow in a vertebral body subjected to radiation...
Bone Marrow Signal Alteration in the Spine and Sacrum

Fig. 13—Hemangioma in 50-year-old woman. 
A and B, Sagittal T1-weighted (A) and T2-weighted (B) MR images of lumbar spine show well-defined, round, nondestructive lesion in L3 vertebral body that is hyperintense on both sequences due to presence of fat. 
C, Axial T2-weighted image best shows stippled appearance within hemangioma.

undergoes conversion to fatty marrow, showing diffusely high T1 signal. A characteristic finding of radiation change is a segmental region of fatty marrow that involves contiguous vertebrae and has abrupt margins at the edges of the radiation portal (Fig. 15).

Fracture

The MRI characteristics of fracture in the axial skeleton vary depending on the acuity of the process (acute or chronic) and the underlying cause (pathologic, osteoporotic, or traumatic). In
general, fractures in the axial skeleton cause focal or multifocal bone marrow signal abnormality with associated vertebral body deformity.

Acute vertebral compression fractures are common and a frequent cause of referral for MRI. They may occur secondary to neoplastic infiltration, osteoporosis, or trauma. MRI may be critical in the often-challenging determination of whether a vertebral compression fracture has a malignant or benign cause. The degree and pattern of bone marrow replacement, contour of the affected vertebral body, number of lesions, and infiltration of the posterior elements are all important features useful in assessing the cause of a fracture.

Pathologic Fractures
Pathologic fractures (Fig. 16) are suspected when there are multiple lesions, expansion of the vertebral body with convex anterior and posterior cortical margins, involvement of the pedicles, and an associated soft-tissue mass. Vertebral bodies containing pathologic fractures often show an expanded marrow cavity with concomitant bone marrow edema that produces abnormal signal throughout the entire vertebral body (diffuse low signal on T1-weighted images, diffuse high signal on T2-weighted and STIR images). It is thought that vertebral body metastases do not result in fracture until the majority of the marrow is infiltrated with tumor. The linear, low-signal-intensity fracture line paralleling the vertebral body endplates because of compressed trabeculae, which is seen in benign compression fractures, is not usually present in pathologic fractures because the osseous trabeculae have been destroyed by tumor.

Acute Benign Compression Fractures
Benign compression fractures usually involve the anterior column of the spine and are characterized by an anterior wedge deformity. The posterior concave margin of the vertebral body is usually preserved. In some cases, however, the middle column of the spine may be involved and retropulsion of fracture fragments into the spinal canal can occur. There is almost always a discernible fracture line, composed of compressed trabeculae, which appears on MRI as a linear, horizontally oriented, low-signal abnormality on both T1 and fluid-sensitive imaging and is surrounded by areas of bone marrow edema (Fig. 17). The fracture line is usually considered as a sign of a benign process because this finding is not seen in pathologic fractures, presumably due to destruction of bone trabeculae.

Chronic Compression Fractures
Chronic benign compression fractures show vertebral body deformity, which may be any combination of anterior wedging, loss of vertebral body height, and concavity of the endplates. Compared with an acute fracture, in a chronic fracture the marrow edema has resolved, making the marrow signal isointense to normal vertebrae on all sequences (Fig. 18). The fracture line has usually healed and is often difficult to see.
Bone Marrow Signal Alteration in the Spine and Sacrum

Burst Fractures

Burst fractures are most frequently associated with trauma but can occasionally be seen with osteoporotic compression fractures (Fig. 19). Burst fractures are considered unstable because the anterior and middle columns, and occasionally the posterior column, are disrupted. The morphology of the posterior vertebral body line distinguishes between burst and anterior wedge fractures. Loss of the normal posterior vertebral body concavity and posterior

Fig. 16—Renal cell carcinoma metastases with acute pathologic T12 fracture in 63-year-old man. A–D, Sagittal T1-weighted (A), sagittal contrast-enhanced T1-weighted with frequency-selective fat suppression (B), sagittal T2-weighted (C), and parasagittal T2-weighted (D) MR images of lumbar spine show multiple enhancing vertebral body metastases. Round, expansile metastases in T11, L1, and L2 vertebral bodies (large arrows, A) cause convexity of posterior margins and show enhancement (arrows, B). There is pathologic fracture of T12 vertebral body with loss of vertebral body height and retropulsion of fragments into canal (small arrows, A and C). No fracture line is seen in T12 vertebral body, feature that is typical of pathologic fracture. A lesion of pedicle of L1 also is seen (arrow, D).
Long et al.

Fig. 17—Acute T11 compression fracture in 68-year-old woman. (Courtesy of A. Sarwar, Boston, MA) A–C, Sagittal T1-weighted (A), T2-weighted (B), and STIR (C) MR images of lower thoracic spine show compression deformity of T11 vertebral body with preservation of concave posterior margin (arrow, A). T11 vertebral body is T1 hypointense and T2 isointense and STIR hyperintense due to bone marrow edema from acute fracture. Horizontally oriented hypointense fracture line (arrowhead, B and C) caused by compressed trabeculae is surrounded by bone marrow edema that is hyperintense on STIR (C).

Bowling of the posterior vertebral body line are indicative of burst fracture; retropulsion of fracture fragments into the spinal canal also commonly occurs. Similar to other acute fractures, vertebral body bone marrow edema from a burst fracture manifests as T1 hypointensity and T2 and STIR hyperintensity.

Sacral Insufficiency Fracture

The sacrum, sacroiliac joints, and medial aspect of the iliac bones are the major weight-bearing structures in the body. Sacral insufficiency fractures are often bilateral and may course vertically or obliquely in the sacral alae. Insufficiency fractures of the sacrum may be associated with postmenopausal or corticosteroid-induced osteoporosis and radiation therapy. The radiographic findings are usually subtle. Sacral fractures may not be clinically suspected, or the history may be confusing. Patients with this condition may undergo MRI of the lumbar spine, with bone marrow signal changes in the sacrum being overlooked. Consequently, it is important to specifically look for sacral fractures on every lumbar spine MRI examination.
Bone marrow edema because of a fracture appears as an area of low signal intensity on T1-weighted imaging and high signal intensity on T2-weighted and STIR imaging. A low-signal-intensity fracture line may be seen (Fig. 20). If the fracture line cannot be detected, however, the bone marrow edema pattern may be misinterpreted as representing metastatic disease or primary malignancy.

**Degenerative Changes**

A common and important cause of focal or multifocal bone marrow signal abnormality in the spine is degenerative disk disease. In this condition, the vertebral body signal abnormality is typically centered around the intervertebral disks at the endplates, an area that may be involved in a variety of pathologic conditions. Modic and colleagues described a spectrum of abnormalities related to degenerative disk disease, characterized by a bandlike pattern of signal change within vertebral endplates with associated signal changes of the adjacent intervertebral disk (Fig. 21).

In type 1 Modic change, there is decreased signal intensity in the vertebral body endplates on T1-weighted imaging and increased signal intensity on T2-weighted imaging. This represents a more acute or edematous reparative response of the vertebral body to disk degeneration. Vertebral body signal alteration in type 1 Modic change may occasionally be confused with signal changes of osteomyelitis. In Modic type 1 changes, however, the intervertebral disk is usually desiccated and shows low T2 signal, unlike the increased fluid and high T2 signal of the disk seen in the presence of infection. In addition, the endplates are intact in degenerative disease, whereas infection causes erosive destruction of the endplates.
Long et al.

A

B

C

Fig. 20—Sacral insufficiency fracture in 82-year-old woman. A and B, Axial T1-weighted (A) and T2-weighted (B) images of sacrum show vertically oriented, low-signal-intensity fracture line through left sacral ala (arrows) with surrounding T1-hypointense and T2-hyperintense bone marrow edema. Low-signal-intensity fracture line is more conspicuous on T2-weighted image (B) because it is highlighted by surrounding T2 hyperintense bone marrow edema.

With progressive degenerative change (Modic type 2), conversion to fatty marrow causes the vertebral endplates to develop increased signal intensity on both T1- and T2-weighted images. There is concomitant suppression of fat signal on STIR images. In chronic degenerative change (Modic type 3), the endplates have decreased signal intensity on all sequences because of sclerosis.

The vacuum phenomenon is the radiographic appearance of liberated nitrogen gas in an intervertebral disk because of negative pressure generated by loss of disk fluid. Vacuum disks have low signal intensity on all MRI sequences but are usually more easily seen on radiographs or CT scans. The presence of gas in a disk indicates that it is not distended with fluid, implying that any adjacent changes in the vertebral bodies and disk are likely not related to infection. The appearance of gas within an intervertebral disk is rare in vertebral diskitis or
Bone Marrow Signal Alteration in the Spine and Sacrum

Infection

Similar to degenerative disease, infection of the spine and sacrum typically appears on MRI as focal or multifocal marrow signal abnormalities that are most often centered around an intervertebral disk or the sacroiliac joints. Infection due to typical bacterial pathogens can have a distinctive MRI appearance that is different from disease caused by mycobacteria. In addition, infection can be mimicked on MRI by degenerative disk disease, inflammatory spondyloarthropathy, and neuropathic arthropathy.

Spine Infection

Pyogenic spondylitis is most often caused by Staphylococcus aureus, which has a predilection for the lumbar region. In most cases, two adjacent vertebral bodies and the intervening disk are infected. MRI is the imaging technique of choice to evaluate possible spinal infection because it can show both vertebral body and disk abnormality earlier and more clearly than CT. Imaging patterns indicative of infection include the following:

- Decreased vertebral body marrow signal on T1-weighted images
- Increased vertebral body signal on T2-weighted and STIR images
- Erosion of vertebral endplates
- Disk hyperintensity on T2-weighted images
- Disk enhancement
- Decreased intervertebral disk height
- Paraspinal inflammatory tissue, soft-tissue mass, or fluid collection

Decreased signal in the bone marrow of involved vertebral bodies on T1-weighted imaging and increased signal on T2-weighted imaging reflect the presence of extracellular fluid in the marrow because of an inflammatory reaction (Fig. 22). All characteristic findings may not be observed, especially loss of disk height.

osteomyelitis (only in infections due to gas-forming organisms), and its presence almost always excludes infection as a cause of endplate marrow signal abnormality.

Fig. 21—Modic degenerative endplate changes in lumbar spine in 60-year-old woman. A–C, Sagittal T1-weighted (A), T2-weighted (B), and STIR (C) MR images of lumbar spine show fatty (Modic type 2) degenerative change in endplates of L4 and L5, which appear hyperintense on T1- and T2-weighted images and hypointense on STIR sequence (white arrows). Modic type 1 degenerative changes, consistent with bone marrow edema, at L3 and L4 appear hypointense on T1-weighted image and hyperintense on T2-weighted and STIR sequences (best seen on STIR image; black arrows, C). Modic type 1 changes can be similar to those seen in diskitis and osteomyelitis, but in degenerative disease intervertebral disk is dehydrated and has low signal intensity on T2-weighted image (arrowhead, B) and cortices of endplates are intact.
Mycobacterial infection of the spine may produce MRI findings (Fig. 23) that are different from bacterial discitis or osteomyelitis. Unlike pyogenic infections, which characteristically involve the intervertebral disk, spinal tuberculosis is thought to originate in the vertebral body and spread beneath the longitudinal ligaments to involve adjacent vertebral bodies. The neighboring disks are preserved, possibly because *Mycobacterium tuberculosis* does not produce proteolytic enzymes. In addition to disk preservation, other characteristics of tuberculosis include skip lesions and involvement of multiple vertebral bodies or only a portion of a vertebral body (such as the posterior elements). In some cases, these features can make it difficult to differentiate tuberculous infection from such neoplasms as lymphoma or metastatic disease, often delaying the diagnosis and the institution of appropriate treatment.

Conditions Mimicking Spinal Infection

Conditions that may mimic spinal infection include degenerative disk disease and degenerative endplate changes, inflammatory spondyloarthopathy, neuropathic arthropathy, and an occasional spinal neoplasm. In Modic type 1 degenerative change, there is decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted sequences in the endplates of adjacent vertebral bodies that correspond to edema. However, these findings can be distinguished from diskitis or osteomyelitis by focusing on the appearance of the intervertebral disk. On T2-weighted images, the disk shows abnormally high signal intensity in infection and loss of signal in degenerative disk disease.
Bone Marrow Signal Alteration in the Spine and Sacrum

Fig. 23—Mycobacterial infection of cervical spine in 52-year-old man.
A and B, Sagittal T1-weighted (A) and STIR (B) MR images show areas of T1 hypointensity and STIR hyperintensity reflecting bone marrow edema in C5 and C6 vertebral bodies (large arrows). Note preservation of vertebral body endplates and intervertebral disk as well as extensive spread of infection beneath anterior longitudinal ligament (thin arrow, B).
C, Sagittal contrast-enhanced T1-weighted image shows enhancement of abnormal vertebral bodies and prevertebral soft tissues.

Fig. 24—Left sacroiliac joint infection.
A and B, Axial T1-weighted (A) and T2-weighted (B) MR images show unilateral T1 hypointensity and T2 hyperintensity on both sides of left sacroiliac joint, representing bone marrow edema. Joint is widened and contains high signal intensity (black arrow, B). Periosteal edema extends along lateral aspect of left iliac bone into gluteal muscles (white arrow, B).
C, Coronal contrast-enhanced T1-weighted image shows enhancement of left ilium, sacrum, and left iliacus muscle (arrow).

Spinal neuropathic arthropathy, most often related to diabetes, is an uncommon destructive process involving the vertebral bodies and disks. The clinical manifestations and imaging characteristics of this condition may simulate infection of the spine. These include endplate erosion, loss of disk height, paraspinal soft-tissue mass, and signal characteristics similar to spinal infection on both T1- and T2-weighted images. The diagnosis of neuropathy can be made if there are vacuum disks and the Ds characteristics of a neuropathic joint—disorganization, debris, and dislocation.
Sacral Iliac Joint Infection

Pyogenic sacroiliitis represents 1–2% of all cases of septic arthritis and is most often caused by *S. aureus*. Imaging findings on MRI include sacroiliac joint effusion and synovial outpouching, surrounding reactive bone marrow edema and enhancement in both the sacrum and iliac bones, loss of the normal low-signal-intensity margins of cortical bone, and rim-enhancing abscess formation in the adjacent iliopsoas muscle or paraspinal soft tissues (Fig. 24). If sacroiliac disease is unilateral, infection must always be considered in the differential diagnosis.

Inflammatory Arthritis

Seronegative spondyloarthropathies are closely related inflammatory diseases that affect the axial skeleton, particularly the sacroiliac joints. These entities include ankylosing spondylitis, psoriatic arthritis, reactive arthritis, and arthritis associated with inflammatory bowel disease. Involvement of the sacroiliac joints can also be seen in gout, rheumatoid arthritis, in the postpartum period (Fig. 25), and with infection (as discussed previously).

MRI is superior to radiography in depicting inflammation of the sacroiliac joints, and gadolinium-enhanced scans are useful for detecting active disease. On T1-weighted images, the normal sacroiliac joint appears as a thin zone of intermediate-signal cartilage with an adjacent low-signal cortex. The articular cartilage of the sacrum is slightly thicker than the iliac cartilage. This may explain why erosions appear to be more prominent on the iliac side of the joint, where the cartilage is thinner and the subchondral bone is less well protected. The dorso-caudal synovial portions of the sacroiliac joints are most frequently involved in the early stages of spondyloarthritis.

Contrast-enhanced T1-weighted images are more sensitive than STIR and fat-saturated T2-weighted images for detecting the presence and extent of acute inflammatory changes in the sacroiliac joints. Erosions appear as contrast-enhancing areas contiguous with the sacroiliac joint. If erosions are large enough, they may appear as areas of hypointensity on unenhanced T1-weighted images.

MRI findings of sacroiliitis include the following:
- Loss of the normal thin band of cartilage on T1-weighted images
- Increased synovial signal on T2-weighted images
- Subchondral bone marrow edema
- Erosions
- Synovial and subchondral bone marrow enhancement

Fig. 25—Postpartum sacroiliitis in 23-year-old woman. 
A and B, Axial T1-weighted (A) and T2-weighted (B) MR images of sacrum show areas of T1 hypointensity and T2 hyperintensity in right iliac bone and sacrum, reflecting subchondral bone marrow edema. Erosions of right sacroiliac joint are seen (arrows, A), and there is loss of normal low-signal-intensity cortices (arrow, B).
C, Coronal STIR image shows subchondral bone marrow edema that is more pronounced on iliac side of sacroiliac joint.
Bone Marrow Signal Alteration in the Spine and Sacrum

Fig. 26—Psoriatic sacroiliitis in 44-year-old woman. A and B, Coronal T1-weighted (A) and STIR (B) MR images show symmetric T1 hypointense and STIR hyperintense subchondral bone marrow edema involving both sacroiliac joints (arrows). Changes are slightly greater on right and more extensive on iliac side of joints. C, Axial contrast-enhanced T1-weighted with frequency-selective fat-suppression MR image shows abnormal sacroiliac joint enhancement that is greater on right (arrows).

Involvement of the sacroiliac joints is the hallmark of ankylosing spondylitis and other sernegative spondyloarthropathies, such as psoriatic arthritis (Fig. 26). In addition to typical clinical features, the diagnosis of ankylosing spondylitis requires radiographic evidence of sacroiliitis, which is defined as erosions, subchondral sclerosis, and irregular joint spaces. Similar changes can be seen in patients with inflammatory bowel disease (e.g., Crohn’s disease). Radiographs are most frequently used to screen for sacroiliitis, but they may show normal findings in early stages of the disease and not show any abnormality for several years.

Osteonecrosis

Osteonecrosis is relatively uncommon in the spine and is most frequently caused by a compression fracture that has failed to unite. The pathognomonic finding on radiographs is an intravertebral vacuum cleft causing a linear lucency in a partially collapsed vertebral body, either centrally or at the endplate. Delayed posttraumatic osteonecrosis of a vertebral body is termed “Kummell’s disease.” On MRI, the vacuum cleft or intervertebral gas is seen as a low-signal-intensity linear signal abnormality on all pulse sequences on a background of bone marrow edema within a partially collapsed vertebral body. There also may be accompanying intervertebral fluid that shows low T1 and high T2 and STIR signal. Correlation of the appearance of osteonecrosis on MRI with the findings of a vacuum cleft on the accompanying radiographs or CT confirms the diagnosis of osteonecrosis, thus minimizing potential confusion with insufficiency or pathologic fracture. In the sacrum, avascular necrosis frequently presents the classic appearance of serpentine linear signal abnormality on T1-weighted imaging and alternating serpentine T2 and STIR hypointense and hyperintense linear signal abnormality seen on the background of bone marrow edema.

Summary

MRI is the most useful technique for assessing bone marrow signal abnormality. A pattern-based approach involving assessment of signal characteristics, distribution, and morphology of bone marrow signal abnormality together with clinical information can help to narrow the list of differential considerations.
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