MRI of Arthritis

Ashley G. Grindol¹, Edward Derrick, Kurt F. Scherer, Christopher Wasyliw, Laura W. Bancroft

The shoulders, hips, spine, and sacroiliac joints are often included in the imaging fields of chest, abdominal, and pelvic MRI examinations interpreted by body imagers. Therefore, body imagers should be familiar with the MRI features of the common arthritides and be comfortable proposing a limited differential diagnosis when encountered in the axial joints and spine. Although several arthritides have relatively specific characteristics, others have nonspecific, overlapping imaging features. In this chapter, the imaging findings of osteoarthritis (OA), inflammatory arthritides (rheumatoid arthritis [RA] and juvenile idiopathic arthritis [JIA]), seronegative spondyloarthropathies (axial spondyloarthropathies, ankylosing spondylitis [AS], enteropathic arthropathies, and psoriatic arthritis), septic arthritis, crystal-deposition and other deposition-induced arthritides, and synovium-based processes will be discussed.

Osteoarthritis

OA, or osteoarthritis, is the most prevalent arthritis and is the most common chronic musculoskeletal cause of morbidity and disability in the elderly population [1]. It is characterized by articular cartilage wear and joint failure secondary to dysfunctional compensatory changes. Although conventional radiography is commonly used as the initial assessment for OA to assess for joint space narrowing and osteophytes, MRI is better able to depict the osseous and soft-tissue structures of the joint, including the articular cartilage and bone marrow. Common findings of OA on MRI include articular cartilage loss, osteophyte formation, bone marrow edema (BME), synovitis, joint effusions, and meniscal or acetabular labral and glenoid labral abnormalities [1] (Fig. 1). MRI also allows evaluation of surrounding soft-tissue components such as the joint capsule, extraarticular ligaments, tendons, and muscle.

Several grading systems have been developed to assess the severity of OA using arthroscopic or imaging criteria. One of the more commonly used classification systems for the assessment of articular cartilage damage is the system proposed by the International Cartilage Repair Society [2]. This grading system can be applied when interpreting MRI: Grade 0 is assigned for normal, grade 1 for superficial cartilage cracks and fissures, grade 2 for lesions involving less than 50% of the cartilage depth, grade 3 for lesions involving greater than 50% of the cartilage depth but not extending to bone, and grade 4 for cartilage lesions extending to the subchondral bone [2]. BME can occur subjacent to areas of articular cartilage loss and is hypointense on T1-weighted images and hyperintense on T2-weighted sequences because of the increased water content of bone marrow. Intraarticular osteochondral bodies may also occur with more advanced OA, sometimes because of fractured osteophytes. Intraarticular bodies, subchondral BME, and cysts in conjunction with osteophytes and cartilage destruction are nearly pathognomonic for OA and are frequently encountered in the elderly population.

The spine is another common location for degenerative change, and Modic and colleagues [3] described a spectrum of degenerative disk disease (DDD) characterized by bandlike signal-intensity abnormalities in the vertebral body endplates and associated changes in the adjacent intervertebral disks. Type 1 Modic changes reflect acute reaction of the vertebral endplates to disk degeneration, with T1-hypointense and T2-hyperintense endplate signal changes [3, 4] (Fig. 1). Occasionally, type 1 Modic changes can be potentially confused with osteomyelitis; however, there are important imaging features that can be used to differentiate between the two causes. The intervertebral disk is usually T2-hypointense due to disk desiccation in DDD unlike the T2-hyperintense disk signal seen in infectious discitis [4]. Erosive endplate changes develop with osteomyelitis, whereas no endplate destruction is seen in Modic changes [4]. Type 2 Modic changes occur with progression of DDD, which is characterized by fatty marrow at the endplates causing hypointense signal on T1- and T2-weighted images [3]. In type 3 Modic change, there is hypointense signal on all sequences secondary to endplate sclerosis [3]. Vacuum phenomenon of the intervertebral disks is another common finding associated with degenerative change and occurs when nitrogen gas enters the disks because of negative pressure generated by loss of disk fluid [4]. The vacuum phenomenon is characterized by hypointense signal on all MRI sequences. Unlike gas in other soft tissues, gas is rare in infectious discitis and its presence is almost always seen in DDD and effectively excludes infection [4].

Inflammatory Arthritides

RA is a chronic autoimmune disorder that most commonly affects women older than 65 years old [5, 6]. It is characterized mainly by systemic inflammation, autoantibodies, and persistent polyarticular synovitis [5]. Early diagnosis and treatment of RA are paramount to the prevention of disease progression, and early treat-
ment has been associated with improved clinical outcomes. MRI is not only beneficial in aiding in the diagnosis of early RA inflammation, but also helpful in tracking treatment response and verifying disease remission [7]. Similar to other inflammatory arthritides, the earliest imaging finding in RA is often synovitis. Synovitis manifests as increased synovial thickness, synovial volume, and synovial enhancement on contrast-enhanced sequences [8]. Synovial volume correlates with joint swelling and tenderness and is predictive of clinical disease activity [9]. The rate and magnitude of synovial enhancement have been shown to be related to the severity of inflammation [10]. Rice bodies are small intraarticular loose bodies the size of rice that may develop in patients with RA. Rice bodies are thought to be dissociated, infarcted synovium and will be low signal intensity on T1-weighted, T2-weighted, and intermediate-weighted sequences (Fig. 2). BME is another common finding in RA that occurs in response to synovitis. BME is seen on fluid-sensitive sequences as ill-defined areas of hyperintense signal and will have corresponding ill-defined hypointense signal on T1-weighted images [11, 12].

The natural progression of RA after early synovitis and simple synovial proliferation is pannus formation [5]. A pannus is essentially hypervascular, hypertrophied synovium and displays intermediate to hypointense signal on T1-weighted and T2-weighted sequences (Fig. 2). As the disease progresses, periarticular bone demineralization, cartilage destruction, and eventually subchondral bone erosions will follow [5]. Erosions appear as sharply demarcated juxtaarticular bone lesions and will be visible in at least two planes [11] (Fig. 2).

JIA shares many imaging findings with RA and is the leading cause of childhood rheumatic disease [13]. JIA occurs in children younger than 16 years old and persists for more than 6 weeks without an underlying identifiable cause [14]. JIA is characterized by chronic inflammation and synovitis, which can lead to devastating structural damage and growth abnormalities. In 55–75% of patients, JIA presents as monoarticular or oligoarticular arthritis [15]. The most commonly involved joint at onset is the knee.
followed by the hands and wrist joints, hips, ankles, and tarsal joints [15]. Hip involvement is a major source of morbidity, with total hip replacement needed in 26–44% of children within the first 10 years from onset [16]. MRI assessment of a patient with active JIA will yield findings similar to those seen in a patient with RA, such as synovitis, increased synovial volume, and pannus formation with enhancement. Chronic fibrotic disease is characterized by a lack of pannus enhancement, subchondral bone changes, erosions, and eventual deformations of joint surfaces [16, 17]. In the spine, these areas of inflammation can lead to spondylodiscitis and other complications such as spinal stenosis and subluxations. Ankylosis resulting from chronic JIA is more common than in adult RA and can cause loss of motion and significant growth disturbances [16] (Fig. 3).

**Spondyloarthropathies**

The seronegative spondyloarthropathies are a group of disorders with common clinical, laboratory, and genetic features, with the most important being human leukocyte antigen (HLA)-B27 positivity [18].
The most common spondyloarthropathies include AS (previously known as ankylosing spondylitis), psoriatic arthritis, reactive arthritis, and enteropathic arthritis. The Assessment of Spondyloarthritis International Society characterizes disease on the basis of sacroiliitis detected on imaging, HLA-B27 positivity, and the presence of defining clinical features (including, but not limited to, inflammatory back pain, arthritis, enthesitis, psoriasis, Crohn disease, and a positive family history of spondyloarthritis) [18].

**Fig. 5**—Classic imaging features of axial spondylitis (AS).
A, Sagittal T2-weighted image of 42-year-old man with AS shows multiple edematous foci of endplate corner enthesis (arrowheads) and anterior Romanus lesions.
B, Sagittal T1-weighted image of thoracic spine in 62-year-old man with advanced AS shows bamboo spine with thick ossification of anterior longitudinal ligament (arrowheads), partial ankylosis across posterior disk space (arrow), and heterogeneously high signal intensity in remaining disks.
C, Sagittal T2-weighted image of 35-year-old man with AS shows extensive reactive edema (asterisks) and early Andersson lesion (arrowheads) caused by noninfectious spondylodiscitis.
D and E, Sagittal T1-weighted image (D) and 2D reconstruction from CT (E) of paraplegic 62-year-old man with advanced AS shows pathologic fracture (arrows) through mid thoracic spine.
MRI of Arthritis

AS is the prototypical spondyloarthropathy, is more common in men than women, and usually affects the axial skeleton [18]. AS is characterized by inflammatory lesions of the entheses or the junctional areas between the bones and the tendons, ligaments, fascia, or capsules. Enthesitis is thought to be responsible for clinical symptoms such as inflammatory back pain and morning stiffness [18]. The earliest presenting symptoms of patients with AS are inflammatory back pain with alternating gluteal pain secondary to sacroiliitis [19]. Sacroiliitis is well evaluated on fluid-sensitive sequences, such as T2-weighted fat-suppressed sequences and STIR sequences. Findings suggestive of active sacroiliitis include loss of the normal thin band of cartilage on T1-weighted images, T2-hyperintense synovial signal, subchondral BME, erosions, and synovial and subchondral bone marrow enhancement [19].

Fig. 6—Septic arthritis.
A, Coronal T2-weighted image of right hip in 45-year-old man with late-stage septic arthritis shows femoral and acetabular erosions (asterisks), synovitis, and extensive periarticular inflammatory change (arrows).
B and C, Axial T1-weighted (B) and T2-weighted fat-suppressed (C) images of 44-year-old man with right sacroiliac joint septic arthritis shows periarticular T1-hypointense marrow signal (asterisks, B); mildly distended sacroiliac joint (arrow, C); and inflammatory changes involving iliopsoas (asterisk, C) and gluteal (arrowheads, C) muscles.

Fig. 7—24-year-old woman with pigmented villonodular synovitis of left hip.
A and B, Coronal intermediate-weighted fat-suppressed (A) and sagittal T2-weighted (B) images of left hip show extensive low-signal-intensity synovial proliferation (asterisks), joint effusion, and acetabular erosions (arrowheads). In B, A = anterior, P = posterior.
Sacroilitis is most often unilateral and can occur after gastric bypass surgery. Most cases of pyogenic spondylitis involve two adjacent vertebral bodies and the intervening disk and have the following MRI characteristics: T1-hypointense and T2-hyperintense vertebral body marrow, erosion of vertebral endplates, disk T2 hyperintensity and enhancement, paraspinal inflammatory tissue, and a soft-tissue mass or fluid collection [4]. When a *Mycobacterium* species is the responsible organism, the infection may be more indolent and will differ in its progression as it spreads beneath the spinal longitudinal ligaments [4]. This mechanism of dissemination allows the involvement of adjacent vertebral bodies and sparing of the intervertebral disks, often resulting in anterior gouge defects. *Mycobacterium* infectious spondylitis can also present with skip lesions and may involve only a portion of the vertebral body, potentially mimicking lymphoma or metastatic disease [4].

Pyogenic sacroilitis is most often unilateral, and *S. aureus* is the most common inoculated pathogen [4]. MRI characteristics include sacroiliac joint effusion, synovial outpouching, surrounding reactive BME with enhancement of the sacrum and ilium,
loss of normal cortical margins, and rim-enhancing abscess formation in the iliopsoas or paraspinal soft tissues (Fig. 6) [4].

Crystal and Deposition-Induced Arthritides
Gout is the most common crystalline arthropathy and the most common inflammatory arthropathy in men. The hallmark of gout is hyperuricemia with deposition of monosodium urate crystals in joints, tendons, and soft tissues, which leads to inflammation and symptomatic disease. There is a strong association of gout with metabolic syndrome, myocardial infarction, diabetes mellitus, and premature death [28]. Gout is generally polyarticular and asymmetric in distribution, with an affinity for the lower extremities (particularly the first metatarsophalangeal joint); involvement of the spine is rare. Early gout has imaging features similar to other inflammatory arthropathies, such as synovitis and joint effusions. Marginal erosions with a so-called “overhanging edge” and BME can develop; gouty tophi (nodular masses of accumulated monosodium urate crystals) can form along tendons, ligaments, cartilage, and other soft tissues as well as within bones. Tophi typically have intermediate to hypointense signal on T1-weighted images, are heterogeneous signal intensity on T2-weighted images, and may enhance homogeneously or heterogeneously [28, 29].

Calcium pyrophosphate deposition disease (CPPD) is a metabolic disorder characterized by intraarticular and periarticular accumulation of calcium pyrophosphate dihydrate crystals. This disease often has alternating phases of modest chronic inflammation with superimposed episodes of intense inflammation [30]. Pseudogout is the most common manifestation of CPPD and refers to the presence of goutlike arthritis and chondrocalcinosis or calcification within fibrous or hyaline cartilage structures [30]. CPPD is more common in women than men and is usually secondary to a familial cause or metabolic disorder such as hyperparathyroidism, hemochromatosis, or hypothyroidism [30].

Pseudogout tends to be symmetric in distribution and affects hyaline cartilage and fibrocartilage of non–weight-bearing joints such as the shoulder, elbow, wrist, patellofemoral, and metacarpophalangeal joints [30]. It can also involve the pubic symphysis, acetabular labra, and annulus fibrosis. Deposition will be evident on MRI as linear or punctate areas of hypointense signal within hyaline cartilage or intervertebral disks on all sequences, and the mineralization may be more prominent on gradient-echo sequences or 3-T imaging. At the time of acute attacks, joint effusion and soft-tissue edema will often be seen. Later stages of disease can have an OA-like appearance or even neuropathic joint–like appearance with osseous fragmentation and collapse and intraarticular bodies [30].

Hydroxyapatite deposition tends to be asymptomatic but can be associated with acute inflammation, soft-tissue deposition, and chronic inflammation [31]. The hydroxyapatite deposits occur in fibrous connective tissues and are usually amorphous, milky, or cheesy in quality. Deposition is most common in the shoulder—specifically, in the rotator cuff tendons [31]. The surrounding inflammatory changes induced by the crystal disease in the shoulder can be detected as hyperintense signal changes and fluid in the subacromial or subdeltoid bursa and are best seen on fluid-sensitive sequences. “Milwaukee shoulder” is an eponym for the extreme, destructive shoulder arthropathy caused by hydroxyapatite deposition, which was originally identified in elderly women and is accompanied by rotator cuff tears [31].

Synovial-Based Processes
Synovial chondromatosis is a monarticular, benign neoplastic, synovial-based process that results in synovial generation of multiple cartilaginous nodules. It is most prevalent in the third to fifth decades and affects men 2–4 times more than women [32]. Patients typically present with joint pain, swelling, and limitation of motion. The knee, hip, and elbow are the most commonly involved joints. Cartilaginous nodules can detach and become intraarticular bodies and can also become ossified (osteochondromatosis). Unmineralized cartilaginous nodules are fairly isointense to hyaline cartilage on all sequences, and ossified bodies will parallel medullary and cortical signal intensity on all sequences. Marginal erosions secondary to increased intraarticular pressure can also occur, especially in tight joints such as the hip.

Pigmented villonodular synovitis (PVNS) is another synovial-based process characterized by hyperplasia of the synovium with hypervascularity and accumulation of histiocytes [33]. Masslike synovial and soft-tissue proliferation is usually monoarticular and most commonly affects the knees, hips, ankles, and shoulders [33]. Patients are typically young to middle-aged adults who present with joint pain, swelling, and limitation of motion. PVNS lesions have a propensity to bleed and cause accumulation of intra- and extracellular hemosiderin [32]. The paramagnetic effect from the hemosiderin deposits causes the nodular intraarticular masses to be hypointense on all sequences (Fig. 7), with blooming effect on gradient-echo sequences. Joint space and bone mineral density tend to be preserved until late in the disease process in PVNS. Erosions are less common compared with osteochondromatosis but can occur, especially in the hip [32].

Conclusion
Arthritic shoulders, hips, spine, and sacroiliac joints are often included in chest, abdominal, and pelvic MRI examinations interpreted by body imagers. Common MRI findings of OA include articular cartilage loss, osteophyte formation, BME, synovitis, joint effusions, meniscal abnormalities, and acetabular and glenoid labral abnormalities. RA is characterized by synovitis, pannus formation, BME, and erosions. The seronegative spondyloarthropathies have a strong association with HLA-B27 positivity, sacroilitis, enthesitis, and inflammatory back pain. Septic arthritis can be rapidly destructive and should be suspected in patients with bacteremia, leukocytosis, joint effusion, synovial enhancement, and periarticular edema. Gout rarely involves the spine or axial joints but can show marginal erosions with an overhanging edge, BME, and tophi. Finally, PVNS has a characteristic “blooming” synovial proliferation due to hemosiderin deposition, and synovial chondromatosis should be considered in patients with joint pain, limited joint motion, and synovial-based or intraarticular bodies.

Grindol et al.