Monoarticular disease is not an uncommon clinical presentation, and the diagnosis can often be challenging due to its nonspecific clinical signs and symptoms, which include pain, joint swelling, and decreased range of motion. Radiography is often used initially to aid the clinician in the detection and diagnosis of monoarticular arthropathies. Because the diagnosis of these similar clinically presenting diseases may often have very different management strategies, careful review of the imaging features is necessary to narrow the list of differentials and, at times, suggest a specific diagnosis.

This article will review the imaging features of the various etiologies of monoarticular arthropathy including, infectious, traumatic, crystalline, deposition, tumefactive, neuropathic, idiopathic, and foreign-body synovitis (Table 1).

### Infectious

When presented with a monoarticular arthropathy, particularly in a patient who presents acutely with significant pain, it is essential for the clinician and radiologist to first consider an infectious etiology. In these cases, laboratory findings and joint aspiration are critical for the prompt diagnosis of the disease. Infectious arthritis refers to involvement of the joint by a microorganism; it may be divided into septic and aseptic, the former accounting for the majority of cases. Delay in diagnosing septic arthritis can lead to considerable morbidity and, in rare instances, death.

#### Septic (Pyogenic)

Septic arthropathy is a suppurative infectious process of the joint which is caused by pyogenic bacteria. The causative microorganisms are dependent on the patient’s age and comorbidities (Table 2). Common etiologies in adults include *Staphylococcus aureus*, *Streptococcus pneumonia*, and Beta hemolytic streptococci. Acute bacterial arthritis in adults and children older than two years is most often caused by *S. aureus.* The infectious agent is usually spread hematogenously to the richly vascular synovium from a pre-existing, distant infection. Less common causes include direct spread from osteomyelitis, contiguous spread from soft tissue infection, iatrogenic secondary to a prior joint injection or operation, and rarely from direct seeding secondary to penetrating trauma. Common risk factors for septic arthritis include recent bacteremia and age greater than 60 years. Other risk factors include skin infection, diabetes mellitus, rheumatoid arthritis, joint surgery, joint arthroplasty, and corticosteroid therapy.

The knee is the most commonly involved joint in septic arthritis, accounting for approximately 50% of cases. Initially, septic arthritis may be radiographically occult. The earliest imaging findings will typically include localized soft tissue edema and joint effusion, followed shortly by peri-articular...
osteopenia related to the hyperemic response to the infection. Within days, joint space narrowing will present as a result of chondrolysis from the proteolytic enzymes released into the joint (Fig. 1A). A Focal erosions and ultimately osseous destruction will result (Fig. 2A, 2B). Findings in children may have an atypical clinical presentation or subtle radiographic findings. Ultrasound may be utilized to confirm the presence of a joint effusion and assist in image-guided aspiration when an effusion is detected (Fig. 2C) MRI is reserved for evaluation of associated soft tissue infection or abscess, as well as the detection of osteomyelitis (Fig. 1B, 1C).

<table>
<thead>
<tr>
<th>Organism</th>
<th>Patient Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>Most common in all age groups</td>
</tr>
<tr>
<td>Group B streptococcus</td>
<td>Most common in neonates and infants</td>
</tr>
<tr>
<td>Hemophilus influenza</td>
<td>Most common in preschool-aged children</td>
</tr>
<tr>
<td>Neisseria gonorrhoeae</td>
<td>Most common among sexually active young adults</td>
</tr>
<tr>
<td>Gram-negative bacteria</td>
<td>Common cause in chronic illness with concurrent GU infection</td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td>Common cause in chronic illness with concurrent pulmonary infection</td>
</tr>
</tbody>
</table>

Table 2. Etiologies of Septic Arthritis

Figure 1. Septic Arthropathy of the Right Hip in a 9-Year-Old Boy.
AP pelvic radiograph (A) shows marked narrowing of the right femoral acetabular joint. Coronal fat suppressed T2W (B) and T1W (C) images demonstrate a large joint effusion. Bone marrow edema across the joint is consistent with associated osteomyelitis.

Figure 2. Septic Arthritis. Coronal CT image of the pelvis (A) and a magnified view of the left hip (B) in a 31-year-old intravenous drug user presenting with left hip pain reveal subtle focal erosion and osteolysis along the lateral aspect of the left hip. Sagittal ultrasound of the left hip (C) reveals a large joint effusion (*) with associated synovitis (arrow).
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Aseptic.

Nonsuppurative joint infections are typically caused by mycobacterial, fungal, or viral infections and are uncommon causes of infectious monoarticular arthritis. An important nonsuppurative bacterial pathogen, particularly in regions where Lyme disease is endemic (i.e., the Northeast and Great Lakes regions of the U.S.), is *Borrelia burgdorferi*. Patients with arthralgia related to Lyme disease will typically have inflammation of a single large joint, commonly the knee. A large joint effusion is usually the striking imaging feature (Fig. 3). Monoarticular disease can also be the presenting manifestation of human immunodeficiency virus (HIV) infection. There are numerous other viral cause of infectious arthritis, including Parovirus B19, Hepatitis A, B & C, Rubella, and Mumps.

Traumatic

Acute or chronic post traumatic arthropathy will often manifest as a monoarticular disease. Acute traumatic arthropathy can be the result of injury to the intrinsic structures of the joint, such as the hyaline or intra-articular fibrous cartilage, to include the meniscus or labrum in the knee or shoulder, respectively. Disruption of the supporting structures of the joint, including the joint capsule or ligaments, can also be the source of traumatic arthropathy. With chronic or repetitive trauma, a pattern of osteoarthritis will develop but will be monoarticular, suggesting a secondary cause.

Acute post-traumatic arthropathy will be seen as soft tissue swelling at the joint and effusion. MRI will often directly identify the specific injury which may include chondral, osteochondral, ligamentous, or capsular injury. In the chronic setting, secondary osteoarthritis at the joint will develop with radiographs or computed tomography (CT) demonstrating the typical non-uniform joint space narrowing, subchondral sclerosis, and osteophytes.

Crystalline

Gout.

Gout is a rheumatic disease which may be exhibited in up to 10% of patients with underlying hyperuricemia at some point during their lifetime. Isolated hyperuricemia is asymptomatic and may result from primary and secondary causes. Primary causes include inborn errors in purine metabolism or inherited defects in renal excretion of urate. Secondary causes include medications, disorders which increase nucleic acids metabolism and thus increase uric acid production, and acquired defects resulting in decreased uric acid excretion. Those who develop gout will have the deposition of monosodium urate crystals within joints or adjacent soft tissues. The stages of gout may be divided into acute, intermediate or intercritical, and chronic tophacious gout.

Figure 3. Lyme Arthritis. Lateral knee radiograph (A) shows a large joint effusion. Fat suppressed T2WI (B) demonstrates the large effusion, a Baker’s cyst, and soft tissue edema. Post-contrast fat suppressed T1WI (C) reveals prominent enhancement of the synovium of the joint and peripherally around the Baker’s cyst. (Images courtesy of Joseph Makris, MD)
primary causes of hyperuricemia account for up to 90% of these cases.\textsuperscript{11}

The imaging features of gouty arthritis will largely be dependent on where the crystals are deposited, as well as the stage of the disease. When the crystals are deposited within the articular cartilage, a nonspecific osteoarthritic pattern will result in the affected joint (Fig. 4).\textsuperscript{12} A monoarticular pattern of osteoarthritis should prompt the radiologist to consider secondary causes including post-traumatic, post-infectious, and crystalline disease. Acute gouty arthritis is often monoarticular and typically diagnosed by clinical and laboratory evaluation. When the crystals are deposited into the soft tissues, chronic tophaceous gout will often be the result. The distribution of tophaceous gout is variable and includes symmetric polyarticular disease, asymmetric polyarticular disease, and monoarticular disease. Common locations of involvement include, in decreasing frequency, feet, hands, wrists, elbows, and knees. Less than half of those who develop gout will exhibit radiographic changes.\textsuperscript{12} When present, radiographic features include eccentric nodular soft tissue density representing the tophus (Fig. 5). Subjacent to the soft tissue tophus, intra-articular or juxta-articular erosions will often develop. The erosions are typically eccentric with sclerotic borders. Joint spaces are preserved until late in the disease.\textsuperscript{13,14} Soft tissue tophi will be hyperdense on CT and variable signal intensity on MRI due to their variable tissue composition.

Milwaukee Shoulder.

Milwaukee shoulder presents as a rapidly progressive arthropathy of the shoulder which exhibits joint destruction, synovial hyperplasia, and a non-inflammatory joint effusion containing both calcium hydroxyapatite (HA) and calcium pyrophosphate dehydrate (CPPD) crystals with a large rotator cuff tear.\textsuperscript{15} This destructive arthropathy most frequently occurs in older women with clinical symptoms of pain and limited range of motion.

The dominant imaging feature of Milwaukee shoulder is its rapid course of joint destruction (Fig. 6). Early, there may be soft tissue swelling and peri-articular calcifications seen radiographically.\textsuperscript{10} As the disease progresses, the joint space becomes narrowed from chondrolysis, followed by subarticular osseous
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Destruction. There is often capsular calcifications and intra-articular bodies. Early, MRI may reveal synovial proliferation and joint effusion with an associated full thickness rotator cuff tear. Later, the features of articular cartilage and osseous destruction are quite evident (Fig 7).

Deposition

Amyloid Arthropathy.

Amyloids are insoluble fibrous protein aggregates. The extracellular deposition of these fibrous proteins is referred to as amyloidosis. Hemodialysis-associated amyloidosis is a form of amyloidosis seen with chronic renal failure. In this form of amyloidosis, the low-molecular-weight serum protein β2 microglobulin (Aβ2M) is not filtered by standard dialysis membranes and subsequently deposits in the musculoskeletal system. These patients may develop carpal tunnel syndrome and osteolytic bone lesions or amyloidomas. Amyloid arthropathy occurs when Aβ2M deposition occurs within the joint.

Amyloid arthropathy is typically a polyarticular process but may at times occur as a monoarticular arthropathy. Large joints are most commonly involved, including the shoulders, knees, wrists, and elbows. Amyloid arthropathy may resemble inflammatory arthropathies and mimic rheumatoid arthritis on radiographic examination due to juxta-

Figure 6. Milwaukee Shoulder. AP radiograph of normal left shoulder (A). Several months later, Grashey view of the same shoulder (B) demonstrates interval subarticular osseous destruction and capsular calcifications.

Figure 7. Milwaukee Shoulder. Oblique coronal T1WI (A) and fat suppressed T2WI (B) from an MRI of the left shoulder demonstrates a large effusion. Post-contrast fast suppressed T1WI (C) shows synovial proliferation exhibited by prominent synovial enhancement. There is frank osseous destruction of the subarticular portions of the humeral head.
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articular soft tissue swelling, periarticular osteopenia, and subchondral cysts. However, amyloid arthropathy affects the hips and shoulder more than peripheral joints and lacks significant distal synovial involvement, allowing for differentiation. As the disease progresses, joint space narrowing and well-defined erosions with sclerotic margins will develop radiographically. The classic "shoulder-pad" sign may be present indicating amyloid deposits not only within the shoulder joint but also within the periarticular structures, including the subacromial-subdeltoid bursa (Fig. 8). MRI examination will reveal intermediate low T1 and intermediate low T2 signal corresponding to areas of amyloid infiltration within the synovium, subarticular bone, and periarticular soft tissues.20

Hemophilic Arthropathy.

Hemophilic arthropathy occurs in patients with hemophilia A or B who have repeated episodes of hemorrhage. Bilateral joint involvement is common; however, repetitive hemorrhage typically involves a single joint. Hemophilic arthropathy occurs in the first and second decades of life and commonly involves the knee, ankle, elbow and shoulder.10 Chronic recurrent hemorrhage within the joint leads to synovial hyperplasia, chronic inflammatory changes, fibrosis, and siderosis of the synovium.10

Radiographic features are varied depending on the stage of the disease. Acute findings can include dense effusion representing hemarthrosis and osteopenia from local hyperemia. A common radiographic feature includes overgrowth of the epiphysis secondary to chronic hyperemia. These changes are more pronounced the earlier the onset of the disease. Chronic changes also may include joint space narrowing, subchondral cystic changes and erosions related to underlying synovial hyperplasia and chronic joint inflammation (Fig. 9). Synovial hypertrophy secondary to the chronic hemorrhage is directly demonstrated by MRI. Hemosiderin within the synovium is exhibited by low signal on all pulse sequences with blooming on gradient echo sequences secondary to the magnetic susceptibility effect.21
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Tumefactive

Primary Synovial (Osteo)chondromatosis.

Synovial (osteo)chondromatosis may be characterized as primary or secondary. Secondary osteochondromatosis is associated with osteoarthritis and merely reflects intra-articular osteochondral bodies within the joint. Primary synovial (osteo)chondromatosis is a benign condition of unknown origin characterized by the formation of cartilaginous nodules within the synovium. The exact pathogenesis of the disease is uncertain but likely involves proliferation and metaplasia of the synovial tissue with formation of multiple cartilaginous or osteocartilaginous nodules. Primary synovial (osteo)chondromatosis typically involves large joints. The knee is most commonly involved, followed by the elbow (Fig. 10), hip, and shoulder; however, any synovial joint may be involved.\textsuperscript{22,23} Symptoms are typically insidious and include pain, swelling, and limited range of motion. The disease may affect a wide range of ages with most presenting within the third to fifth decades of life. A male predominance as high as 4:1 has been reported.\textsuperscript{24}

The appearance of the intra-articular bodies varies depending on the content, which may include cartilage, cartilage and bone, or mature bone with fatty marrow.\textsuperscript{10} Radiographically, these intra-articular bodies may vary from fine calcifications to laminated bodies, typically of similar size. Treatment includes removal of the intra-articular bodies and synovectomy with a recurrence rate of up to 11%.\textsuperscript{25,26} Malignant transformation in synovial (osteo)chondromatosis has been reported but is very rare.\textsuperscript{27}

Figure 10.
Synovial Osteochondromatosis.
Lateral radiograph of the elbow demonstrates innumerable calcified intra-articular bodies throughout the joint.

Figure 11.
Pigmented Villonodular Synovitis. Lateral radiograph (A) of the right knee demonstrates soft tissue swelling and a joint effusion without calcifications. PA radiograph (B) demonstrates preservation of the joint space and bone density.

Pigmented Villonodular Synovitis (PVNS).

PVNS is a benign, locally aggressive, proliferative disorder of the synovium.\textsuperscript{28} PVNS may be characterized in one of three forms: extra-articular, localized intra-articular, and diffuse intra-articular.\textsuperscript{29} The origin of PVNS is unknown with neoplastic and inflammatory etiologies being the leading considerations. Similar to synovial (osteio)chondromatosis, PVNS predominately involves large joints. The knee is the most frequently involved joint, accounting for up to 80% of intra-articular PVNS.\textsuperscript{30,31} Other joints include the hip in up to 18% of cases, and less frequently the ankle, glenohumeral, and elbow joints. Additional locations and polyarticular involvement are rare.\textsuperscript{32} PVNS typically presents in the second and fourth decades of life with men and women equally affected.\textsuperscript{28,32}

In cases of monoarticular disease, intra-articular PVNS will present with normal radiographs or may demonstrate periarticular soft tissue swelling without calcifications (Fig. 11). There is neither periarticular osteopenia nor significant joint space narrowing until late in the disease process. Well-defined erosions with sclerotic margins may be present in less capacious joints compared to the knee, such as the hip.\textsuperscript{33} MRI directly demonstrates low signal on all pulse sequences within the synovium representing the hemosiderin deposition (Fig. 12).

Figure 11.
Pigmented Villonodular Synovitis. Lateral radiograph (A) of the right knee demonstrates soft tissue swelling and a joint effusion without calcifications. PA radiograph (B) demonstrates preservation of the joint space and bone density.
Treatment includes synovectomy; however, local recurrence is common, affecting approximately half of those treated.\textsuperscript{34} Few cases of malignant transformation have been reported and are exceedingly rare.\textsuperscript{35}

**Neuropathic**

First described in patients with tabes dorsalis, neuropathic arthropathy or Charcot joint may complicate disease processes which alter sensation within a joint. Although the exact mechanism is not well understood, current consensus supports the loss or diminished deep sensory innervation and proprioception, combined with repetitive trauma to the joint, as central to its pathogenesis.\textsuperscript{36} The location of involvement in neuropathic arthropathy is dependent on the underlying cause. The common neuropathic joint involvement from peripheral neuropathy secondary to diabetes mellitus or alcoholism at the foot is more typically oligo or polyarticular. Monoarticular neuropathic joint involvement is more commonly related to syringomyelia with preferential involvement of the glenohumeral joint.

Radiographic features vary from an atrophic joint with total resorption to a hypertrophic joint with excessive repair.\textsuperscript{37} Early changes can overlap with osteoarthritis. As the process progresses, typical imaging features include fragmentation or depression of subchondral bone, sclerosis, subluxation, intraarticular osseous fragments or debris, and effusion (Fig. 13).

Figure 12. **Pigmented Villonodular Synovitis.** Right knee MRI demonstrates small pressure erosions on axial T1WI (A) and low signal on the pre (B) and post (C) contrast fat suppressed T1WI within the enhancing synovial tissue. Sagittal T1WI (D) and fat suppressed T1WI (E) show diffuse low signal throughout the joint, representing the hemosiderin ‘pigmentation’ of the hypertrophied synovium.

Figure 13. **Neuropathic Arthropathy.** Sagittal CT image through the shoulder demonstrates destruction and depression of the subchondral proximal humerus, sclerosis of the remaining humeral head, and a joint effusion with extensive intra-articular osseous debris.
These areas of localized inflammatory response result in local osteolysis which may occur anywhere along the bone-component interface, including areas such as empty screw holes in the acetabular component. Early in the process, patients may be asymptomatic. Radiographically, focal lucencies adjacent to the prosthesis classically develop, representing local areas of osteolysis (Fig. 15). Radiographs may underestimate the extent of osteolysis, which can be more accurately assessed with CT. The patient may experience pain as osteolysis becomes increasingly severe and aseptic loosening occurs. Ultimately, failure of the femoral and/or acetabular components may result. When this occurs, spontaneous periprosthetic fracture due to migration of the components, limb shortening, and severe pain are usually seen. Although several medical therapies have been described, surgical revision is almost always necessary to correct the defects.

Idiopathic

Rapidly Progressive Osteoarthropathy. This unusual type of osteoarthritis most frequently involves the hip and is almost always monoarticular. Rapidly Progressive Osteoarthropathy (RPO) also goes by the name Rapidly Destructive Articular Disease or Rapidly Destructive Osteoarthritis, each describing the acute and destructive nature of this form of osteoarthritis (Fig. 14). RPO frequently involves older women with an average age in the seventh decade. These patients will have typical osteoarthritic changes elsewhere. The exact trigger or cause is not well understood. However, the common clinical symptom is a rapid clinical course of hip pain. Almost all patients will require total joint replacement due to the severity of joint damage.

Foreign Body Synovitis

Particle Disease. The most frequently encountered foreign body synovitis is particle disease. Particle disease is also known as aggressive granulomatosis and is typically a late complication of joint replacements. There is little evidence to suggest that co-morbid illnesses, medication use, or specific surgical factors predispose one to the development of this condition. It is believed to result from an inflammatory response of the bone to small components of the high-density polyethylene worn from the articular lining of metallic hardware. These areas of localized inflammatory response result in local osteolysis which may occur anywhere along the bone-component interface, including areas such as empty screw holes in the acetabular component. Early in the process, patients may be asymptomatic. Radiographically, focal lucencies adjacent to the prosthesis classically develop, representing local areas of osteolysis (Fig. 15). Radiographs may underestimate the extent of osteolysis, which can be more accurately assessed with CT. The patient may experience pain as osteolysis becomes increasingly severe and aseptic loosening occurs. Ultimately, failure of the femoral and/or acetabular components may result. When this occurs, spontaneous periprosthetic fracture due to migration of the components, limb shortening, and severe pain are usually seen. Although several medical therapies have been described, surgical revision is almost always necessary to correct the defects.

Metallosis.

Metallosis occurs as an uncommon complication of joint replacements. This metal synovitis may occur after failure of the interposed polyethylene surface in total knee replacements. More recently, this complication has been seen in metal on metal total hip replacements, where fine particles from the prosthesis produce a metal-induced chronic synovitis. Similar to particle disease, focal osteolysis occurs at the prosthesis-bone interface. Radiographic features

Figure 14. Rapidly Progressive Osteoarthropathy. Baseline (A) and followup (B) AP radiographs of the right hip demonstrate the rapid course of articular cartilage destruction with typical osseous features of osteoarthritis, including subarticular sclerosis and cystic changes.

Figure 15. Particle Disease of the Left Hip. AP radiograph of the pelvis in a patient with left total hip arthroplasty. There is asymmetric position of the femoral head component within the acetabular cup resulting from wear of the polyethylene liner. There is focal lobular lucency about the acetabular component of the prosthesis as a result of osteolysis (arrows) from foreign body reaction to the particles.
findings include soft tissue edema around the joint (Fig. 17). These metal particles produce the low signal or susceptibility within thickened synovium on all MRI pulse sequences.

Silicone Synovitis.

Also known as prosthetic synovitis, silicone synovitis is a form of chronic foreign body synovitis caused by silicone particles which have displaced from the native prosthesis, inciting a foreign body reaction. Silicone synovitis is seen in patients with metacarpophalangeal joint implant arthroplasty or carpal implants with a silastic spacer. Radiographic findings include fine metal particles within or outlining a distended joint (Fig. 16). These metal particles produce the low signal or susceptibility within thickened synovium on all MRI pulse sequences.

Summary

Monoarticular arthropathy may be caused by a variety of conditions. It is important to correlate the imaging features with the clinical presentation when developing an appropriate diagnosis or list of differentials. The most important diagnosis to consider in the acute setting is septic arthritis, as delay in recognition and management may have considerable morbidity. Joint aspiration may be required for diagnostic confirmation, especially in the setting of suspected infectious or inflammatory etiologies. Understanding the imaging features of the various causes of monoarticular arthropathy is essential in aiding with diagnosis and guiding proper management of this common clinical entity.
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