JAOCR
Official Journal of the American Osteopathic College of Radiology

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In this Issue

Courtney Scher, D.O., 1 and Leah Davis, D.O. 2

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I would like to thank Drs. William O’Brien and Daniel Wale for the invitation to contribute to the January MSK issue of the JAOCR and for the opportunity to collaborate with my dear friend and colleague, Dr. Leah Davis, as co-guest editors. Dr. Davis and I completed our MSK fellowship training at Henry Ford Hospital in Detroit and became friends when she was one of my fellows a few years ago. Our training and time at Henry Ford Hospital has allowed us to gain a significant amount of experience with MSK ultrasound (US), which we have incorporated into our articles in this issue. The field of MSK US is expanding quickly and many of our clinical colleagues have begun to utilize this tool. It is our hope to encourage more radiologists to take an interest in this growing field as well. In this issue, we have included something of interest for all levels—residents, fellows, and staff, both general and subspecialized.

The review articles in this issue examine the imaging evaluation and common pathology of the lateral ankle and elbow joint. In our experience, the ankle and elbow can be intimidating at first due to the somewhat complex anatomy and their relative infrequency of imaging compared with other joints. Our hope is that we have simplified the evaluation of these joints with a thorough anatomic review and structured approach to image interpretation. We have included US along with MR imaging findings in hopes of highlighting the unique capabilities of each modality.

Our case presentations and Viewbox articles contain a variety of interesting cases that can cause diagnostic dilemmas upon initial presentation. We have attempted to provide a detailed approach to these cases to make them more recognizable in clinical practice.

We would both like to acknowledge our mentors at Henry Ford Hospital, Drs. Joseph Craig and Marnix van Holsbeeck who supported us during our training and have continued to do so throughout our professional careers.

We hope that you enjoy this issue and that it inspires you to delve deeper and expand your knowledge not only of musculoskeletal radiology, but also MSK US.

“Surround yourself with people that push you to do better. No drama or negativity. Just higher goals and higher motivation.”

Warren Buffett
Musculoskeletal ultrasound (US) is an ideal imaging modality for assessing elbow joint anatomy and pathology. The elbow joint is particularly amenable to US evaluation as it is a small joint with 360-degree accessibility by the transducer and is easily manipulated, both actively by the patient and passively by the examiner. Beyond diagnostics, US can also aid in image-guided treatment of certain conditions.

MRI also plays an important role in evaluating the elbow. In addition to soft-tissue anatomy and pathology, MRI also depicts radiographically occult fractures, bone contusions, and intra-articular pathology exquisitely. Magnetic resonance arthrography (MRA) utilizing gadolinium-based contrast placed directly into the joint allows for optimal evaluation of articular cartilage/intracapsular structures, intra-articular loose bodies, and the collateral ligaments.1,2

This article will review the anatomy and pathology of the elbow joint and review the US examination by compartment, featuring the most pertinent anatomic structures and their related pathology (Table 1). Correlative MRI evaluation of the elbow joint and pathology will also be discussed.

Osseous Articulations

The ulnohumeral articulation is a hinge joint, providing the majority of flexion and extension motion at the elbow. The radiocapitellar articulation is a “hinge and pivot” joint without inherent osseous mobility. The proximal radioulnar joint, held in place by the annular ligament, allows for rotation of the radial head in the sigmoid notch of the ulna in supination and pronation. These three articulations, as well as anterior and posterior fat pads, are encased by the joint capsule.1

Many muscles, tendons, ligaments, and nerves work together with this bony foundation to create the elegant, dynamic, and complex function of the elbow joint. These structures and their related pathologies will be discussed further, based on the compartment in which they reside.

Anterior Compartment

Structures of interest in the anterior elbow include the joint recess, distal biceps tendon insertion, brachialis muscle, as well as the median and radial nerves (Figure 1).4,5

The biceps brachii, composed of a long head originating from the supraglenoid tubercle and a short head from the coracoid process, crosses the shoulder and elbow joints. As it courses distally over the anterior humerus, the muscle bellies of the lateral long head and medial short head gradually, but incompletely, insinuate with one another.
Just proximal to the elbow joint, the biceps brachii muscle separates again into two distinct tendon heads. Both heads contribute to the distal biceps tendon coursing through the cubital fossa and inserting on the radial tuberosity.6

Evaluation of the distal biceps tendon (DBT) can be challenging due to its oblique course and deep insertion.4,5 The normal tendon may appear hypoechoic as it dives deep, secondary to an artifact known as anisotropy.1 Keeping the probe parallel to the tendon in long axis and perpendicular in short axis is important for avoiding this artifact and causing subsequent misinterpretation. Long-axis images are ideal for evaluating the distal biceps tendon insertion on the radial tuberosity (Figure 2).1,5

At the anterolateral aspect of the elbow, the radial nerve and its posterior interosseous branch (PIN) can be visualized. Of note, it is important to visualize the PIN as it pierces and then travels through the two heads of the supinator muscle and the Arcade of Frohse (Figure 3). The course of the PIN, as it travels from the anterior to the posterior compartments, may be obtained in the transverse plane as the subject pronates the forearm.4,5

**Distal Biceps Tendon Tear**

The biceps tendon is the most powerful forearm supinator and is the most commonly torn tendon in the elbow. Injury is common at or near the insertion of the DBT on the radial tuberosity, owing to its relative hypovascularity in this location.1 Tendon rupture most often results from acute trauma rather than overuse, and is relatively common in rugby players and weight lifters when the arm is forcefully extended against a flexed elbow.7 Of note, rupture of the proximal long-head biceps tendon at the shoulder joint is approximately 10 times more common than rupture of the DBT at the elbow.7

In the setting of complete tendon rupture, the lacertus fibrosus may prevent retraction of the DBT proximally. If the lacertus fibrosus is also torn, a complete tear can present as a palpable painful mass over the proximal anterior arm. Early diagnosis of a complete DBT tear is critical, as clinical outcome is greatly improved with early surgical intervention within the first few weeks following injury. This is in contrast with tears of the proximal biceps tendon, where conservative treatment is the therapy mainstay.7

Complete tear of the DBT is amenable to US diagnosis. Findings include nonvisualization of the tendon at the insertion site with hypoechoic fluid collection/hematoma in the tendinous bed (Figure 4). Partial tears of the DBT are
often more difficult to diagnose by US compared to MRI. In the setting of partial DBT tear, the thinned tendon will assume an irregular, attenuated profile or wavy contour (Figure 5).7

MRI is particularly useful for diagnosing a DBT injury and discerning the degree of severity. DBT is best evaluated on axial images, although fluid-sensitive sagittal images can help confirm a complete rupture and estimate the degree of stump retraction. Focal T2 hyperintensity in and adjacent to the DBT indicate a partial tear (Figure 5).7

**Lateral Compartment**

Important lateral elbow structures to evaluate include the common extensor tendon, lateral collateral ligament complex, and radiocapitellar joint.4,5

Placement of the transducer longitudinally over the lateral epicondyle will reveal the radiocapitellar articulation, radial collateral ligament (RCL), and overlying thin, long common extensor tendon (CET) (Figure 6).1

The CET is best visualized on the coronal plane/long axis and is composed of the origins of the extensor carpi radialis longus and brevis, extensor digiti minimi, and extensor digitorum communis tendons.5 On US, the CET is a smooth hyperechoic beak-shaped structure originating from the anterolateral aspect of the lateral epicondyle, with the radial collateral ligament just deep to the tendon origin (Figure 6).1 The radiocapitellar joint is visible deep to the CET and the ligament.4,5

**Lateral Epicondylitis**

Lateral epicondylitis is the most common sports-related elbow injury, classically seen in tennis players, but is also commonly seen secondary to repetitive work-related activities.7 Lateral epicondylitis involves the common extensor tendon origin at the lateral epicondyle, with the extensor carpi radialis brevis tendon the most commonly affected.8 Despite the “-itis” suffix, lateral epicondylitis is degenerative in nature, resulting from overuse of the extensor and supinator muscles.7,8

Tendinosis can be recognized by US as an abnormal hypoechoic thickened appearance of the CET origin. Areas of osseous irregularity underlying the CET may be present with hyperemia seen during color or power Doppler interrogation (Figure 7). An anechoic cleft with complete or incomplete disruption of tendon fibers will be seen with partial and full thickness tears, respectively.
On MRI, lateral epicondylitis is demonstrated by intermediate T1- and increased T2-weighted signal representative of intratendinous mucoid degeneration and neovascularization (Figure 7). These findings result from microscopic tearing and formation of reparative tissue. Over time, macroscopic tearing or injury to the radial collateral ligamentous complex may also occur. Many secondary findings suggestive of lateral epicondylitis may be observed: bone marrow edema, periostitis of lateral epicondyle, anconeous muscle edema, or fluid in the radial head bursa.7

Of special importance in the setting of lateral elbow trauma, is evaluation for concomitant injury of the CET and underlying lateral collateral ligament complex, which can result in posterolateral rotary instability (PLRI). Tears of the RCL are especially important to note as surgical debridement of the CET could result in further destabilization of an already injured RCL.7

The lateral ulnar collateral ligament (LUCL) is a component of the lateral collateral ligament complex. It arises from the inferior surface of the lateral epicondyle, taking an oblique medial course to insert on the proximal ulna. It is a primary restraint to varus stress of the elbow and when injured, can also contribute to PLRI. The LUCL is best visualized on coronal MRI sequences (Figure 8).9 It is not reliably well visualized by US.

**Medial Compartment**

US evaluation of the medial elbow is primarily performed to evaluate the common flexor tendon (CFT), ulnar collateral ligament (UCL), and the ulnar nerve.4,5

The CFT origin on the medial epicondyle of the humerus includes the flexor carpi radialis and ulnaris, palmaris longus, flexor digitorum superficialis, and pronator teres tendons. Positioning the transducer longitudinally with the proximal aspect over the medial epicondyle will reveal the CFT in long axis (Figure 9).1

The UCL is the primary static stabilizer of the elbow against valgus stress.1
With the probe oriented obliquely longitudinally over the medial epicondyle, the anterior bundle of the UCL will be visible in long axis (Figure 10). Dynamic imaging of the UCL with valgus stress on the ulnohumeral joint should also be performed for complete assessment. Subsequent joint space widening will accentuate ligament laxity, if present, and may reveal a partial tear of the ligament.4,5

At the level of the cubital tunnel, the ulnar nerve can be well evaluated. In cross-section/short axis, the nerve will appear as a hypoechoic rounded structure surrounded by echogenic fat, creating a honeycomb appearance (Figure 11).1 Dynamic maneuvers can be performed while visualizing the ulnar nerve in short axis to assess for subluxation or dislocation over the medial epicondyle during elbow flexion.4,5

Medial Epicondylitis

Medial epicondylitis, also known as golfer’s or pitcher’s elbow, develops in the setting of overuse or trauma and involves the CFT at its origin, most commonly affecting the pronator teres and flexor carpi radialis tendons.4,5,8 Patients may complain of aching pain over the medial elbow and, in chronic cases, grip strength may weaken.7 Medial epicondylitis is much less common than lateral epicondylitis.7

US is an important modality for diagnosing medial epicondylitis and is especially useful to distinguish medial epicondylitis from a tear of the deeper UCL.7 Tendinosis can be recognized as abnormally thick, hypoechoic areas in the CFT at its origin (Figure 12). Additionally, areas of bony irregularity of the medial epicondyle may be present, as well as peritendinous fluid, hyperemia, or soft-tissue swelling.7,8 An anechoic cleft with complete or incomplete disruption of tendon fibers will be seen with partial and full thickness tears, respectively.8 Because of the close spatial and functional relationship of the CFT, UCL, and ulnar nerve, these structures are susceptible to concomitant injury. As many as 60% of patients...
undergoing surgery for medial epicondylitis have been found to exhibit signs of ulnar nerve neurapraxia.\textsuperscript{7,10}

MRI imaging in the setting of medial epicondylitis demonstrates increased T1- and T2-weighted signal, representative of intratendinous mucoid degeneration and neovascularization as a result of microscopic tearing and formation of reparative tissue. Over time, macroscopic tearing may occur.\textsuperscript{7}

**Injury to the Anterior Band of the Ulnar Collateral Ligament**

The most clinically relevant and commonly injured component of the UCL is the anterior band that extends from the undersurface of the medial epicondyle to the sublime tubercle on the proximal ulna.\textsuperscript{4,5} The UCL is a primary elbow joint stabilizer, providing nearly 50% of the protection against valgus stress. Injury to the anterior band of the UCL is classically seen in

**FIGURE 13.** Complete tear anterior band UCL. US shows acute rupture of the anterior band UCL in a baseball pitcher. The entire ligament is thickened (small arrows) and there is a complete hypoechoic tear proximally (large arrow). Note also the redundancy of the ligament secondary to the complete tear. A small amount of hypoechoic fluid is seen in the medial elbow joint (arrowhead) (A). MRI shows proximal tear of the anterior band UCL (long arrow) and associated thickening of the more distal ligament (short arrows) (B).

**FIGURE 14.** Partial tear of the anterior band of the ulnar collateral ligament. Note the irregularity and slight swelling of the anterior band (small arrows) and the widening of the medial elbow joint space (large arrow) (A). Corresponding fat-suppressed T1-weighted MR arthrogram image from the same patient. Note the slight thickening of the anterior band proximally (large arrow) and the undercutting of the attachment of the distal anterior band on the sublime tubercle (small arrows) forming a “T” sign, indicative of the partial tear (B). (Med Epic = medial epicondyle, ST = sublime tubercle)
overhead throwing athletes, especially pitchers, secondary to repetitive micro-trauma in the form of valgus stress (Figure 13). Acute trauma, such as a fall on an outstretched arm, or posterior elbow dislocation, is a less common cause of UCL injury.

On MRI, the UCL is normally homogeneous and hypointense on all sequences. Injury to the UCL is best displayed on coronal MRI images, where it will appear as T2 hyperintense, discontinuous, and ill-defined in shape. While increased signal alone may not be indicative of a tear, signal abnormality with architectural distortion is. Acute injury is also suggested by flexor digitorum superficialis muscle and/or periligamentous edema (Figure 13). Partial tear or sprain of the UCL presents as hypoechoic thickening and/or heterogeneity of the ligament without complete fiber disruption (Figure 14). Ligament laxity, demonstrated with valgus stress maneuvers, without ligamentous disruption may indicate remote injury, partial tear, or acquired laxity in high-level overhead athletes such as baseball pitchers. Complete fiber disruption signifies a full thickness tear. In the acute setting with hemorrhage and edema, it may be difficult to discern full and partial thickness tears. Again, dynamic imaging with valgus stress may reveal separation of torn ligament ends, suggesting complete tear.

Of note, professional or high-level amateur pitchers may demonstrate areas of hypoechogenicity or foci of calcification, ligamentous thickening, and laxity without symptoms, especially during the sporting season (Figure 15).

**Ulnar Nerve Injury**

The ulnar nerve at the medial elbow is prone to injury or entrapment, especially as it passes beneath the cubital tunnel and the retinaculum between the medial epicondyle of the humerus and olecranon process. Etiologies for ulnar nerve injury at the elbow include acute trauma, overuse or repetitive injury, and nerve subluxation or dislocation.

Ulnar nerve entrapment, or cubital tunnel syndrome, is a common elbow pathology. US will reveal an enlarged, hypoechoic ulnar nerve proximal to the cubital tunnel (Figure 16). Within the tunnel, the nerve often normalizes in

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**FIGURE 15.** A 15-year-old amateur baseball pitcher with UCL thickening (arrows) and joint space laxity affecting his right side/pitching elbow (A). Contralateral/left elbow in the same patient demonstrates a normal UCL (arrows) and joint space (B). (ME = medial epicondyle, ST = sublime tubercle)

**FIGURE 16.** Ulnar nerve compression. Ulnar nerve enlargement in short axis (SAX) and long axis (LAX) at the level of the cubital tunnel showing ulnar nerve thickening (thin arrows). In this case, the neuropathy was due to a large impinging bone spur (thick arrow).

**FIGURE 17.** Posterior compartment of the elbow. Normal longitudinal view of the posterior elbow and the olecranon fossa. Note the normal posterior fat pad (arrows) with underlying joint recess. (HS = humeral shaft, T = triceps tendon, T = trochlea, O = olecranon)
size. A maximal cross-sectional diameter of the ulnar nerve > 9 mm sq, or a change in caliber > 2-3 mm sq proximal to or within the cubital tunnel, suggests pathology. Dynamic evaluation may reveal the ulnar nerve dislocating over the medial epicondyle during elbow flexion, with relocation in extension. While this transient dislocation may be seen with ulnar nerve irritation/injury or injury to the overlying Osborn’s ligament, up to 20% of the asymptomatic population will demonstrate this variation.

In contrast, dynamic imaging may also demonstrate dislocation of the medial head of the triceps tendon and the ulnar nerve over the cubital tunnel and medial epicondyle with flexion. This phenomenon is referred to as snapping triceps syndrome and is considered a pathologic entity. MRI can also be used to evaluate the ulnar nerve as it courses through the cubital tunnel. The benefit of US assessment includes superior resolution and magnification of the nerve, as well as the ability to perform a dynamic examination.

Posterior Compartment

Structures of importance in the posterior elbow include the triceps tendon, olecranon bursa and the posterior joint recess. The triceps tendon appears as a short, broad, echogenic fibrillar structure inserting on the olecranon. MRI can also be used to evaluate the triceps tendon as it courses through the cubital tunnel. The benefit of US assessment includes superior resolution and magnification of the nerve, as well as the ability to perform a dynamic examination.

Triceps Tendon Injury

Injury to the triceps tendon or muscle is relatively uncommon and may be post-traumatic, spontaneous, or degenerative. When it is sports related, weight lifters, bowlers, baseball pitchers, and football players are most often affected with post-traumatic injuries. Spontaneous injury to the triceps tendon can be
seen in patients with systemic illnesses, such as lupus, chronic renal failure with secondary hyperparathyroidism, rheumatoid arthritis, and other conditions for which steroid therapy is utilized.4,5

Tears of the distal triceps tendon (DTT) may be full or partial thickness and often involve the combined lateral and long heads. Most tears are incomplete with the short muscular medial head attachment remaining intact.8 Complete tendon disruption and a variable degree of tendon retraction can be seen following a full-thickness tear, with the tendon itself appearing lax and heterogenous (Figure 18). In the setting of radial head fracture, be careful to evaluate for DTT injury, as they occur with similar injury mechanisms.7,8

Olecranon Bursitis and Joint Pathology

Rheumatoid arthritis, gout, infection, and hemorrhage, whether spontaneous or post-traumatic, are potential etiologies of bursal or joint distention with complex-appearing fluid. In patients with gout, evaluation may reveal hyperechoic synovial hypertrophy with internal hyperechoic foci/crystals (Figure 19). Synovial hypertrophy will demonstrate increased color or power Doppler flow, while lack of internal flow on color Doppler suggests complex fluid (Figure 20).8

Injection and Aspiration

In addition to its diagnostic capabilities, US is an ideal modality for image-guided intervention in and around the elbow joint. In the setting of osteoarthritis, inflammatory or crystalline arthropathy, US can guide joint aspiration and steroid injection. When an infectious etiology is suspected, US-guided joint aspiration can confirm diagnosis, allowing for timely treatment.

Dry needling, or repeated lancing of an abnormal area of tendon to incite internal hemorrhage and elicit the consequent inflammatory response, is felt to promote healing and granulation tissue formation. Peritendinous steroid injection may also be performed with US guidance, taking care to not inject steroids into the tendon itself; intratendinous steroid injection increases the likelihood of post-procedure tendon rupture and should be avoided.12

The olecranon bursa is also amenable to injection and/or aspiration when distended with fluid or thickened synovial tissue. Steroid injection should not be performed if septic bursitis is suspected.12

Conclusion

In summary, US is a useful tool in the diagnostic evaluation and treatment of the elbow joint and related pathologies. US is particularly beneficial in evaluating the superficial structures of the elbow including the tendons, muscles and ligaments. The dynamic capabilities of US allow for additional evaluation of the structural integrity of the ulnar collateral ligament, ulnar nerve and triceps tendon. During image-guided intervention, US allows for patient comfort adaptations in addition to providing exceptional anatomic visualization.

MRI examination of the elbow allows for exquisite visualization of the deeper intra-articular structures including the cartilage and bone marrow, as well as the tendons, muscles, and ligaments. However, a benefit of US includes its ability to obtain dynamic imaging of the joint. These principles should be considered when choosing the optimal imaging modality for diagnostic purposes, as well as intervention of the elbow and related pathologies.

References


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Ultrasound and MRI Evaluation of the Lateral Ankle

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Lateral ankle pain is a common clinical complaint and may result from an acute traumatic event, chronic repetitive trauma, impingement syndromes or alignment abnormalities resulting in altered biomechanics. Additional etiologies of ankle pain include synovial proliferative processes, inflammatory arthropathies and crystalline deposition disease. While these articular-based processes typically result in diffuse ankle pain, they may result in localized symptoms and should also be considered in patients with lateral ankle pain. The purpose of this article is to review the common anatomy and pathology of the lateral ankle and to discuss the common imaging findings seen on ultrasound (US) and MRI.

Acute lateral ankle injuries are common and include fractures, ligamentous sprains, and tendinous injuries. Lateral malleolar fractures may occur in isolation or in the setting of bi-or trimalleolar injuries. Lateral ligament complex injuries at the ankle are the most common reason for missed athletic participation, and occur during inversion when the ankle is in the relatively unstable, plantarflexted position.1 Peroneal tendon injuries, including tendinosis, tears, or peroneal retinaculum injuries may occur in isolation but are common in the setting of lateral ligamentous injuries. Entrapment of peroneal tendons between fracture fragments may occur; tendons should be closely interrogated in patients with ankle fractures. In the acute setting, peroneal tendon injuries may be overlooked and underdiagnosed, as pain and laxity from ankle sprain often dominates the clinical picture.2,3

Radiographs are the first line of imaging and should be obtained in weight-bearing, when possible, to evaluate alignment and integrity of the ankle mortise. While radiographs demonstrate most acute osseous injuries well, direct visualization of soft-tissue pathology often requires additional imaging. Both MRI and US can evaluate the ligaments and tendons of the lateral ankle. US has the advantage of assessing tendon motion during dynamic imaging and ligamentous integrity with applied stress; MRI is superior for the evaluation of intra-articular pathology and marrow signal abnormalities.

Osseous Anatomy of the Ankle
The ankle is composed of three main articulations: the tibiotalar (talocrural) joint, subtalar (talocalcaneal) joint, and the transverse tarsal (midtarsal joint) joint. The tibiotalar joint is a synovial hinge joint between the tibial plafond and talor dome and bears most of the load during weight-bearing. Its primary function is dorsiflexion and plantarflexion of the foot, although it aids in inversion/eversion and abduction/adduction as well.4 The subtalar joint consists of three distinct calcaneal articulations and contributes to inversion and eversion of the foot. The transverse tarsal joint, also known as Chopart joint, is a compound joint that includes the talonavicular component of the talocalcaneonavicular joint as well as the calcaneocuboid joint, and assists with inversion and eversion. Collectively, the joints allow for complex motions such as supination (adduction, inversion and plantarflexion) and pronation (adduction, eversion and dorsiflexion) of the foot.4

Acute trauma may result in fractures of the malleoli or fifth metatarsal, which are typically well visualized on radiographs. Fractures of the body or anterior process of the calcaneus, lateral talar process, lateral calcaneus, talor dome and lateral fibula from superior peroneal retinaculum avulsion injuries may be subtle or even occult on radiographs. Advanced imaging is performed in cases when there is a high clinical suspicion for occult fracture, or when more detailed evaluation of the soft tissues is clinically indicated.

Traumatic osteochondral injuries of the talor dome may result in a stable or unstable osteochondral lesion. Symptomatic osteochondral lesions often result in decreased range of motion and cause deep ankle pain with weight-bearing but patients may localize pain to the lateral ankle in the setting of lateral talor dome injuries. MRI is the modality of choice for evaluating osteochondral...
Ultrasound and MRI Evaluation of the Lateral Ankle

injuries as it can detect signs of instability, including a rim of fluid signal intensity surrounding the lesion, cysts underlying the lesion, discontinuities in the subchondral bone plate, or displaced intra-articular fragments (Figure 1).

Ligaments of the Lateral Ankle

The lateral ligaments of the ankle consist of the low ankle ligaments; the anterior talofibular, calcaneofibular and posterior talofibular ligaments; and the high ankle ligaments, the anterior and posterior tibiofibular, and interosseous ligaments (Figure 2). Lateral ankle ligament sprains account for 16% to 21% of sports-related injuries, with a predictable pattern of injury involving the weakest ligament, the anterior talofibular ligament (ATFL), followed by the calcaneofibular ligament (CFL), and finally the posterior talofibular ligament (PTFL).

Low Ankle Ligaments

The ATFL extends from the lateral malleolus to the talus and functions to prevent anterior displacement as well as excessive inversion and internal rotation of the talus on the tibia. The ATFL is well visualized on MR as a hypointense band of tissue seen best on axial images (Figure 3A) and on US as a hypoechoic, linear structure seen best with the transducer in an oblique, short-axis plane (Figure 3B).

The CFL arises from the posterior aspect of the lateral malleolus, courses anteriorly on an oblique long axis, deep to the peroneal tendons, to insert on the lateral calcaneus. It functions to prevent excessive inversion and internal rotation as well as prevent excessive supination. The CFL is stressed most in dorsiflexion and is the second most injured ligament during ankle injuries. On MRI, evaluation of sequential coronal or axial imaging is required to visualize its oblique course from the tip of the lateral malleolus to the lateral calcaneus (Figure 4A). On US, the CFL is well visualized on a long axis with the transducer in an oblique, short-axis plane (Figure 4B).

The PTFL runs from the posterior aspect of the lateral malleolus, with...
variable insertion on the posterolateral aspect of the talus, lateral talar process or os trigonum (if present) and protects the talocrural joint from excessive inversion and internal rotation. It is the least injured low ankle ligament. The PTFL is typically well visualized on axial MR images but can also be seen well in cross-section on sagittal MR images (Figure 5), which may be helpful in assessing the architecture of the ligament in cases of questionable injury on axial images. As the PTFL is injured infrequently, it is not routinely imaged by US, but in cases of clinical suspicion, it can be seen well with the transducer in the short-axis plane over the posterolateral ankle.

Lateral ankle ligament sprains are classified on a three-point grading scale with grade I representing a stretch injury without tear, grade II a partial tear, and grade III a complete tear. Some authors classify low ankle sprains on an anatomic basis, with grade I injuries resulting in partial disruption of the ATFL, grade II involving partial disruption of the ATFL and CFL, and grade III demonstrating complete disruption of the ATFL or CFL. With US or MR imaging, partial thickness injuries demonstrate heterogeneity and thickening of the ligament in the acute period, but are variable in appearance in the chronic setting, with thickening or thinning, ligamentous elongation, or wavy contour.

Complete tears of the ATFL result in a focal gap in the acute setting with
adjacent hyperemia, ligament redundancy and disorganization of fibers on both MR and US (Figure 6). If varus stress is applied to the ankle during the US examination, gapping of the lateral clear space can be visualized.

**High Ankle Ligaments**

The distal tibia and fibula form a syndesmotic joint, composed of three major ligaments: the anterior tibiofibular, posterior tibiofibular, and interosseous (or transverse) tibiofibular ligament, which stabilize the high ankle. Injuries to the high ankle ligaments occur in < 11% of ankle sprains. The anterior tibiofibular ligament is trapezoidal in shape and runs from the anterior tubercle of the distal tibia obliquely to the anterior tubercle of the distal fibula. A thickened distal fascicle of the anterior tibiofibular ligament, termed the anterior inferior tibiofibular or Bassett’s ligament, is present in some individuals and can be visualized on MRI. Given its oblique orientation, the anterior tibiofibular ligament is difficult to visualize fully on a single MR image, but can be seen on axial MR at the level of the distal syndesmosis (Figure 7A). On US, the anterior tibiofibular ligament is easily visualized with the transducer in the short axis oblique position at the level of the distal syndesmosis (Figure 7B).

The posterior tibiofibular ligament is a triangular ligament that runs from the posterior tibial malleolus to the posterior tubercle of the fibula. It is extremely strong and formed by two independent components, superficial and deep, and has fibers that form a broad base at the tibial insertion. The posterior tibiofibular ligament is the least injured high ankle ligament and can be assessed on axial MR images at the level of the distal syndesmosis. The interosseous ligament is at the far inferior aspect of the distal interosseous membrane and is formed by a dense mass of short fibers, which span the tibia to the fibula. Its contribution to ankle stability is controversial, with some claiming it is insignificant and others claiming it is the primary stabilizing bond between the tibia and fibula.

On initial radiographs, widening of the ankle mortise or distal tibiofibular syndesmosis as well as the presence of Weber B or Weber C lateral malleolus fractures should raise concern for high ankle ligamentous injury, as syndesmotic tears occur in approximately 50% of Weber B and in all Weber C fractures. Injuries to the high ankle ligaments are also graded on the standard three-point scale with grade I representing a stretch injury without tear, grade II a partial tear, and grade III a complete tear. On US and MRI, focal discontinuity of the ligament is consistent with a grade III, or complete, tear (Figure 8). Ligamentous thickening, laxity or irregular contour suggests a less severe grade...
Ultrasound and MRI Evaluation of the Lateral Ankle

II injury (Figure 9). Abnormal signal or echogenicity without structural abnormality is consistent with a grade I injury. Surrounding edema or hyperemia on Doppler US interrogation provides insight into acuity, as chronic injuries typically demonstrate irregular morphology without surrounding soft-tissue abnormalities.

Tendons of the Lateral Ankle

The peroneus longus muscle originates from the head of the fibula, and the peroneus brevis muscle originates from the mid-distal lateral fibula and the intermuscular septum. They both course distally in the lateral compartment of the lower leg. At the ankle, the tendons course through the retromalleolar groove of the fibula where they are both within a common peroneal tendon sheath and stabilized by the superior peroneal retinaculum (Figure 10). From there, the peroneus longus tendon courses inferomedially along the plantar foot to insert on the lateral plantar aspect of the first metatarsal base and stabilized by the superior peroneal retinaculum (Figure 10). The peroneus brevis functions primarily in eversion of the foot, but also assists with ankle plantarflexion.

FIGURE 7. Normal anterior and posterior tibiofibular ligaments on axial T1-weighted MRI (A, arrows), seen as linear hypointense bands of tissue at the distal tibiofibular syndesmosis. Normal anterior tibiofibular ligament on US (B, arrows), seen as a linear, fibrillar hypoechoic band of tissue at the distal tibiofibular syndesmosis.

FIGURE 8. Acute high ankle sprain with a grade III injury to the anterior tibiofibular ligament, seen on axial T1-weighted (A) and fat-saturated T2-weighted (B) axial MR images. The anterior tibiofibular ligament is focally disrupted at its fibular attachment (arrows) and there is surrounding edema on fat-saturated, T2 axial images. The posterior tibiofibular ligament is intact (interrupted arrows).

FIGURE 9. Acute high ankle sprain with grade II injury to the anterior tibiofibular ligament on US, seen as thickening of the ligament (arrows) with central architectural distortion and hypoechogenicity (asterisks), compatible with partial ligamentous disruption.
Pathology

Peroneal tendon pathology may result from acute injury or chronic repetitive trauma. Pathologic entities include tendinosis, tenosynovitis, tendon tears and injury to the superior peroneal retinaculum (SPR), which may predispose a patient to tendon subluxation or dislocation.3

MR imaging has been described as the “gold standard” for imaging peroneal tendon pathology, and is a well-established technique to visualize fixed subluxation/dislocation of the peroneal tendons.3,6,13,14 Normally, peroneus longus is anterior and slightly lateral (or superficial) to the peroneus brevis, which is more posterior
and medial, abutting the posterior cortex of the distal fibula. The supramalleolar tendons are relatively equal in size on axial MR and short-axis US (Figure 11). Normal peroneal tendons appear hypointense on MRI, as oval echogenic structures on short-axis US (Figure 12A), and demonstrate linear, fibrillar echotexture on long-axis US (Figure 12B).

Tenosynovitis is seen as T2-hyperintense material on MR or anechoic material on US contained within the tendon sheath, surrounding intact tendons (Figure 13). Tendinosis is seen
as thickening of the intact tendons, with intermediate T2 signal intensity on MRI and hypoechochogenicity and architectural distortion on US and can range from mild tendinosis (Figure 14) to severe tendinosis with early intrasubstance tearing (Figure 15). Longitudinal split tears, most commonly affecting the peroneus brevis tendon, are seen as an anechoic or hypoechoic linear defect paralleling the tendon fibers on long-axis imaging, or can be demonstrated by separating the tendon into two distinct components on short-axis imaging (Figure 16). The presence of three distinct tendons on short-axis US or axial MR images is diagnostic of a longitudinal split tear. Full thickness tears are much less common but may occur in tendons with longstanding or severe tendinosis. Occasionally, a full thickness tear of the peroneus longus tendon may be detected radiographically by noting proximal displacement of an os peroneum (Figure 17).

SPR tears and resultant peroneal tendon subluxation/dislocation are also well visualized by US. Peroneal tendon subluxation is most commonly found in athletes, resulting from disruption of the superior peroneal retinaculum (SPR), and is often associated with dorsiflexion of the ankle and concomitant eversion or inversion of the foot.3,14 Although the retinaculum is not directly visualized radiographically, an avulsion fracture of the lateral, distal fibular cortex may be identified, indicating SPR deficiency.
Ultrasound and MRI Evaluation of the Lateral Ankle

**Figure 18.** Frontal (A) and oblique (B) radiographs demonstrating a superior peroneal retinaculum avulsion fracture of the lateral malleolar cortex (arrows), predisposing the patient to peroneal tendon pathology, including tendon subluxation/dislocation.

**Figure 19.** Short-axis US demonstrating dislocation of the peroneal tendons (PB, PL), which are positioned superficial and lateral to the distal fibular cortex (A). Even though the SPR is not directly visualized in this image, it is disrupted, allowing for the lateral and anterior displacement of the tendons. A normal short-axis image at this level is included (B) for direct comparison.

**Figure 20.** Multiple static sonographic images during ankle circumduction (A) demonstrating intrasheath subluxation of the peroneal tendons in which peroneus longus and brevis tendons reverse their normal positions in the common peroneal tendon sheath but remain within the retromalleolar groove. Normal tendon motion is demonstrated for comparison (B).

Peroneal tendon subluxation is relatively uncommon, occurring in 0.3% to 0.5% of traumatic ankle injuries, and is frequently masked by other ankle pathologies or misdiagnosed as an ankle sprain. However, accurate diagnosis of pathologic peroneal tendon subluxation is critical, as conservative measures are often inadequate and surgery is commonly required for complete return of function and symptom resolution.

MRI and US may directly demonstrate disruption of the superior peroneal retinaculum and resultant fixed subluxation/dislocation of the peroneal tendons (Figure 19). Transient, or dynamic, tendon subluxation or dislocation is more difficult to assess on MR, but US is ideal for assessing dynamic peroneal tendon subluxation, as the tendons can be visualized in real time. During
dynamic US evaluation, the patient performs provocation maneuvers such as ankle dorsiflexion and plantarflexion or circumduction, while the peroneal tendons are imaged in the short axis.\(^1,15,16\) Displacement of the peroneal tendons lateral to the fibular cortex is diagnostic.

A specific subtype of peroneal subluxation is intrasheath subluxation, during which the peroneus longus and brevis tendons reverse their normal position in the common peroneal tendon sheath but remain within the retromalleolar groove, with an intact retinaculum.\(^15\) (Figure 20) Patients with intrasheath subluxation may present with “popping” or “snapping” during ankle circumduction, as the tendons are displaced. It is unknown whether intrasheath peroneal tendon subluxation represents a pathologic entity or is a result of normal supramalleolar peroneal tendon motion. Therefore, it is important to note any symptom reproduction during the dynamic US examination.

**Anatomic Variants**

Aside from traumatic injuries, several anatomic variants of the lateral ankle may predispose a patient to peroneal tendon pathology. Lack of concavity of the distal posterior fibula of the retromalleolar groove may predispose a patient to peroneal tendon dislocation and subluxation.\(^2,14,17\) Additionally, a prominent peroneal tubercle on the lateral calcaneus may predispose a patient to peroneal tendon pathology. Anatomic variations in muscles, such as a peroneus quartus muscle or a low-lying peroneus brevis muscle belly may cause crowding of the tendons in the retromalleolar groove, leading to pathology.\(^2,13\)

**Other Pathology of the Lateral Ankle**

Anterolateral ankle impingement is a distinct entity, often seen in young, athletic patients, and is likely secondary to repetitive microtrauma and microinstability.\(^18\) Over time, microtrauma results in excessive hemorrhage, scar tissue formation and synovial hypertrophy in the lateral gutter of the ankle, eventually leading to impingement. The lateral gutter is defined by the tibia (posteromedially), fibula (lateral), tibiotalar joint capsule, ATFL and CFL (anterolaterally). As there is no associated high-grade ligamentous injury, these patients present with a stable ankle on examination, but
often have decreased dorsiflexion and a palpable soft-tissue mass at the anterolateral ankle.

Another subgroup of patients with anterolateral ankle impingement includes those with an accessory ligament or thickened distal fascicle of the anteroinferior tibiofibular ligament, a normal variant that may be present in 21% to 97% of ankles.9,10,19 In these patients, a prior lateral ankle sprain results in ligamentous instability, which allows for anterior extrusion of the talar dome and increased pressure at the anterolateral ankle during dorsiflexion.9 Over time, this leads to synovial hypertrophy and impingement of soft tissues between the anterolateral talus and the accessory anteroinferior tibiofibular ligament. Both MRI and US can identify synovial/capsular hypertrophy in the anterolateral gutter, seen as heterogenous synovial mass on US and T1/T2 intermediate- to low-signal synovial hypertrophy and scar tissue in the anterolateral gutter on MRI (Figure 21).

Extra-articular soft-tissue and osseous impingement may also occur at the lateral ankle. Talocalcaneal and subfibular impingement may occur in the setting of pes planovalgus secondary to lateral shift of weight-bearing forces from the talar dome to the lateral talus and fibula (Figure 22).20 Eventually, progressive deformity leads to secondary osteoarthritis of the subtalar, talonavicular, and calcaneocuboid joints. MRI can provide detailed information about intra-articular pathology such as osteochondral lesions, osteoarthritis, and marrow edema-like signal in the setting of acute trauma and contusion.

Finally, articular-based processes such as synovial chondromatosis, pigmented villonodular synovitis, and inflammatory arthropathies, as well as crystalline deposition diseases such as gout and pseudogout, may be seen in the ankle. Although these often result in diffuse symptoms, they can present with lateral ankle pain if disease is asymmetric or focal.

Conclusion

Lateral ankle pain most commonly results from osseous, ligamentous or tendinous injury, but other etiologies should be considered in the nontraumatic setting. Although radiographs are an important first line of imaging, MRI and US can provide important diagnostic information regarding soft-tissue pathology around the ankle and should be considered in patients with lateral ankle pain.

References

Case Report: Kim, Scher, Davis

Bone Destruction and Soft-tissue Masses of the Knee

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Case Presentation
A 56-year-old African American man presents with knee pain and palpable masses.

FIGURE 1. Lateral (A) and AP (B) radiographs of the left knee demonstrate well-margined erosions in the distal femur, proximal tibia and inferior patella. High-density, soft-tissue masses around the knee and a dense suprapatellar joint effusion are also present.

FIGURE 2. Sagittal T2-weighted (A) and coronal T1-weighted (B) MRI images demonstrate multifocal erosions involving the femoral condyles, proximal tibia and patella (arrows) with multiple heterogeneously T2 hyperintense and T1 hypointense soft-tissue masses within the osseous structures, juxta-articular soft tissues and the prepatellar bursa (asterisks).
Differential Diagnosis

Gout
- Calcium pyrophosphate deposition disease
- Rheumatoid arthritis
- Septic arthritis
- Metastases

Discussion

Gout

Gout is a common deforming inflammatory polyarthropathy, the most common crystalline arthropathy, and is associated with significant morbidity.\(^1\)\(^2\) It most commonly affects middle-aged to elderly white men with a 20:1 male to female predilection.\(^1\)\(^3\) When afflicted, women are typically postmenopausal.\(^1\)\(^3\)

Clinical presentation includes intermittent acute joint pain with associated redness and swelling, which may mimic septic arthritis.\(^1\) Gout is often suspected during the clinical presentation. The diagnosis is supported by imaging findings combined with clinical and laboratory findings including hyperuricemia, monosodium urate (MSU) crystal formation (which is negatively birefringent in the aspirate), and/or rapid resolution of symptoms following colchicine administration.\(^1\)

Radiographic findings of gout are pathognomonic and include well-defined juxta-articular erosions that may extend outward secondary to reactive new bone formation to produce “overhanging edges.”\(^1\)\(^3\)\(^4\) In order to cause erosions, tophi must be present over an extended period, which differs from the earlier onset bone destruction seen with more aggressive processes such as septic arthritis.\(^3\)\(^4\) When intraosseous tophaceous deposition occurs, it may demonstrate a more destructive appearance, emulating infection.

Distribution is often polyarticular and asymmetric, typically involving small joints.\(^1\)\(^5\) The feet are most commonly involved with the greatest predilection for the first metatarsophalangeal (MTP) joint.\(^1\)\(^5\) Other commonly involved joints include the interphalangeal joints of the feet and the interphalangeal and intercarpal joints of the hands. Bone mineral density and cartilage are generally preserved in contrast to other entities such as rheumatoid arthritis (RA) or septic arthritis, thereby generally preserving the joint space until later in the disease course.\(^3\)\(^5\) Larger joints are less commonly afflicted, but when involved, the surrounding bursa (ie., prepatellar or olecranon bursa) are often affected.

Dual-energy CT and MRI may play a role when the presentation is atypical and can help demonstrate the soft tissue burden of the disease, but they should not be routinely used to diagnosis gout. On MRI imaging, tophi will demonstrate intermediate- or low-signal intensity on T1-weighted sequences and can show variable T2 signal intensity.\(^1\) Enhancement patterns of tophi are also variable. Due to this variability, correlation with radiographs and the patient’s clinical presentation is imperative as the MRI appearance is nonspecific and can be misinterpreted as infection or tumor.

Dual-energy CT scanners can acquire information at different energy levels (eg, 140 kVp and 80-100 kVp). The datasets from the low and high x-ray energies contain different information on the x-ray attenuation characteristics of the region imaged, allowing for the creation of a decompensation algorithm of the datasets. This algorithm uses soft tissue as a baseline, and the differences in attenuation between low and high energy allows for accurate and specific characterization and separation of calcium and monosodium urate.\(^5\) While dual-energy CT can be useful in challenging cases, it should not be relied upon routinely to diagnose gout.

Ultrasound (US) may demonstrate underlying bony erosive changes and tophi.\(^1\) The combination of joint effusion, tophus, bony erosions and a double contour sign (hyperechoic MSU crystals layering over hypoechoic hyaline cartilage) is reported as diagnostic in 97% of cases.\(^2\)

CPPD Arthropathy

Calcium pyrophosphate deposition (CPPD) disease is the second most common arthropathy of the knee, predominantly in elderly patients.\(^3\)\(^8\) Clinically, CPPD may be asymptomatic or mimic other entities due to acute, episodic attacks of joint pain.\(^3\)\(^8\) Calcification of hyaline or fibrocartilage, known as chondrocalcinosis, is a hallmark finding in CPPD. Of the imaging modalities, radiographs are the most important when evaluating for CPPD, as diagnosis requires chondrocalcinosis deposition in two or more areas of the skeletal system.\(^1\)\(^3\)

Severe degenerative changes isolated to the patellofemoral compartment in the knee, with associated chondrocalcinosis and preservation of medial and lateral joint space, are highly suggestive of CPPD arthropathy.\(^1\)\(^8\) The crystal deposition inevitably accelerates breakdown of cartilage, resulting in joint space narrowing, osteophyte formation and subchondral cystic change, which can become so severe that CPPD may be mistaken for neuropathic (Charcot) joint. The lack of true erosions differentiates CPPD from gout and rheumatoid arthritis (RA). Preservation of bone mineralization differentiates CPPD from septic arthritis and RA. Extra-articular CPPD may occur focally in the soft tissues presenting as large soft-tissue masses adjacent to any joint.

CT and MRI are not usually obtained in the workup of CPPD arthropathy. US may be helpful in detecting CPPD cartilage deposits, which appear as linear hyperechoic foci within the hypoechoic cartilage, as opposed to hyperechoic foci layering on top of the cartilage as seen with gout.\(^1\)

Rheumatoid Arthritis

RA is a systemic inflammatory disease of unknown etiology that primarily affects the musculoskeletal system. In RA the pathologic process targets the synovium of joints and tendon sheaths with involvement of the adjacent bone, tendons, joint capsule(s) and ligaments.\(^9\) RA is a generally symmetric, polyarticular inflammatory arthropathy
with predilection for the distal extremities. Diagnosis is based on clinical and imaging findings and biochemical markers, such as elevated rheumatoid factor or anti-cyclic citrullinated peptide (anti-CCP/ACPA) antibodies.\(^1\)

Radiographs are the imaging modality of choice for the initial workup. Early radiographic findings include soft-tissue swelling, joint distension, and tenosynovitis, as well as periarticular osteopenia and edema. The hallmark of RA is pannus formation that leads to cartilage loss and the classic marginal erosions at the intracapsular articular margins or “bare areas.” As the disease progresses, osteopenia becomes more generalized and further cartilage loss results in worsening joint space narrowing.\(^1,3\) Joint derangement progresses with ankylosis and/or joint subluxation/ dislocation with eventual end-stage joint destruction.

MRI is excellent for detecting soft-tissue involvement, synovial inflammation and bone marrow edema signal, which are typically present early in the disease course.\(^1,8\) US is an effective modality to detect synovial thickening, active synovitis and early erosions and can be used to monitor treatment response.

**Septic Arthritis**

In adults, joint infections usually result from hematogenous spread. The most common offending nongonococcal infectious agent is *Staphylococcus aureus*. Initial clinical presentation includes acute onset of monoarticular joint pain and swelling.\(^9\) Laboratory evaluation typically shows elevated white blood cell count, erythrocyte sedimentation rate, and C-reactive protein levels. Urgent arthrocentesis should be performed to identify causative agent(s) and exclude other entities with similar clinical presentations such as crystalline and inflammatory arthropathies.\(^10\) Septic arthritis in a native joint is a medical emergency and requires antibiotic administration as well as surgical washout within 24-48 hours. If untreated, the proteolytic enzymes in the bacteria can lead to permanent joint destruction and loss of function.

Conventional radiographs may be normal initially, or may demonstrate a joint effusion, juxta-articular osteopenia, or early erosive change with indistinctness of the cortex. Over time, as the cartilage and bone are destroyed, radiographs may demonstrate joint space narrowing and progressive bone destruction.

US allows for real-time imaging guidance for joint aspiration to facilitate diagnosis and treatment. CT and MRI do not play a role in imaging septic arthritis, as the diagnosis should be based on the patient’s clinical picture, with emergent aspiration performed prior to any advanced imaging.

**Metastatic Disease**

Osseous metastases are common in the adult population and may be purely lytic, purely sclerotic, or mixed lytic/sclerotic in appearance. Most bone metastases occur in areas rich in vascularized red marrow such as the skull, spine, ribs, pelvis and proximal long bones.\(^11\) Since the small bones of the hands and feet are filled with fatty marrow, metastatic lesions are rarely found in these locations.\(^11\)

Lytic osseous metastases generally have ill-defined margins and adjacent periosteal reaction, in contrast to the well-defined sclerotic margins common in benign bone lesions or in the setting of erosive/crystalline arthropathy.\(^11\) Malignant bone tumors, whether primary or metastatic, may extend into a joint, but almost never begin in an intra-articular location.

**Diagnosis**

Tophaceous gout of the knee

**Summary**

Extensive osseous articular erosions with associated juxta-articular soft-tissue masses on radiographs may be caused by several entities, but attention to key radiographic characteristics and disease distribution often aids diagnosis, although correlation with clinical history and tissue sampling provides confirmation. The well-defined erosions, preserved bone mineralization, and surrounding increased soft-tissue density seen in this case help differentiate gout from other arthritides, as well as from more aggressive etiologies including septic arthritis and metastatic disease.

**REFERENCES**

Solitary Lytic Bone Lesion

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Case Presentation

A 66-year old woman presented with a four-year history of left lower extremity pain that began after she had been diagnosed with a left lower-extremity venous stasis ulcer. The ulcer had healed but the pain had acutely worsened over the previous three months. Pertinent medical history included stroke, antiphospholipid antibody syndrome, and end-stage renal disease on hemodialysis. Her physical examination was unremarkable other than pain with weight bearing. The patient had previously undergone two CT-guided biopsies at outside hospitals, both of which were nondiagnostic.

FIGURE 1. Initial presentation frontal radiograph of the lower leg demonstrating a mottled appearance of the midfibular diaphysis without pathologic fracture (arrows).

FIGURE 2. Concurrent axial CT image in bone window (A) and soft-tissue window (B) shows cortical scalloping of the midfibular diaphysis (A, arrows) with mild inflammatory change in the superficial tissues (B, asterisk) and within deep fascial planes, but without discrete soft-tissue mass.

FIGURE 3. Repeat frontal radiograph three months later of the lower leg demonstrates progressive permeative destruction of the midfibular diaphysis (arrows).
Case Report: Wallace, Davis, Scher

FIGURE 4. Noncontrast T1-weighted image demonstrates destructive and permeative lesion at the midfibular diaphysis (arrows). Axial (B) and sagittal (C) STIR-weighted images demonstrate soft-tissue and muscle edema (arrows) adjacent to the midfibular diaphyseal lesion without discrete soft-tissue mass. Contrast was not administered secondary to patient’s decreased renal function.

Key Imaging Finding(s)
Lytic, destructive lesion of the fibula

Differential diagnoses
- Infection (osteomyelitis)
- Metastatic disease
- Brown tumor
- Gorham’s disease
- Peripheral vascular malformation

Discussion

Infection (Osteomyelitis)
In adults, osteomyelitis typically occurs via hematogenous spread or direct extension from an overlying ulcer or trauma. As the vascular supply to bone changes with age, so does the typical location of osteomyelitis. Osteomyelitis tends to affect the metaphyses in children and the epiphyses in adults. The most common pathogen in osteomyelitis is *Staphylococcus aureus*, even in patients with sickle cell anemia who are at increased risk for salmonellosis osteomyelitis. Bacterial infection results in a local inflammatory response in and around the affected bone.

On initial radiographs, there may be no findings in the initial 10-14 days in adults. Once present, radiographic findings of osteomyelitis include regional osteopenia, cortical bone loss, periosteal thickening, and eventual peripheral sclerosis as the infected bone begins to heal. MRI is the most sensitive modality for detecting osteomyelitis and is more specific than bone scintigraphy. Findings include low T1 and high T2/STIR signal of the affected bone and postcontrast enhancement of the bone, periosteum, and surrounding soft-tissue collections.

Metastatic Disease
Metastatic lesions to bone are far more common than primary malignant bone lesions, often arising from malignancies of the lung, breast, kidney, and prostate. Myelomatous lesions should also be considered in patients over age 40 and typically present as punched out lytic lesions. The most common route of spread of metastatic disease is hematogenous, and common locations include ribs, vertebrae, the pelvis, and ends of long bones. Symptomatic lesions can present with pain and pathologic fracture. Usually, the primary malignancy is known at the time of diagnosis of osseous metastatic disease, but bone biopsy can be performed to provide definitive diagnosis.

The radiographic appearance of skeletal metastases can vary. Lesions may be lytic, sclerotic, or mixed lytic and sclerotic. It is important to note that radiographs are insensitive for the detection of asymptomatic osseous metastatic disease. Bone scintigraphy can also be used to detect skeletal metastases, can conveniently evaluate the entire skeleton at one time, and is very sensitive for the detection of osseous metastases. Usually, there is increased radiotracer uptake by the metastatic lesion. An exception is when a lesion is purely lytic, which will produce a photopenic defect on the bone scan. It is helpful to correlate scintigraphic findings with findings on other imaging modalities to exclude benign causes.

CT provides detailed information about cortical and trabecular bone involvement. MRI is highly sensitive and specific for detecting metastatic lesions to the bone marrow (90% and 95%, respectively) before cortical destruction occurs, and can evaluate extent of surrounding soft-tissue involvement. Additionally, PET/CT imaging can be performed to evaluate for metastatic disease that is F-18 fluorodeoxyglucose (FDG) avid.

Brown Tumor
Chronic renal disease causes excessive urinary excretion of calcium, thereby lowering serum calcium levels. To maintain normal serum calcium levels, parathyroid hormone levels increase and cause calcium resorption
from bone. Brown tumor forms when granulation tissue with active, vascular proliferative fibrous tissue replaces the bone marrow.5

Radiographic findings include a well-circumscribed lucent lesion, sometimes with cortical thinning and expansion but no cortical break. MRI appearance can vary depending on tumor components, which can be entirely solid, entirely cystic, or mixed cystic and solid. Cystic components will be T2 hyperintense, and solid components will be T1 and T2 hypointense. Solid components can show postcontrast enhancement.6

**Gorham’s Disease**

A rare skeletal disease of unknown cause, Gorham’s disease is also called Gorham-Stout syndrome, progressive massive osteolysis, or vanishing bone disease. Disease manifests with progressive osteolysis and proliferation of thin-walled vascular and lymphatic channels. The process typically begins in one bone and can spread to adjacent bones and soft tissues. Diagnosis is made by exclusion of more aggressive osseous lesions, exclusion of underlying infection and the presence of angiomatous tissue on biopsy.7,8

Radiographic appearance is a lytic lesion with progressive, often circumferential osteolysis secondary to osteoclastic hyperactivity with resorption of the affected bone. The vascular and lymphatic channels often result in a permeative appearance of the bone. There is no osteoblastic activity in this process, so there is no periosteal reaction. This process typically plateaus and stabilizes over time.7,8

**Vascular Malformations**

Vascular malformations develop from dysplastic vascular channels and do not involute over time. They are classified based on the predominant vessel type and subcategorized as high- or low-flow lesions. Radiographic findings of erosive changes, periosteal reaction, cortical scalloping, or pathologic fracture could indicate bony involvement.9 Venous malformations account for two-thirds of all vascular malformations and 40% of venous malformations occur in the extremities.10

MRI combined with MR angiography is best for evaluating vascular malformations to determine the extent of the lesion as well as involvement of adjacent structures. MRI appearance varies depending on the characteristics of the vascular malformation. Venous and lymphatic malformations are usually septated, lobular T1 hypointense and T2 hyperintense masses with fluid-fluid levels and no flow voids. Arteriovenous malformations demonstrate no well-defined mass but instead demonstrate enlarged feeding arteries and draining veins with flow voids.10

**Diagnosis**

Gorham’s disease. A third biopsy was performed under CT with samples sent for culture and surgical pathology. Histologic evaluation demonstrated skeletal muscle and tissue fragments composed of well-formed capillary and venous spaces with rare multinucleated giant cells but no evidence of malignancy or infection. Over the subsequent three months, the pain in the patient’s lower leg began to subside.

**Conclusion**

The broad differential for solitary lytic bone lesions can be narrowed based on patient age and lesion location. Radiographic appearance can also help narrow the differential by noting the presence of cortical thinning, cortical breakthrough, sclerosis, and periosteal reaction. MRI with and without contrast may also further narrow the differential; however, biopsy is important to exclude osteomyelitis and malignancy.

**References**

Hydroxyapatite Deposition Disease of the Lateral Collateral Ligament

A 35-year-old man presented with worsening left lateral knee pain, nonresponsive to conservative treatment. On examination, he had joint effusion, decreased range of motion, and lateral joint line tenderness.

AP and internal oblique radiographs (Figures A, B) demonstrated a 3.3-cm calcification (arrows in A, B) near the expected origin of the lateral collateral ligament (LCL). Fat-saturated T2- and nonfat-saturated T1-weighted MR images (Figures C, D) demonstrate focal hypointensity (arrows in C, D) in the proximal LCL with surrounding soft-tissue, edema-like signal.

Hydroxyapatite deposition disease (HADD) results from calcium hydroxyapatite crystal deposition in periarticular soft tissues, often in areas of decreased vascularity. The rotator cuff is most commonly affected, but it can also be seen in other tendons, ligaments, and joints, including the longus coli muscle/tendon. Intra-articular deposition results in accelerated degenerative disease.

The presentation is variable, but three phases have been described. In the initial silent phase, calcium is contained within the tendon/ligament, seen as amorphous high density on radiographs with minimal symptomatology. During the mechanical phase, the deposits enlarge and liquefy, seen as poorly defined areas of increased density on radiographs, and the patients have acute, often severe pain. Eventually, the calcium disperses into the adjacent soft tissues/bursa and is resorbed. If resorption fails to occur, calcium crystals may persist in the chronic phase, characterized by mature calcification on radiographs and pain and decreased range of motion clinically.

Initial treatment is conservative but if symptoms persist, bursal steroid injections, ultrasound-guided barbotage, extracorporeal shockwave therapy or surgical resection may be performed.

References

Painful Os Peroneum Syndrome (POPS)

A 62-year-old man with chronic lateral foot pain presented with acutely worsening pain and swelling at the base of the fifth metatarsal after feeling a popping sensation while walking down the stairs.

A lateral foot radiograph (Figure A) demonstrates an os peroneum (OP) (asterisks in A) with subtle areas of mineralization in the region of the inframalleolar peroneus longus tendon (arrows in A). Sagittal T1- (Figure B), sagittal fat-saturated T2- (Figure C), and coronal fat-saturated T2-weighted images (Figure D) demonstrate T1 hypointensity and T2 hyperintensity within the OP (asterisks in B, C) and a partial, intrasubstance tear of peroneus longus (arrows in D).

The OP is located in the substance of the peroneus longus tendon at the level of the calcaneocuboid joint and is ossified in approximately 20% of patients.1 Patients with painful os peroneum syndrome (POPS) present with plantar/lateral foot pain, localized along the course of the peroneus longus. Pathology may involve the OP (fracture or diastasis of multipartite OP), the peroneus longus tendon (tenosynovitis or tendon tear), or a large peroneal tubercle, which entraps the peroneus longus or OP during motion.2

Initial radiographs may demonstrate fragmentation, fracture or proximal retraction of the OP. Further evaluation with MR is often performed, which may demonstrate marrow edema within the OP (with or without a discrete fracture line). Additional findings often include tendinosis, tenosynovitis, or tearing of the peroneus longus tendon.

Treatment for POPS begins with conservative management and may progress to surgical excision of the OP. Repair, debridement or tenodesis of the peroneus longus tendon may also be required.

REFERENCES
Psoriatic Arthritis

A 28-year-old man presented to his primary care physician for hand and foot joint pain. Radiographs of the hands (Figures A, B) and feet (Figures C, D) were obtained. They demonstrate diffuse soft-tissue swelling of the digit (A) as well as marginal joint erosions (red arrow in A) and periostitis of the proximal and middle phalanges (white arrows in A). There is increased periosteal and endosteal bone formation in the middle and distal phalanges (red arrows in B). Other digits demonstrate late stage marginal erosions resulting in pencil in cup deformity (red arrow in C). There was a retrocalcaneal erosion (red arrow in D) with insertional Achilles enthesitis (white arrow in D).

Psoriatic arthritis is a seronegative spondyloarthropathy that manifests in up to 30% of patients with psoriasis. Occasionally, the only clue to the diagnosis of psoriasis may be the imaging features, as the characteristic skin rash does not have to be present. Differentiating spondyloarthropathies can be challenging but knowledge of key differences can lead radiologists to the correct diagnosis. The radiographic features of psoriatic arthritis include dactylitis (“sausage digit”); marginal bone erosions (“pencil in cup”); bone proliferation including “ivory phalynx;” normal bone mineralization; enthesitis; and asymmetric bilateral distribution most commonly involving the hands, feet, and sacroiliac joints.

In contrast to rheumatoid arthritis, psoriatic arthritis should have normal mineralization, bone proliferation, and more prominent findings in the interphalangeal joints.

References