JAOCR
Official Journal of the American Osteopathic College of Radiology

Aims and Scope
The Journal of the American Osteopathic College of Radiology (JAOCR) is designed to provide practical up-to-date reviews of critical topics in radiology for practicing radiologists and radiology trainees. Each quarterly issue covers a particular radiology subspecialty and is composed of high-quality review articles and case reports that highlight differential diagnoses and important teaching points.

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Sara Boyd, D.O., Julia Cameron-Morrison, D.O., Timothy McKnight, D.O.
I would like to thank Dr. Wale and the JAOCR for allowing me to serve as guest editor for this issue. Dr. Wale deserves high praise for his seamless transition into the JAOCR editor-in-chief position from the exceptional Editor Emeritus Dr. William O’Brien.

This JAOCR issue is unique in that it highlights the Annual Exhibits from the 2018 AOCR Annual Meeting. This meeting was an enormous success providing excellent educational content and social interaction for everyone who attended. Authors of the top Annual Exhibits from the conference were invited to submit to this issue, which highlights the involved residents and their institutions.

The first review article focuses on musculoskeletal radiology and examines the imaging findings of carpal instability. Drs. Treiman, Strle, and Matthews wrote an excellent review that will assist radiologists in understanding the fundamental wrist anatomy and common patterns of injury.

Our second review article covers hindfoot pain and the MRI findings associated with the differing etiologies. Heel pain is a common complaint among the general population and is imaged frequently. Our goal is to assist the general radiologist in moving beyond the basic diagnosis of hindfoot pain to understand the more uncommon etiologies and their associated MRI appearance. This was written by my colleagues Drs. Cheney, Smith, and Brooks at the OSU Center for Health Sciences.

Our last review article is on cavitary diseases of the lungs. Drs. Cho, Lee, and Vassiliou review the differential diagnosis and imaging findings of pulmonary cavities. The foundation of this Annual Exhibit was the common mnemonic “CAVITY,” which is used as a guide to review the differing etiologies and how they present on imaging.

Our Viewbox articles were based on the Annual Exhibits presented by the radiology residents and faculty at the Beaumont Farmington Hills Department of Radiology. The residents Drs. Boyd and Cameron-Morrison were instrumental in putting together the articles, Parotid Gland Myxofibrosarcoma and Fibroma of the Tendon Sheath.

I am thankful for all the authors and their essential contributions. My hope is that our issue entices radiology residents, faculty, and practicing radiologists to attend the AOCR meetings in the future and submit their research and educational material for future Annual Exhibits.

Last and most importantly, thank you to my wife for her steadfast love and support. Without her I’d be lost in the woods.
Carpal Instability: Clarification of the Most Common Etiologies and Imaging Findings

Intrinsic ligament injuries of the wrist are common with varying degrees of scapholunate ligament tears occurring in more than one-third of involved wrists.1 A fundamental understanding of wrist anatomy and common patterns of injury help increase early detection and proper management of carpal instability.

Plain film radiographs offer an adequate cursory evaluation of carpal instability and exclude other entities that may mimic instability such as fracture. Additionally, radiographs display the alignment of the wrist and carpal bones (Figure 1). Ensuring proper positioning of the wrist (ie, frontal and lateral projections) allows for appropriate assessment of the joint spaces and carpal arcs2 as well as initial screening for acute fracture or dislocation.

MRI provides a more thorough evaluation for underlying pathology and associated sequelae3,4 (Figures 2). Incorporating an appropriate protocol for MRI of the wrist (Table 1 and 2) offers exquisite detail of the bones, cartilage, ligaments, tendons and nerves. At the authors’ institution, this includes axial proton density-weighted sequences with and without fat saturation, coronal T1-weighted images, coronal proton density-weighted images with fat saturation, and coronal 3D double echo steady state as well as sagittal proton density-weighted images with fat saturation.

Carpal Instability Classification
The first step toward accurate wrist radiography is to ensure proper anatomic positioning. In the frontal projection, the carpal bones should be parallel with undisrupted arches, normal in shape (implying normal tilt and axis), and equally spaced. The shape of the lunate, capitate, and scaphoid requires close attention as they are the most common carpal bones to be malaligned.5 The lateral projection is also critical, particularly in determining the alignment and/or angulation of the capitate, lunate, scaphoid and radius, and is critical in evaluating intercalated segment instability.6

Overall classification of carpal instability is separated into 4 large groups (Table 3).3 Within this classification scheme, the most common etiologies include dorsal intercarpal (DIC) and dorsal perilunate (DPL) dislocations. These common clinical entities have specific radiographic and MRI characteristics.

Carpal Instability Dissociative
These injuries include scapholunate dissociation, scapholunate advanced collapse, scaphoid nonunion advanced collapse, and the less common lunotriquetral dislocation, which can range from incomplete tears to complete dissociation.7 These entities can be adequately evaluated and diagnosed with radiography; however, specific ligamentous injuries and extent of degenerative disease is better appreciated with MRI.

Dorsal intercalated segment instability (DISI) and volar intercalated segment instability (VISI) are the most common patterns of carpal instability and are associated with scapholunate and lunotriquetral ligament injuries, respectively.8 They can be suggested on radiographic evaluation with typical findings and abnormal angulation of the carpal bones, but are not always evident even in the setting of ligamentous injury, which is better seen by MRI.9 Dorsal perilunate dislocations can be separated into 4 stages of injury with progressive perilunate instability occurring secondary to ligamentous injuries. These stages of instability include scapholunate dissociation, perilunate dislocation, midcarpal dislocation, and
lunate dislocation. Each stage has a differing radiographic appearance allowing accurate diagnosis with specific ligamentous injury, well visualized by MRI.\(^\text{10}\)

Carpal instability dissociative (CID) involves injury within or between bones of the same carpal row. Most commonly, this instability pattern occurs in the proximal carpal row as a result of scapholunate or lunotriquetral ligament injury and the different specific type of injuries include scapholunate dissociation, dorsal intercalated segment instability, scapholunate advanced collapse, scaphoid nonunion advanced collapse, and lunotriquetral dislocation.\(^\text{11}\)

**Scapholunate Dissociation**

Scapholunate dissociation (SLD) is disruption of the ligamentous connection between the scaphoid and lunate (Figures 3). This is seen on radiography as diastasis of the scapholunate interval

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**FIGURE 1.** Proper wrist position and alignment. Frontal (A) and lateral (B) radiographs of the wrist demonstrate normal parallel carpal arcs (orange lines) on the frontal view. On the lateral view (B), there is proper alignment of the radius (orange arrow), lunate (blue arrow), and capitate (white arrow).

**FIGURE 2.** Normal MRI of the wrist demonstrating protocol utilized at the authors’ institution. Axial proton density (PD)-weighted (A), axial fat-saturated (FS) PD-weighted (B), coronal FS PD-weighted (C), coronal T1-weighted (D), and sagittal FS PD-weighted (E) provide exceptional anatomic detail and clarification of suspected carpal instability.
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with a gap of > 3mm (‘‘Terry Thomas’’ or ‘‘David Letterman’’ sign). This is the most frequent carpal instability pattern and can be isolated or associated with scaphoid fractures.

Dorsal Intercalated Segment Instability

Dorsal intercalated segment instability (DISI) involves injury to the scapholunate ligament and concomitant failure of the scaphoid stabilizers, which often results in permanent carpal malalignment. In this pattern, the lunate is dorsiflexed and the scaphoid is tilted volarly with a scapholunate angle > 60˚ (normal range is 30˚ to 60˚) and a capitolunate angle > 30˚, as measured on a lateral radiograph (Figure 4).

Scapholunate Advanced Collapse

Scapholunate advanced collapse, commonly abbreviated as ‘‘SLAC wrist,’’ occurs with degenerative joint disease centered at the radioscaphoid joint from chronic SLD. There are 3 progressive stages of SLAC wrist: stage I, which involves radial styloid and scaphoid degeneration; stage II (Figure 5), which involves degeneration between the scaphoid and the entire scaphoid facet of the radius; and stage III (Figure 6), which involves degeneration between the capitate and lunate. The hallmark of a SLAC wrist is

Table 1. Example of MRI Wrist Protocol

| Arthrogram | Axial T1FS, Coronal T1FS, Coronal T2FS, Coronal STIR, Sagittal T1FS, Sagittal T2FS |
| Non-Arthrogram | Axial proton density fat suppression (PD FS), Axial PD, Coronal PD FS, Coronal T1, Coronal 3D, Sagittal PD FS |

Table 2. Routine MR Parameters for Select Sequences on 1.5 T Imaging of the Wrist

<table>
<thead>
<tr>
<th>T1</th>
<th>T2</th>
<th>STIR</th>
<th>PD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repetition time</td>
<td>400-700*</td>
<td>4000-7000*</td>
<td>5275</td>
</tr>
<tr>
<td>Echo time</td>
<td>14</td>
<td>68</td>
<td>42</td>
</tr>
<tr>
<td>Echo train length</td>
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<td>20</td>
<td>13</td>
</tr>
<tr>
<td>Field of view</td>
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<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Slice thickness</td>
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<td>3 mm</td>
<td>3 mm</td>
</tr>
<tr>
<td>Gap thickness</td>
<td>0.5 mm</td>
<td>0.5 mm</td>
<td>0.5 mm</td>
</tr>
</tbody>
</table>

* Auto adjust. Key: PD = proton density, STIR = short-T1 inversion recovery

Table 3. Mayo Classification of Carpal Instability

<table>
<thead>
<tr>
<th>Type of Instability</th>
<th>Features of Instability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpal instability dissociative (CID)</td>
<td>Derangement within or between carpal bones in the same row</td>
</tr>
<tr>
<td>Carpal instability nondissociative (CIND)</td>
<td>Derangement between the radius and proximal carpal row or between the proximal and distal carpal rows</td>
</tr>
<tr>
<td>Carpal instability complex (CIC)</td>
<td>Features of both CID and CIND</td>
</tr>
<tr>
<td>Carpal instability adaptive (CIA)</td>
<td>Injury proximal or distal to carpal bones causing instability</td>
</tr>
</tbody>
</table>

FIGURE 3. Scapholunate dissociation. Axial T1-weighted (A) and coronal fat-saturated proton density-weighted (B) MRI, and frontal radiograph (C) of the wrist demonstrating scapholunate dissociation. Tear of the dorsal component of the scapholunate ligament (orange arrow) and the proximal zone (white arrow) in the setting of a scapholunate dissociation. Widening of the scapholunate interval (asterisk) is noted on plain film.
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Table 4. Groups of Carpal Instability Complex (CIC)

<table>
<thead>
<tr>
<th>Group #</th>
<th>Pattern of Instability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Dorsal perilunate dislocation (lesser arc injury)</td>
</tr>
<tr>
<td>Group 2</td>
<td>Dorsal perilunate fracture-dislocation (greater arc injury)</td>
</tr>
<tr>
<td>Group 3</td>
<td>Palmar perilunate dislocation (lesser or greater arc injury)</td>
</tr>
<tr>
<td>Group 4</td>
<td>Axial dislocation</td>
</tr>
<tr>
<td>Group 5</td>
<td>Isolated carpal bone dislocation</td>
</tr>
</tbody>
</table>

**FIGURE 4.** Dorsal intercalated segmental instability. Lateral radiograph of the wrist demonstrating dorsal intercalated segmental instability (DISI). Intersecting axes of the lunate and capitate (orange lines) form a 43° capitolunate angle (normal is < 30°).

**FIGURE 5.** Stage II scapholunate advanced collapse. Coronal T1-weighted image depicting a stage II scapholunate advanced collapse (SLAC) wrist. There is radioscaphoid degenerative joint disease (orange arrow) with subchondral sclerosis, chondromalacia, and joint space narrowing. There is also a proximal zone scapholunate ligament tear resulting scapholunate interval widening (asterisk). Note the lack of significant degeneration of the capitolunate joint.

**FIGURE 6.** Stage III scapholunate advanced collapse. Frontal radiograph depicting a stage III scapholunate advanced collapse (SLAC) wrist. There is radioscaphoid degenerative joint disease involving the entire scaphoid facet of the radius (orange arrows) with subchondral sclerosis, cartilage thinning, and joint space narrowing. There is also scapholunate interval widening (asterisk) and arthrosis at the capitolunate joint (blue arrow).

**FIGURE 7.** Scaphoid nonunion advanced collapse. Coronal T1-weighted image depicting a scaphoid nonunion advanced collapse (SNAC) wrist. Nonunion fracture of the scaphoid (orange arrow) with associated radioscaphoid degenerative change (white arrow) and radioscaphoid interval widening (blue arrow). There is diffuse hypointense signal of the scaphoid proximal pole (asterisk) consistent with osteonecrosis.

**FIGURE 8.** Ulnar translocation. Lateral (A) and frontal (B) radiographs of the wrist depict ulnar translocation (type I). Posterior displacement of the proximal carpal row in relation to the radius (white arrow). Widening of the radioscaphoid joint (orange arrow) with ulnar shift of the proximal carpal row (blue outline).
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Scapholunate Ligament Tear and Progressive Scapholunate Interval Widening

Scaphoid Nonunion Advanced Collapse

Scaphoid nonunion advanced collapse, commonly abbreviated as a “SNAC wrist,” occurs with a scaphoid fracture (particularly nonunion fractures) with distal scaphoid fracture segment flexion and results in abnormal radioscaphoid articulation and degeneration (Figure 7). The hallmark of SNAC wrist is post-traumatic arthritis and carpal collapse following a nonunion scaphoid fracture.

Lunotriquetral Dissociation

Lunotriquetral dissociation can occur following trauma or ulnocarpal abutment associated with triangular fibrocartilage complex pathology. Injury to the lunotriquetral ligament results in volar intercalated segment instability (VISI), in which the lunate is flexed volarly secondary to the scaphoid flexion. In this pattern, the radiographic findings demonstrate a capitolunate angle > 30° and a scapholunate angle > 30°.

Carpal Instability Nondissociative

Carpal instability nondissociative (CIND) refers to dysfunction between the radius and first carpal row (radio-carpal) or between the first carpal row and the second carpal row (midcarpal). This dysfunction can involve either the intrinsic or extrinsic ligaments of the wrist; however, there is no disruption between carpal bones in the same row, as in CID. In this pattern, the individual carpal bones in each carpal row maintain their normal anatomic relationship with each other. As a result, the carpal rows and arcs maintain their intrinsic morphology and positioning.
Ulnar translocation occurs the extrinsic ligaments of the wrist are torn; resulting in ulnar shift of the proximal carpal row. Type I ulnar translocation involves tearing of the radioscapoid and radioscapohamate extrinsic ligaments with resultant widening of the radioscapoid interval and ulnar shift of the entire proximal carpal row (Figure 8). In type II ulnar translocation, the radioscapoid joint is maintained with ulnar shift of the remaining proximal carpal row.16

Carpal Instability Complex

Carpal instability complex (CIC) refers to carpal derangement involving an altered relationship between bones in the same carpal row and between the proximal and distal carpal rows (ie, both CID and CIND injuries).17 There are 5 subgroups of CIC (Table 4), with the more common groups 1 and 2 reviewed below.

Dorsal perilunate dislocations are ligamentous lesser arc injuries within the carpal instability complex class of injuries. There are 4 stages of progressive perilunate instability involving ligamentous injuries surrounding the lunate:18

Stage I: Scapholunate dissociation, which is defined by disruption of the dorsal scapholunate ligament when torque on the scapholunate ligament reaches threshold. Ligamentous injury is well visualized by MRI.

Stage II: Perilunate dislocation, which is when the scaphoid-capitate complex dislocates dorsal to the lunate. The extent of dorsal translation is determined by laxity of the radioscapohamate extrinsic ligament. Radiographic findings include dorsal displacement of the capitate in relation to the lunate while alignment of the lunate with the distal radius is maintained (Figure 9).

Stage III: Midcarpal dislocation involves progressive carpal hyperextension, which pulls the triquetrum into abnormal extension. This leads to tearing of the lunotriquetral ligament with possible avulsion injury of the triquetrum, leaving only the short radiolunate and volar ulnolunate ligaments as stabilizers. This injury is best evaluated by MRI. Radiographic findings include abnormal alignment of the lunate and radius (Figure 10).

Stage IV: Lunate dislocation, in which the capitate is pulled proximal and volar by the intact radioscapohamate extrinsic ligament causing the capitate to push the lunate volarily. Radiographic findings demonstrate maintained alignment of the capitate with the radius and volar tilting and displacement of the lunate in relation to the radius. There is increased volar tilt of the lunate compared to stage III (Figure 11).

Dorsal perilunate fracture-dislocation involves perilunate dislocation secondary to carpai bone fracture (eg, scaphoid, capitate, hamate, or triquetrum). The most common subtype is the trans-scaphoid perilunate dislocation (Figure 12).

Carpal Instability Adaptive

Carpal instability adaptive (CIA) occurs when the carpal rows adapt and change their angle in response to pathology or abnormal anatomy near the wrist.5 CIA results most commonly from abnormal tilt of the radius (ie, Madelung’s deformity or fracture malunion). Intrinsic ligament injury should be excluded with MRI of the wrist.

Conclusion

Carpal instability is a significant source of chronic pain and disability. The wrist is a highly organized group of ligaments and bones that normally allow for stable transition of strength, dexterity, and fine-motor control from the forearm to the hand—functions that are progressively limited as carpal instability worsens. Therefore, discerning the most common etiologies of instability and their imaging findings is important to avoid increased morbidity and degenerative disease that can result from misdiagnosis. While carpal instability is recognizable on plain-film radiography, MRI offers superior visualization of the extent of carpal instability, specific ligamentous injuries, and its long-term sequelae.

References

Heel pain is a common complaint among the general population and is present in 1 of 8 individuals, particularly those over age 50. Additionally, heel pain is one of the most prevalent complaints necessitating referral to a foot specialist. There are multiple etiologies for heel pain, which generally originates from 6 major anatomic structures: the plantar fascia, calcaneus, tarsal tunnel, tendons, bursae, and plantar fat pad.

This article will review the differential diagnosis for hindfoot pain as well as discuss pertinent MRI findings for each condition.

**Plantar Fascia**

The plantar fascia is a fibrous aponeurosis that arises along the medial calcaneal tuberosity. From the calcaneus, the plantar fascia divides into the medial, central, and lateral components (Figure 1).

The central band is the largest, adhering to the undersurface of the flexor digitorum brevis. At the midsole, the central band divides into 5 superficial and deep components extending toward the digits as the flexor tendons. This, along with the medial and lateral marginal superficial tracts, inserts onto each proximal phalanx. The plantar fascia plays a significant role in longitudinal arch support.

**Plantar Fasciitis**

Plantar fasciitis is one of the more common causes of plantar heel pain. Plantar fasciitis is related to microtrauma at the os calcis attachment and can result from repetitive trauma, enthesopathy, pes planus, pes cavus, or heel cord contractures. Stress-related trauma is the most common etiology and usually affects obese middle-age or elderly patients. Imaging-related findings for fasciitis include thickening of the plantar fascia (> 6 mm) at the proximal...
attachment and high-signal intensity on T2-weighted sequences with low to intermediate signal on T1-weighted sequences (Figure 2).6

**Plantar Fascia Tear**

A fascial tear is usually traumatic in etiology with sudden onset and localized tenderness. Fascial tears are commonly seen in running or jumping athletes.7 Imaging findings of acute tears will demonstrate a partial or full-thickness defect of the fascia with focal hyperintense signal on T2-weighted or short tau inversion recovery (STIR) sequences.7 Perifascial fluid-like signal can also be seen (Figure 3).

**Plantar Fibromatosis**

Plantar fibromatosis (Ledderhose disease) is a fibroproliferative disorder in which benign fibrous nodules develop within the plantar fascia.8 Plantar fibromatosis can be associated with many other fibroproliferative disorders such as Dupuytren disease and Peyronie disease.9 Plantar fibromatosis usually involves the more distal fascia and the central or medial bands. Imaging findings of plantar fibromatosis show nodular-thickening of the nonweight-bearing portions of the plantar fascia, which is hypointense on both T1- and proton density (PD)-weighted sequences (Figure 4). Hyperintense signal of the adjacent subcutaneous soft-tissues on T2- or PD-weighted sequences can also be seen.

There are multiple treatment options for plantar fibromatosis, which include surgical resection, radiation, and chemotherapy, used alone or in combination. The treatment is determined based on disease aggressiveness, patient age, and the risk of disability with resection. Surgical resection is the most common treatment; however, recurrence rates are high and can be deforming, even requiring amputation in some cases. If there is a possibility of functional loss, marginal excision and postoperative radiation therapy can be used. In the case of severe neurovascular or significant limb involvement, chemotherapy and radiation are often the sole option for treatment. Chemotherapy alone can also be used in young children to avoid disfiguring surgery and complications of radiation.8

**Calcaneus**

The calcaneus is responsible for significant axial load-bearing forces and is the most commonly fractured tarsal bone, responsible for up to 60% of all tarsal bone fractures in adults.10

**Calcaneal Stress Fracture**

Most stress fractures result from repetitive activity as opposed to direct trauma. Thus, calcaneal stress fractures are common in patients undergoing a
new occupation or repetitive motions (ie, military recruits or runners). Stress fractures are further classified as fatigue fracture (overuse in normal bone) or insufficiency fracture (normal use in abnormal bone).\(^2\) Conditions related to insufficiency fractures include those that weaken the bone integrity such as metabolic disorders, inflammatory conditions, bone dysplasias, and neurological disorders.\(^1\)

Calcaneal stress fractures are a cause of hindfoot pain that is commonly not visualized or radiographically occult, especially in the early stages. Reported radiographic sensitivity for the diagnosis of lower extremity stress fractures ranges from 12% to 56% with a specificity of 88% to 96%. MRI sensitivity and specificity for detecting stress fracture can be as high as 99% and 97%, respectively.\(^1\) MRI demonstrates linear hypointense signal in bone marrow on T1-weighted images, which extends to the cortex with surrounding increased marrow signal on T2-weighted and STIR sequences (Figure 5)\(^1\) Calcaneal stress fractures are more common in the posterior calcaneus.

**Tarsal Tunnel and Nerve Entrapment**

**Tarsal Tunnel Syndrome**

The tarsal tunnel is a fibro-osseous canal in the medial aspect of the ankle, which is a common location for compression and entrapment of neurovascular structures. The tarsal tunnel contains the tibialis posterior (TP) tendon, flexor digitorum longus (FDL) tendon, posterior tibial artery/vein and tibial nerve, and the flexor hallucis longus (FHL) tendon. The tarsal tunnel is created by the tibial arteries, the sustentaculum tali, and the medial calcaneal wall. The flexor retinaculum forms the roof of the tarsal tunnel (Figure 6).\(^2\)

Tarsal tunnel syndrome results from neuropathic entrapment or compression within the tunnel. The posterior tibial nerve and its branches can be compressed as it passes through the fibro-osseous tunnel, deep to the flexor retinaculum. Patients suffering from tarsal tunnel syndrome generally experience pain and sensory deficits.\(^1\)

Numerous etiologies can cause tarsal tunnel syndrome with up to 40% of cases being idiopathic, and less likely etiologies including ganglion cysts, tenosynovitis, varicosities, and osseous deformities.\(^2\) The patient’s symptoms can suggest the location of nerve entrapment as well as the branch of the posterior tibial nerve that is involved. For example, medial plantar nerve entrapment can occur at the tarsal tunnel or distal to the tarsal tunnel with loss of sensation along the distal third of the foot.

Imaging findings of tarsal tunnel syndrome depend on underlying etiology. Space-occupying lesions are well depicted on MRI, such as varicosities (Figure 7) and a large aneurysmal bone cyst of the talus causing mass effect on the tarsal tunnel (Figure 8).

**Baxter Neuropathy**

Baxter neuropathy is a syndrome caused by the entrapment of the inferior calcaneal nerve.\(^1\) The inferior calcaneal nerve is the first branch of the lateral calcaneal nerve, which if entrapped, can result in chronic heel pain often mimicking plantar fasciitis. Entrapment of the inferior calcaneal nerve occurs at 3 locations in the hindfoot: the medial border of the quadratus plantae...
muscle, along the fascial edge of an enlarged/hypertrophied abductor hallucis muscle, and most commonly at the medial calcaneal tuberosity. Imaging findings are usually related to denervation with increased signal intensity or atrophy of the intrinsic muscles of the foot. Incidental atrophy of the abductor digiti minimi likely reflects a prior clinically missed entrapment (Figure 9) and is not an uncommon finding.16,17

Tendons

Tendons normally have a homogeneous hypointense signal on all MRI sequences within the hindfoot.18 The main tendons of the hindfoot include the peroneus longus, peroneus brevis, Achilles, posterior tibial, FDL, and FHL tendons.

Achilles Tendon

The Achilles tendon is formed by the communion of the gastrocnemius muscle, along the fascial edge of an enlarged/hypertrophied abductor hallucis muscle, and most commonly at the medial calcaneal tuberosity. Imaging findings are usually related to denervation with increased signal intensity or atrophy of the intrinsic muscles of the foot. Incidental atrophy of the abductor digiti minimi likely reflects a prior clinically missed entrapment (Figure 9) and is not an uncommon finding.16,17

FIGURE 9. Baxter neuropathy. Coronal PD-weighted image of the ankle demonstrating moderate to severe muscle atrophy with fatty infiltration of the abductor digiti minimi (long arrows) compared to the normal abductor hallucis (short arrow) and flexor digitorum brevis muscles (dashed arrow). Incidentally, the central bundle of the plantar fascia is also very thickened, consistent with fasciitis (asterisk).


FIGURE 11. Achilles tendinosis. Sagittal STIR image of the ankle demonstrating diffuse thickening of the Achilles tendon (thin arrows) with intermediate intrasubstance signal (asterisk) consistent with non-insertional tendinosis. Fluid-like signal running parallel with, and anterior to, the Achilles tendon (thick arrow) represents inflammation of the paratenon (paratenonitis).

FIGURE 12. Achilles tendinosis. Sagittal STIR image of the ankle showing increased signal intensity of the distal Achilles tendon near the insertion on the calcaneus (arrows) representing insertional tendinosis. Longitudinal focus of signal intensity is also noted (arrowheads) representing a partial interstitial tear.

FIGURE 13. Achilles tendon disruption. Sagittal STIR image of the ankle demonstrating complete disruption of the Achilles tendon with increased signal of the tendon fibers (thick arrow). Retraction of the fibers is also noted (small arrows).

FIGURE 14. Posterior tibialis tendinosis and tenosynovitis. T2-weighted fat-suppressed image of the ankle with thickening of the posterior tibialis tendon (arrow) consistent with tendinosis and increased fluid with the tendon sheath (arrowhead) consistent with tenosynovitis.
and soleus muscles. The tendon inserts at the posterior calcaneus, os calcis. The Achilles tendon normally has a concave anterior and posterior convex contour and has low signal intensity on all sequences. Thickness of the tendon averages 6 mm. A common normal finding is linear or punctate increased signal intensity on low echo time (TE) sequences, usually more anterior within the tendon. This signal represents normal fascicular anatomy but can be mistaken for interstitial tears, therefore knowing this common appearance and location is imperative (Figure 10).

Achilles tendinopathy or tendinosis can be insertional or mid-substance. Non-insertional tendinosis is usually acute in onset and often proximal to the retrocalcaneal bursa. This entity usually occurs in older individuals who are less active and overweight. Insertional tendinopathy results from repetitive trauma and micro tears which usually present with weight-bearing pain in less athletic or active individuals and more commonly associated with running and jumping. Imaging findings on MR will demonstrate focal or fusiform thickening with diffuse or linear low to intermediate signal on fluid sensitive sequences (Figures 11 and 12). The Achilles tendon is unique as it does not have a true synovial sheath. However, it does have a thin sheath-like structure surrounding the tendon that is separated from the tendon by a lubricating layer of mucopolysaccharides. This structure is called the paratenon. Similar to tendosynovitis, with overuse the paratenon can become inflamed and cause pain. This is called paratenonitis (Figure 11).

Achilles ruptures, or complete tears, usually occur from 25-40 years of age. Activities that require dorsiflexed position while running or jumping are at a greater risk. Complete tears will demonstrate a T2-hyperintense signal defect of the tendon (Figure 13). Partial or complete retraction of fibers can be seen, depending on the degree of tearing. Chronic tendon pathology may lack intrasubstance signal but can be diffuse or focaly thickened.

Tibialis Posterior, Flexor Digitorum Longus, and Flexor Hallucis Longus Tendons

The FDL and FHL tendons course through a shallow groove in the posteromedial aspect of the talus and continue under the sustentaculum tali. On the plantar aspect of the heel, the FHL tendon crosses deep to the FDL tendon at the Master Knot of Henry, before their insertion on the base of the great toe and lesser distal phalanges, respectively. The TP tendon originates at the posterior tibia, fibula, and interosseous membrane. From its origin it courses along the deep posterior compartment of the lower leg, through the tarsal tunnel, under and around the medial malleolus and into its insertion at the plantar aspect of the navicular, cuneiforms, and metatarsal bases.

The TP, FDL, and FHL tendons are prone to tendonitis and tenosynovitis, which results in a painful posteromedial heel. These findings are more commonly seen in athletes performing repetitive forceful push off motion with...
the forefoot. Tenosynovitis is demonstrated by increased fluid-like signal intensity on T2-weighted sequences distending the tendon sheath (Figures 14 and 15). However, it is important to note that fluid in the FHL tendon sheath may be considered physiologic if similar to the volume of intraarticular fluid, as these structures often communicate.

**Peroneus Longus and Brevis Tendons**

The peroneus longus and brevis tendons course along a groove posterior to the fibula in the lateral ankle and curve anteroinferiorly along the undersurface of the foot. The peroneus longus inserts on the medial cuneiform and the base of the first metatarsal. The peroneus brevis inserts on the base of the fifth metatarsal. The peroneal tendons are also susceptible to tendonitis and tenosynovitis. Repetitive acute tenosynovitis can result in fibrous scar formation in the tendinous sheath, known as stenosing tenosynovitis. Imaging findings of stenosing tenosynovitis will demonstrate an intermediate signal intensity rind surrounding the tendon on both the T1- and T2-weighted sequences (Figures 16 and 17).

**Bursae**

There are two bursae of the hindfoot lying near the insertion of the Achilles tendon to the calcaneus. The retrocalcaneal bursa is located between the Achilles tendon insertion and the calcaneus. The retroachilles bursa (or subcutaneous calcaneal bursa) is situated between the skin and the Achilles tendon. On MRI a normal retrocalcaneal bursa is usually present and measures < 6 mm in the transverse plane and 1 mm in the anterior-posterior dimension. When these bursae become inflamed, they can generally be seen as uninterrupted MRI fluid-like signal in the expected locations of the bursae.

**Haglund Syndrome**

A Haglund deformity is a prominent bursal bony projection of the calcaneus, which can be a normal anatomical structure or associated with other findings. Haglund syndrome is the result of both soft tissue and osseous abnormalities consisting of a Haglund deformity, insertional tendinopathy, and pre-Achilles and/or retrocalcaneal bursitis. This entity is commonly associated with low-back or high-heel shoes. The imaging findings of Haglund syndrome include a prominent posterosuperior tuberosity of the calcaneus with or without increased marrow signal on the fluid-sensitive sequences, fluid-like signal within the pre-Achilles or retro-Achilles bursa, increased signal on T2/STIR sequences in the Kager (pre-Achilles) fat pad, and insertional tendinopathy of the Achilles tendon (Figure 18).

**Retroachilles and Retrocalcaneal Bursitis**

Bursitis in the retroachilles and retrocalcaneal bursa is most commonly a manifestation of Achilles pathology but can also occur as a separate entity. One of the more common causes of retroachilles and retrocalcaneal bursitis is repetitive trauma or friction. Bursitis can also be seen in the setting of rheumatoid arthritis and seronegative spondyloarthropathies. The retrocalcaneal bursa should measure < 1-2 mm in anteroposterior dimension. If enlarged, it may represent disease, especially if surrounding edematous changes are present. The subcutaneous fat should be seen between the Achilles tendon and the skin. If this fat cannot be seen on MRI, a blister or retro-Achilles bursitis may be present.
In particular, retro-Achilles bursitis is distinguished by edematous changes without mass effect on the skin.²

**Plantar Fat Pad**

The heel fat pad is composed of elastic fibrous septae with closely packed fat cells that act as a shock-absorber for the heel. Several plantar fat pad pathologies such as ulcers, abrasions and contusions can be identified with a detailed history and physical examination. However, the use of MRI is sometimes required to provide a differential diagnosis for heel pad abnormalities that cannot be explained clinically. Numerous causes of heel pain can arise from the fat pad, including infection, trauma (rupturing of the septa), neoplasms, inflammatory conditions, and rheumatoid nodules.

Rheumatoid nodules of the heel pad occur in 20% of patients who test positive for rheumatoid factor.² Rheumatoid nodules usually develop on the plantar surface of the heel, but may occur near the Achilles tendon insertion. Imaging findings are related to their histologic composition. Solid nodules are composed of chronic inflammatory cells and usually show decreased signal intensity on T1- and T2-weighted sequences with postcontrast enhancement. Nodules can have central necrosis with increased signal intensity on T2-weighted sequences and peripheral enhancement (Figure 19).³¹

**Conclusion**

Heel pain is a common musculoskeletal complaint for presentation to primary care or a foot specialist, and the specific etiology is often difficult to ascertain clinically. The heel is a complex area to assess with numerous sources of pain. However, compartmentalizing the heel into different anatomic structures and understanding the imaging findings and differential diagnoses for each location will help guide the clinician to a more accurate diagnosis and earlier treatment.

**References**

Cavitary lesions are often encountered during radiographic evaluation of the chest. During early radiology training, residents are introduced to the mnemonic “CAVITY” for the differential diagnosis of pulmonary cavitary lesions: cancer (bronchogenic carcinoma, especially squamous cell carcinoma), autoimmune (granulomatosis with polyangiitis or rheumatoid arthritis), vascular (pulmonary emboli – septic or bland), infection (tuberculosis, fungal, *staphylococcus aureus*), trauma (pneumatocele or laceration), and youth (congenital pulmonary airway malformation, pulmonary sequestration, bronchogenic cyst). Although this mnemonic is an efficient way of expanding the differential diagnosis for novice radiologists, a deeper understanding of each condition is necessary to make the correct diagnosis in practice. In addition, these differentials must be given in the appropriate clinical context. This article will discuss imaging findings of common cavitary lesions, which along with clinical history, can lead to the correct diagnosis and expedite appropriate management.

Cavity or Pulmonary Cyst

To arrive at the correct diagnosis, the difference between pulmonary cysts and cavities must be defined. According to the Fleischner Society, a pulmonary cyst is “any round circumscribed space surrounded by an epithelial or fibrous wall.” The wall thickness of the cyst is usually < 2 mm. Meanwhile, a pulmonary cavity is defined as “a gas-filled space that is seen as a lucency or low-attenuation area within a pulmonary consolidation, mass, or a nodule” (Figure 1). Wall thickness of a cavitary lesion is usually > 2 to 4 mm. Wall thickness also helps to predict malignancy of a lesion. Cysts or cavities with wall thickness < 4 mm are likely benign, while wall thickness > 15 mm suggests malignancy.

Table 1 includes a broad differential diagnosis of both pulmonary cysts and cavities.

Bleb/Bulla

Blebs and bullae are often incidentally found in asymptomatic patients, mostly in thin younger males or patients with an extensive smoking history. Blebs are formed as a result of spontaneous rupture of subpleural alveoli. Both are usually located in the periphery of lungs with thin walls < 1 mm. The main distinction between blebs and bullae is size (Figure 2). Blebs are defined as a “small gas-containing space within the visceral pleura or in the subpleural lung, not larger than 1 cm in diameter.” Bullae are defined as “airspace measuring > 1 cm in diameter, sharply demarcated by a thin wall that is no greater than 1 mm in thickness.” Reporting large bullae is
important as they can rupture, resulting in a pneumothorax.

Cancer

Bronchogenic Carcinoma

There are 3 proposed mechanisms of how primary lung cancer presents as a cystic mass. The first mechanism is by having a rapid growth of lung cancer during which the blood supply cannot meet the demand of the neoplastic growth and causes central necrosis of the tumor.\(^3\) The second mechanism is due to mass effect causing bronchial obstruction, scarring, or bronchiectasis resulting in infection distal to the obstructive mass; infection later leads to the breakdown of the lung parenchyma and forms the cystic cavity. The third mechanism is by “spillover abscess,” which describes spillage of the primary infection to the

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**FIGURE 2.** Bulla. Axial contrast-enhanced CT image demonstrating a large bulla (hollow arrow) occupying the azygos lobe (solid arrow).

**FIGURE 3.** Metastasis. Axial unenhanced CT and fused F-18 fluorodeoxyglucose (FDG) PET/CT images demonstrate a cavitary right upper lobe mass (yellow arrow) with FDG avidity (red arrow) and history of head and neck squamous cell carcinoma.

**FIGURE 4.** Metastatic colon cancer. Axial-enhanced CT image demonstrating multiple bilateral cavitary lesions (yellow arrows), which were biopsied and consistent with metastatic colon cancer. There is associated surrounding hemorrhage presenting as ground-glass density (red arrows).
distant site of cavitation, even involving different lobes of the lung.\textsuperscript{4}

Cavitating lung cancer is most often seen in the sixth to seventh decades of life in patients with significant smoking history. Unfortunately, cavitation in primary lung cancer is associated with a poor prognosis. Out of all the types of primary lung carcinoma, squamous cell carcinoma is most commonly associated with cavitary lesions.\textsuperscript{4} Cavitary lesions are rarely associated with small cell carcinoma. Findings associated with primary lung cancer include thick and irregular inner walls. Cavity size varies from 1 to 10 cm. According to a study by Woodring et al, most cavitary lesions with wall thickness < 4 mm were benign, >15 mm were malignant, and between 4 and 15 mm had mixed results.\textsuperscript{3,5}

**Metastasis**

Cavitation of lung metastases from extrapulmonary primary cancer is uncommon, occurring in only 4\% of cases.\textsuperscript{5} The average age for presentation with pulmonary metastasis is 60 to 70 years. The most common primary origin of pulmonary metastatic disease is squamous cell cancer of the head and neck. Other primary sites include large
intestine, cervix, stomach, esophagus, pancreas, and kidney. Cavitation size varies from 1 to 6 cm, and the wall thickness also varies from 0.3 to 2.5 cm.\textsuperscript{5} Similar to primary lung cancer, thick and irregular walls are the most common imaging findings, and metastasis often presents with multiple cavities, mostly seen in the periphery of lungs (Figures 3 and 4). The diagnosis is often made by biopsy of the lesions due to their indeterminate imaging characteristics.

**Autoimmune (Cyst)**

**Lymphangioleiomyomatosis**

Lymphangioleiomyomatosis (LAM) is defined as progressive growth of smooth muscle cells in the pulmonary parenchyma, vasculature, lymphatics, and pleurae. LAM exclusively affects females 20 to 40 years old. The etiology of LAM is not well defined, but the close relationship between tuberous sclerosis and LAM suggests that somatic mosaicism on the TSC-2 gene may have a role.\textsuperscript{6} Another theory is that LAM is associated with a metastatic neoplasm originating from the uterus, hence the reason for involving only the female population.\textsuperscript{7} Most patients present with cough, hemoptysis, and chest pain. The symptoms may be exacerbated during pregnancy as estrogen level increases. Diagnosis can be made with CT, which demonstrates diffuse bilateral thin-walled cysts, measuring up to 5 mm in diameter, with associated hemorraghic ground-glass opacities (Figure 5A).\textsuperscript{8} When CT is not diagnostic, histopathologic diagnosis of smooth muscle cells can confirm the diagnosis. Other imaging manifestations of tuberous sclerosis include renal angiomyolipomas (Figure 5B) and cardiac rhabdomyomas.\textsuperscript{9} LAM carries a poor prognosis as it can lead to progressive respiratory failure. Current treatment includes mTOR inhibitor (eg, Sirolimus) or lung transplant.\textsuperscript{6}

**Interstitial Lung Disease**

Interstitial lung disease (ILD) is a broad category encompassing many different idiopathic interstitial pneumonias. ILD affects the interstitium that surrounds alveoli. The most common and concerning condition with a poor prognosis is idiopathic pulmonary fibrosis (IPF). IPF most often occurs between ages 40 and 70 years. However, in patients who have more than 2 first-degree relatives with pulmonary fibrosis, this may present before their fifth decade.\textsuperscript{10} As with all other types of interstitial pneumonia, IPF assessment is best performed with thin-section high-resolution CT (HRCT). Imaging findings of IPF are a definite or probable UIP pattern including general parenchymal volume loss, basilar and subpleural predominant fibrotic change, reticular abnormality, and honeycomb with or without traction bronchiectasis (Figure 6). The most defining characteristic of UIP/IPF on HRCT is the honeycomb fibrotic appearance, described as multilayered cystic changes ranging from 2 to 10 mm most commonly distributed in the lung bases. With the recent introduction of Pirfenidone treatment in these patients, the survival time among IPF patients improved by 52 weeks, from 2 to 3 years.\textsuperscript{11} Five-year survival rate of IPF ranges from 30% to 50%. Pirfenidone and Nintedanib can be used in cases of mild to moderate disease to delay lung transplantation.\textsuperscript{10}

**Progressive Systemic Sclerosis (Scleroderma)**

Systemic sclerosis (SSc) is a connective tissue disorder characterized by progressive fibrosis of multiple organs, including the lungs, skin, vessels, and visceral organs. This condition is seen 4 times more often in women.
and commonly between ages 20 and 50. Patients with pulmonary involvement present with a restrictive lung disease pattern of low lung volumes, preserved flow rate, and low diffusion capacity. Interstitial lung disease can also develop, which is seen in about two-thirds of patients. On HRCT, either usual interstitial pneumonia (UIP) or nonspecific interstitial pneumonia (NSIP) pattern may be observed with variable presentation from early ground-glass opacities to late fibrosis. Lung bases and subpleural spaces are most commonly affected. In some cases, cystic changes may occur with each cyst ranging from 1 to 5 cm in diameter (Figure 7). Dilatation of the esophagus is a crucial finding and unique to SSc. Once the lung disease has progressed to fibrosis/UIP, chances of disease reversal are poor. As of now, cyclophosphamide, glucocorticoids, or N-acetylcysteine can be attempted to halt disease progression. However, the efficacy of these treatments is better in earlier phases of the disease.

Pulmonary Langerhans Cell Histiocytosis

Langerhans cell histiocytosis (LCH) is a rare pediatric disease with male predilection that is mainly diagnosed between the ages of 1 and 3. As the name implies, LCH is caused by uncontrolled monoclonal proliferation of langerhan dendritic cells of the skin and other tissues contacting the external surface. The overactivation of the “langerhans-like” histiocytes begin to release large amount of oxidants, proteases, and fibronectin causing destruction of the lung parenchyma. In addition to the wide-spread disseminated form, a pulmonary manifestation of this disease, also known as pulmonary langerhans cell histiocytosis, can be seen in young males between 20 to 40-years-old and is highly associated with smoking. The most common presenting symptoms are dyspnea and dry cough. Patients can also present with pleuritic chest pain, weight loss, or spontaneous pneumothorax. Classic imaging findings are thin-walled, small, irregular shaped cysts (usually less than 10mm in diameter) which are upper lobe predominant and associated small nodules. Fibrotic changes can be observed in the later stages of disease. Prognosis is good with 50% of patients showing spontaneous resolution after smoking cessation. Corticosteroids are often used as a treatment option with good results.

Autoimmune (Cavitary)
Granulomatosis with Polyangiitis

Granulomatosis with Polyangiitis (GPA) is a multisystem necrotizing granulomatous vasculitis that affects primarily the small to medium-sized vessels and was previously termed “Wegener granulomatosis.” The lungs are most commonly involved in this disease and the patients present with cough, hemoptysis, and dyspnea from ages 40 to 60.

Common imaging findings of the lungs include multiple, bilateral pulmonary masses with cavitation in more than 50% of the lesions. Cavitations are more common in the larger lesions and are thick with irregular, “shaggy” cavity walls (Figure 8). This pulmonary disease can also present with alveolar hemorrhage in approximately 10% of patients. There can be a pulmonary vessel coursing directly to the mass in approximately 88% of cases (Figure 9), coined as the “feeding vessel sign.” However, this is a nonspecific sign that can be seen in other entities of cavitary disease. Other signs on imaging include a “reversed halo sign,” which has consolidation surrounding a central ground-glass density.

This disease is an autoimmune disease of uncertain etiology and is treated

**FIGURE 10.** Pyoderma gangrenosum. Coronal contrast-enhanced CT of a patient with pyoderma gangrenosum demonstrates a thick-walled cavitary lesion (yellow arrow). Extracutaneous involvement was suspected considering the lesion was sterile and negative for malignancy on biopsy.

**FIGURE 11.** Septic emboli. Axial contrast-enhanced CT image demonstrating multiple peripheral cavities (yellow arrows) in various stages of evolution with feeding vessels (red arrows).
with immunosuppressive drugs. The nodules and pulmonary disease increase in size and number with progression; however, remission rate is approximately 90% with appropriate treatment. Renal failure is the most common cause of death in this patient population.

**Rheumatoid Arthritis**

Rheumatoid arthritis (RA) is a progressive, systemic autoimmune disorder that is commonly an articular disease, although does present with extra-articular symptoms. The lung is a common site of extra-articular disease and can manifest as airway, parenchymal, or pleural disease.

Necrobiotic rheumatoid nodules of the lung are uncommon and are usually seen in conjunction with a high rheumatoid factor. These nodules are usually large, discrete subpleural nodules, which may develop cavitation. This cavitation can lead to hemoptysis, spontaneous pneumothorax, and the development of a bronchopleural fistula.

RA commonly presents with interstitial lung disease, most commonly as UIP and NSIP. There are multiple risk factors for developing RA interstitial lung disease including smoking history, advanced age, high-titer rheumatoid factor, and a family history of RA.

**Pyoderma Gangrenosum (PG)**

Pyoderma Gangrenosum is a rare neutrophilic dermatologic disease that occasionally can present with extracutaneous manifestations, which include pulmonary involvement. Etiology is unknown; however, more than 50% of these patients are associated with an underlying systemic disorder such as inflammatory bowel disease, rheumatoid arthritis, or hematological disorders. Pulmonary disease manifestations are rare and are usually diagnosed simultaneously or weeks to years after a cutaneous disease diagnoses.

Chest imaging is nonspecific in these patients; however, they can present with pulmonary cystic change and large cavitating nodular lesions or consolidation. The cavitating masses are secondary to nodular lesions that develop central caseation (Figure 10) and commonly require histopathological assessment for diagnosis.

**Sarcoidosis**

Sarcoidosis is a systemic chronic granulomatous disease characterized by unique noncaseating granulomas in multiple organs. The lungs are involved in more than 90% of these patients. Peak age for presentation is 20 to 30 years and patients generally present with mild cough, dyspnea, or fatigue. This disease should be considered in patients younger than 40 with mild clinical symptoms and bilateral hilar and mediastinal lymphadenopathy on chest x-ray.

The greatest morbidity and mortality in this patient population is from thoracic involvement and approximately 20% of these patients will progress to chronic interstitial lung disease. Patients display CT findings of cavitary nodules when central cavitation occurs from ischemic necrosis or angiitis of the nodules. However, the classic pulmonary presentation of sarcoidosis is bilateral hilar and right paratracheal lymphadenopathy with perilymphatic micronodular disease.

**Vascular Septic Emboli**

Infected embolic material can seed the lung parenchyma from an extrapulmonary source through the pulmonary vasculature. This occurs most commonly through infected foreign body material or by less likely etiologies such as infective endocarditis or Lemierre syndrome. Staphylococcus aureus is the most common organism related to foreign body infection and IV drug abuse. This can occur with infected venous catheters, pacemaker wires, or other
indwelling catheters. Infective endocarditis of the tricuspid valve is the most commonly affected valve leading to septic embolic disease. Also, Lemierre syndrome is a possible etiology that occurs when acute pharyngotonsillitis leads to jugular vein septic thrombosis, possibly resulting in septic emboli.

Imaging findings of pulmonary septic emboli include multiple discrete nodules ranging from 0.5 - 3.5 cm. The nodules are usually bilateral and peripheral with central cavitation (Figure 11). Other imaging features seen in this entity include a ground-glass halo surrounding the nodules as well as a “feeding vessel sign.” Vessels can be seen leading directly to the nodule and are found in 60% to 70% of patients.

These patients are treated with broad spectrum antibiotics, sometimes up to 6 to 8 weeks in cases of infective endocarditis. Removing the source of infection is imperative for improvement.

Infection

**Tuberculosis and Mycobacterium Avium Complex**

A myriad of bacterial, fungal, and parasitic organisms can lead to cavitary lung disease. Among them, the most familiar causative organism is *Mycobacterium tuberculosis* (TB). The incidence of TB has been decreasing in the US but remains a daunting threat among the immunocompromised population. In addition, immigrants from endemic regions (Asia, Africa, Russia, Eastern Europe and Latin America), those with low incomes and limited access to health care, intravenous drug users, people who live or work in high-risk residential centers (nursing homes, correctional facilities and homeless shelters), and health care workers are still vulnerable to opportunistic organisms.

TB is divided into primary vs postprimary tuberculosis. Imaging findings for primary tuberculosis include pulmonary consolidation, effusion, and lymphadenopathy. In postprimary tuberculosis, the most common imaging findings include cavitary lesions in which patients present with fever, night sweats, weight loss, and cough. Cavitary lesions in postprimary TB tend to be in the apical regions with thick irregular walls. Surrounding airspace opacity can also be observed. In cases of superinfection, cavities may present with internal air-fluid levels. Detection of cavitary lesions on imaging do prolong the overall length of treatment (Figure 12).

**Bacterial Pneumonia/Abscess**

Pulmonary abscesses are most often caused by organisms in the oral cavity, with *staphylococci* and *streptococci* the most common. Lung abscesses are divided into acute (< 6 weeks) vs chronic (> 6 weeks). Patients often present with symptoms of fever, chills, fatigue, night sweats, productive cough, and weight loss. Unlike TB, abscesses > 2 cm are almost always found with internal air-fluid levels (Figure 13).

Lung abscesses are commonly treated with antibiotics for at least 3 to 6 weeks. Moreover, abscesses > 6 cm should be considered for surgical resection or percutaneous transthoracic tube drainage.**Aspergillosis**

Pulmonary aspergillosis is almost exclusively seen in patients with immundeficiency or with chronic lung disease such as chronic obstructive pulmonary disease (COPD). Many people in the general population are exposed to aspiration of aspergillosis, but only immunodeficient patients will develop clinical symptoms. Types of aspergillosis include aspergilloma, invasive aspergillosis, and semi-invasive aspergillosis. An aspergilloma is when a fungal collection, or “fungal ball,” develops in a pre-existing cavity and is commonly seen in immunocompetent patients. Semi-invasive aspergillosis is a chronic necrotizing pulmonary aspergillosis, which can develop cavitary
Lastly, invasive aspergillosis is seen in severely immunocompromised patients with a rapidly progressive angioinvasive fungal infection and often presents with a pulmonary cavitary lesion and “air crescent sign.” Typical patient presentation of chronic pulmonary aspergillosis is a middle-aged male with symptoms of weight loss, loss of appetite, productive cough, pleuritic chest pain, and often hemoptysis. Sometimes the cavity may contain an aspergilloma, which is a conglomerate of aspergillosis hyphae, fibrin and cell debris. Treatment options for aspergillosis include azoles followed by inhaled amphotericin B for a prolonged period of 6 to 12 months.

Trauma

**Pulmonary Laceration**

Pulmonary laceration, the tearing of lung parenchyma, occurs secondary to traumatic compression, shearing forces, direct injury from rib fractures, or at the site of previously formed pleural adhesions.

Having a round or oval shape with varying number of lesions and sizes, laceration has a highly variable appearance on imaging. Pulmonary laceration is often obscured on chest radiography the first 48 to 72 hours because of surrounding associated pulmonary contusion (Figures 15 and 16). CT is more sensitive in detecting pulmonary lacerations offering a more comprehensive assessment of the extent of pulmonary injury and laceration. Air and/or blood may fill in the laceration creating a thin pseudomembrane. Occasionally, active bleeding into a pulmonary laceration can be seen on contrast-enhanced CT. It presents as a linear density, similar to that of the blood pool, forming in or along the periphery of the laceration. Uncommon complications include bronchopleural fistula and abscess, with the former occurring more often in the setting of peripheral lacerations. Healing times vary from weeks to several months for laceration, depending on the severity and associated injury. Having an accurate history during the interpretation of subsequent radiographs or CT scans is critical to prevent mistaking a blood-filled cystic laceration for a neoplasm.

**Pneumatocele**

Pneumatoceles are thin-walled cystic spaces in the parenchyma of the lung. It is not uncommon for pneumatoceles to contain fluid, creating an air-fluid level observed on imaging. Infection is the major cause of pneumatoceles, with other

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**FIGURE 16.** Pulmonary laceration and associated hemorrhage. Axial and coronal contrast-enhanced CT images in a patient with a gunshot wound demonstrating a pulmonary laceration and hemorrhage (yellow arrow) with a pneumothorax (red arrow). Left supraclavicular and lateral chest wall subcutaneous emphysema (blue arrows) is also present.

**FIGURE 17.** Congenital pulmonary airway malformation (CPAM). Frontal radiograph in a pediatric patient demonstrating a left central thin-walled lesion (yellow arrow) consistent with a CPAM.

**FIGURE 18.** Bronchogenic sequestration. Unenhanced CT image demonstrating a thin-walled septate cystic lesion (yellow arrow) with a feeding systemic vessel (red arrow).
etiolgies including positive pressure ventilation, hydrocarbon ingestion, and blunt trauma.\textsuperscript{33, 34} In the setting of antecedent trauma, pneumatoceles are most often seen as cystic spaces with peripheral ground-glass attenuation. As with pulmonary lacerations, healing may take several weeks, and will often completely resolve. Rare complications include spontaneous rupture with pneumothorax and secondary infection.\textsuperscript{35} Rarely, surgical resection of a pneumatocele may be required if the patient has recurrent infections, mass effect, or recurrent rupture with resultant pneumothorax.\textsuperscript{34}

**Congenital (“Youth”)**

**Congenital Pulmonary Airway Malformation**

Congenital pulmonary airway malformation (CPAM) includes a spectrum of pulmonary disease that affects varying aspects of the tracheobronchial tree and distal airways. The pathophysiology of CPAM development is controversial, but the entity involves an abnormal mass of pulmonary tissue with differing levels of cystic components that communicate with the tracheobronchial tree. This lesion has normal vascular supply and drainage, which differentiates it from pulmonary sequestrations.\textsuperscript{36} The abnormality is usually identified during routine obstetric care, due to increased use of ultrasound, or in children presenting with cough and abnormal chest radiography. It can rarely present in adulthood with recurrent pulmonary infections.

These lesions vary in histological and imaging presentation and have been classified into type 0 through IV. Type 0 is acinar dysplasia or agenesis and is incompatible with survival. Type I is the “large-cyst type” on imaging and typically affects a single lobe with cyst size 1-10 cm (Figure 17). Type II is the “small cyst type” on imaging, which has small lesions <2.0 cm. Type III is a solid-appearing lesion with microcysts that coalesce to form a solid-appearing mass on imaging. Type IV disease typically affects a single lobe and has large thin-walled cysts and cannot be readily discernible from type I by imaging. Type II and III lesions have a poor prognosis while type I and IV lesions have a much better prognosis.\textsuperscript{37}

**Bronchopulmonary Sequestration**

A sequestration is a congenital region of abnormal lung tissue that does not connect to the bronchial tree or pulmonary arteries and can present as a cystic lesion on imaging (Figure 18). The abnormality commonly presents in the lower lobes, most often the left lower lobe. Often the abnormality presents on chest radiography as a persistent lower-lobe opacity in a patient with recurrent pneumonia. Sequestrations often have a systemic feeding artery arising from the descending aorta, which can be visualized on CT or MR imaging. Sequestrations can also be seen with elements of other congenital pulmonary malformations, including CPAM, with “hybrid” lesions sometimes visualized.\textsuperscript{38} Bronchopulmonary sequestrations can be divided into intralobar and extralobar types.

Intralobar sequestration is more common (approximately 75%), is intrapleural in location, and commonly has pulmonary venous drainage. It usually presents as an isolated anomaly and is often present in older children or adults who present with recurrent pneumonia. Extralobar sequestration is less common (25%) and is extrapleural in location with a separate pleural lining from the normal lung parenchyma. The extralobar sequestration usually has systemic venous drainage.\textsuperscript{39} In symptomatic cases, patients often require surgical resection of the abnormal lung tissue.\textsuperscript{40}

**Bronchogenic Cyst**

Bronchogenic cysts are ventral foregut cysts that occur with abnormal foregut budding between the 26th and 40th day of gestation. They can occur in the mediastinum and pulmonary parenchyma. The mediastinal cysts predominantly occur in the middle or posterior mediastinum and are typically subcarinal extending toward the right hilum. The pulmonary bronchogenic cysts are commonly lower lobe, and more often in the medial aspect. These cysts are well-margined with thin walls, spherical, and often are simple cysts with no internal debris. They can have internal high attenuation on CT with intraluminal mucoid, hemorrhagic, or viscous contents. They rarely contain internal air or air-fluid levels. CT is often diagnostic of these lesions documenting their fluid-attenuation; however MR, can be useful in patients with indeterminate lesions.\textsuperscript{41} These cysts are often asymptomatic, but can rarely present with chest pain, cough or infection. For symptomatic lesions, aspiration or ablation is common, or surgical resection can be performed if they are recurrent and symptomatic.\textsuperscript{42}

**Conclusion**

When radiologists encounter pulmonary cavitary lesions the differential diagnosis is broad. Pertinent clinical history and imaging findings can help distinguish between the multitude of entities and allow the clinician to expedite appropriate patient management, ultimately improving clinical outcomes.

**References**

Fibroma of the Tendon Sheath

A 53-year-old man with an 18-month history of an enlarging dorsal left wrist mass was referred for an MRI, which demonstrated a heterogeneous soft-tissue mass involving the tendon sheaths of the dorsal third through fifth extensor compartments. The mass was essentially isointense to skeletal muscle on T1-weighted precontrast (star annotation, Figures A and B) and fat-saturated proton density-weighted sequences (star annotation, Figure C). Postcontrast fat-saturated imaging showed mild heterogeneous enhancement (star annotation, Figure D). The pathologic diagnosis was a fibroma of the tendon sheath (FTS).

FTS, also known as synovial fibroma, is a benign soft-tissue tumor representing 2% to 3% of all hand tumors. The typical presentation is an asymptomatic palpable mass in the third to fifth decades with a male predominance. The tumor most commonly affects the flexor surfaces. Imaging features are nonspecific with radiographs often appearing normal. CT demonstrates a mass isodense to musculature with encasement of tendons. MRI demonstrates a lobulated heterogeneous mass with T1-signal intensity similar to skeletal muscle and low to intermediate signal on T2-weighted images. Enhancement is variable.

The differential diagnosis includes giant cell tumor, nodular fasciitis, and fibrous histiocytoma. FTS is composed of scattered benign fibroblasts within a dense background of collagen. By definition, no giant cells, xanthoma cells, inflammatory cells, or areas of necrosis are seen on histology. Surgical excision is the treatment of choice given the inability to prospectively distinguish FTS from other common tumors.

**References**

Parotid Gland Myxofibrosarcoma

An 85-year-old woman presented to an otolaryngologist with left-sided facial swelling and facial paralysis. A contrast-enhanced CT of the neck was performed (Figures A, B) demonstrating a mass infiltrating the left parotid gland impressing upon the left parapharyngeal space (arrow in A).

The patient underwent a total left parotidectomy and parapharyngeal dissection. Intraoperatively, the left parotid mass was noted to encase the branches of the left facial nerve, as well as the marginal mandibular nerve. Pathology revealed a soft-tissue sarcoma, specifically a myxofibrosarcoma.

As the most common malignant connective tissue sarcoma in older adults, myxofibrosarcoma was initially classified as a myxoid variant of malignant fibrous histiocytoma.\(^1,2\) These mesodermal-derived neoplasms most often arise from the subcutaneous layer of the lower extremities in older patients. Advanced disseminated cases of myxofibrosarcomas have been known to metastasize to the lung, bone and liver.\(^3\)

Amongst the anatomic head and neck regions, myxofibrosarcomas have been reported to involve the nasal cavity and paranasal sinuses, mandible, orbit, larynx, parotid gland, oral cavity, ear, eyelid, and infratemporal space.\(^2,3\) The most common head and neck location is the nasal cavity and paranasal sinuses, seen in up to 30% of cases.\(^3\) Primary myxofibrosarcoma of the parotid gland is rare, with approximately 30 cases reported worldwide.\(^4\)

Unfortunately, predicting these rare tumors on imaging is challenging as their imaging features are nonspecific.\(^3\) On CT, they most often appear as a lobulated soft-tissue mass, similar to muscle in attenuation. They also often appear with centralized hypodensity, which may be related to underlying necrosis or the myxoid composition of the tumor.\(^3\) In up to 20% of patients in a study by Parks et al, ossification or calcification was detected within the lesion.\(^3\)

Due to their nonspecific imaging features, myxofibrosarcomas arising in the head and neck region are often confused with several other soft-tissue and fibrous abnormalities and neoplasms such as fibrosarcoma, pleomorphic rhabdomyosarcoma, nodular fasciitis, and fibromatosis in addition to the more common classic parotid neoplasms.\(^3\)

Local recurrence rates are high regardless of tumor grade, making negative surgical markings critical.\(^2\) As in this case, many oncologists perform subsequent adjuvant radiotherapy in these patients.\(^5\) After successful surgery, this patient completed 31 rounds of adjuvant radiotherapy. However, localized disease recurred.

**References**