Muscle-Tendon-Enthesis Unit

Anthony S. Tadros, MD[1]   Brady K. Huang, MD[1]   Mini N. Pathria, MD[1]

[1] Department of Radiology, University of California, San Diego School of Medicine, San Diego, California

Address for correspondence Anthony S. Tadros, MD, Department of Radiology, UC San Diego Medical Center, 408 Dickinson Street, MC: 8226, San Diego, CA 92103 (e-mail: atadros@ucsd.edu).

Abstract

Keywords

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Injuries to the muscle-tendon-enthesis unit are common and a significant source of pain and loss of function. This article focuses on the important anatomical and biomechanical considerations for each component of the muscle-tendon-enthesis unit. We review normal and pathologic conditions affecting this unit, illustrating the imaging appearance of common disorders on magnetic resonance imaging and ultrasound. Knowledge of the anatomy and biomechanics of these structures is crucial for the radiologist to make accurate diagnoses and provide clinically relevant assessments.

Function of the Muscle-Tendon-Enthesis Unit

The muscle-tendon-enthesis complex is the functional unit of locomotion and a useful conceptual framework for considering musculoskeletal pathology. Its purpose is to transmit force from one bone to another,[1] enabling skeletal movement and providing dynamic stabilization to the skeleton. The general construction of the classic muscle-tendon-enthesis unit includes (1) a muscle that has an origin and an insertion; (2) a tendon that interdigitates with muscle via a myotendinous junction; and (3) an enthesis or footprint, whereby the tendon inserts onto bone (►**Fig. 1**). There are anatomical variations to this general build. The muscle may have only one tendon or a tendon at both its ends, the muscle may directly insert onto bone without any discrete tendon, or the tendon may attach to bone without a defined enthesis.[2]

Patterns of Failure

In a skeletally immature person, the weakest point in the muscle-tendon-enthesis unit is the bone, specifically the cartilaginous growth plate found at epiphyses and apophyses.[2] Avulsion fractures are most common during the adolescent growth spurt when rapid expansion of cartilage cells weakens the growth plate.[2,3] The risk of such injuries may be exacerbated by changes in the precise geometry of muscle force acting on bone related to rapid changes in height during adolescence.[3] These injuries are commonly seen at the pelvic apophyses, most often at the ischial tuberosity.

In the young adult, the weakest site in the muscle-tendon-enthesis unit is the myotendinous junction.[4] This junction is made up of ridge-like protrusions between tendon and muscle,[5] allowing collagen fibers to interdigitate via deep recesses with muscle fibers.[1] Sarcoplasmic invaginations increase the contact area between tendon and muscle and improve the dissipation of force generated during muscle contraction.[5] Despite these structural solutions, the myotendinous junction has less energy absorption capacity compared with tendon or muscle and is therefore at high risk for failure.[4]

With aging, the site of failure transitions to the tendon that is uncommonly injured in children and young adults. When failure occurs within the substance of a tendon, it is nearly always because the tendon is abnormal, typically due to degenerative tendinosis.[6] Common mechanisms for injury at the diseased tendon include excessive tensile and shear forces, compounded by compression within a fibro-osseous tunnel.[1]

Imaging Techniques

Magnetic resonance imaging (MRI) and ultrasound (US) are well suited for evaluation of the muscle-tendon-enthesis unit. Although both modalities are accurate for the assessment of tendon and muscle abnormalities,[7–9] each provides unique advantages. MRI provides excellent soft tissue contrast, depicts the severity and extent of injury in a more recognizable fashion, and it allows for a global simultaneous assessment of all anatomical structures including the adjacent bone and
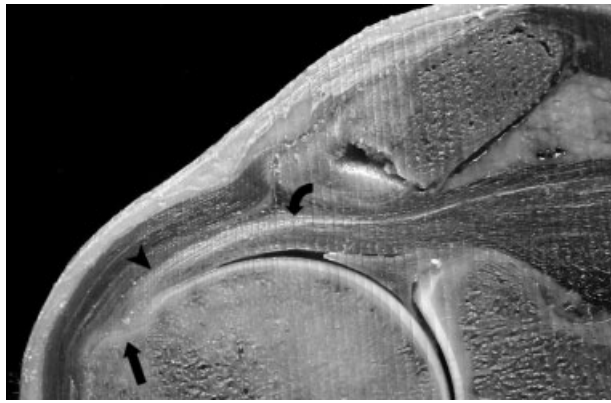
Fig. 1 Coronal cadaveric section of a normal supraspinatus tendon. Supraspinatus muscle fibers interdigitate with its tendon (arrowhead) via a myotendinous junction (curved arrow). The tendon shows a white pearly appearance with densely packed fibers inserting onto the horizontal facet of the greater tuberosity at the enthesis or footprint (straight arrow).

cartilage (►**Fig. 2**).[9,10] US allows for dynamic evaluation, exhibits higher spatial resolution, and is less prone to artifacts such as susceptibility and magic angle.[9–11] US is also rapid, less expensive, facilitates intervention, assesses tissue vascularity, and can be combined with clinical assessment.[10] The latter is particularly important in the evaluation of enthesopathy, where structural alterations correlate poorly with clinical symptomatology (►**Fig. 3**).[12,13]

Fig. 2 An 81-year-old man with right knee pain and swelling after jumping off a 3-foot wall. Sagittal T2-weighted fat-saturated MR image depicts the severity and extent of a high-grade partial quadriceps tendon tear. The rectus femoris (solid arrow), vastus lateralis (open arrow), and vastus medialis (arrowhead) components are all torn at their patellar attachments, with preservation of the posterior vastus intermedius fibers. Extensive edema is present within the vastus intermedius musculature (curved arrow).
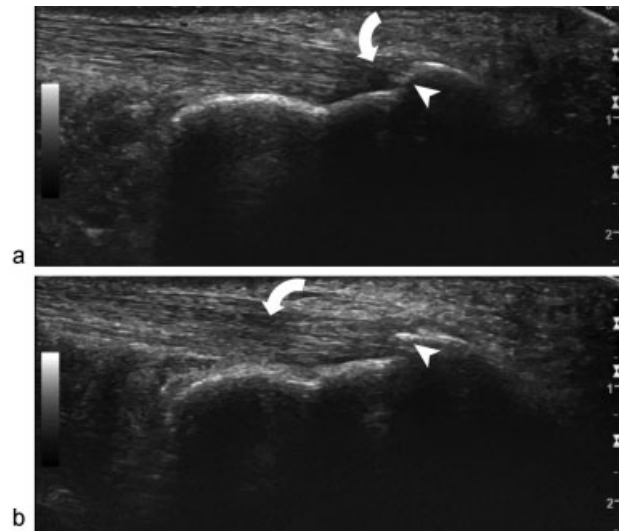

Fig. 3 A 39-year-old woman with left heel pain. (**a**) Longitudinal ultrasound (US) image of the area of maximal tenderness reveals decreased echogenicity of the normal fibrillar architecture of the Achilles tendon (curved arrow) adjacent to a small enthesophyte (arrowhead), compatible with a low-grade partial-thickness tear at the insertion. (**b**) Comparison longitudinal US of the contralateral asymptomatic right Achilles tendon in the same patient also reveals an enthesophyte (arrowhead), but the tendon insertion remains intact. There is mild tendinosis with slight loss of the fibrillar architecture just above the insertion (curved arrow).

Muscle

Anatomy and Biomechanics

Muscle shares the same hierarchical organization as peripheral nerves, tendons, and ligaments: fibers bundled into fascicles surrounded by layers of connective tissue. The muscle fiber is a specialized multinucleated cell that consists of multiple myofibrils. The two types of muscle fibers are type 1 fibers, slow-twitch fibers that produce less power but resist fatigue, and type 2 fibers, fast-twitch fibers that generate more power, are able to utilize anaerobic metabolism, and are more prone to injury.[9,14] The myofibrils that make up the fiber are constructed of repeating adenosine triphosphate–dependent contractile units called sarcomeres. The interaction between actin and myosin as they glide over each other within the sarcomere is the foundation of muscle movement.[15] Voluntary muscle contraction is driven at the neuromuscular junction. Motor neurons branch into several small nerve endings that are tightly coupled to specialized regions of sarcolemma called junctional folds.[16] Electrical impulse is transduced across the neuromuscular junction via diffusion of acetylcholine across the synaptic cleft.[16] Action potentials generated across transverse tubules in the muscle fiber result in release of calcium from sarcoplasmic reticulum and ultimately actin-myosin cross-bridging.[17]

The orientation of the fascicles in a given muscle determines the macroscopic morphology of its myotendinous junction. The two principal fascicle orientations found in skeletal muscle are oblique and parallel.[15] Obliquely arranged fascicles predominate throughout the body and

produce pennate muscles with multiple muscle fibers inserting at an angle along a considerable length of the tendon. Depending on the number of sides or planes of fasciculi converging on the tendon, unipennate (e.g., biceps femoris), bipennate (e.g., indirect head rectus femoris), or multipennate (e.g., deltoid) muscles are constructed. When the fascicles are oriented parallel to the force of contraction, a straplike (e.g., sartorius) or fusiform (e.g., biceps brachii) muscle is observed,[18] with a shorter zone of transition from muscle to tendon. The fascicular architecture governs a muscle's range of motion, velocity, and force generation.[15] In general, parallel muscles maximize muscle fiber length and range of motion at the expense of power. Conversely, pennate muscles maximize force generation but are less able to lengthen and shorten. The relationship between muscle force and velocity is reciprocal. Hence slow contractions (i.e., eccentric or lengthening contractions) result in higher tension and thus more muscle damage. Fast contractions (i.e., concentric or shortening contractions) produce less force and consequently less muscle damage.[15]

Muscles are invested by fascia. At the level of muscle fibers, the surrounding fascia is a loose connective tissue called endomysium. Similar loose connective tissue fascia envelops the muscle as a whole (epimysium) and forms thin layers between nearby muscles to allow for movement. As one progresses more peripherally, deep fascia is encountered. Deep fascia is made of dense connective tissue that separates functional groups of muscles into osteofascial compartments.[19] Although usually considered in the context of compartment syndrome or the spread of infection or tumor, fascia also plays an important role in muscle function (►Fig. 4). Muscle transmits force not only to tendon, but also to both intra- and extramuscular fascia, constricting its fascial envelope and thereby restricting volume changes.[20] This myofascial force transmission is important for integrating muscle activity and load transfer.[19]

Imaging: Normal

On MRI, normal muscle is relatively symmetrical in volume, with smooth convex peripheral margins. On T1-weighted images, normal muscle exhibits distinctive architecture related to which shows linear, branching, or feathery patterns of interspersed fat. On fluid-sensitive sequences, muscle fibers maintain low-intermediate signal. Interspersed collagen, including tendon, is also low signal on all sequences. Normal muscle fasciculi are hypoechoic on US. Between the fascicles and surrounding the whole muscle, linear echogenic layers of perimysium and epimysium are seen. Interspersed within the hypoechoic fasciculi are small echogenic fibrofatty septa that appear linear on longitudinal images and dot-like on transverse images. The myotendinous junction can be observed as a discrete echogenic structure where muscle and the tendon converge.[9]

Imaging: Pathologic Conditions

Muscle pathology has a limited range of imaging appearances, demonstrating either a focal mass within the muscle, edema, or atrophy, or some admixture of these. Differential diagnosis is aided by noting whether the pathologic condition is focal or systemic. Focal conditions include denervation, direct or indirect injuries, infection, ischemia, and tumors. Systemic muscle disorders include congenital, autoimmune, metabolic, drug-induced, and degenerative disorders.[18] Many of these disorders are discussed in other articles in this issue; the discussion here emphasizes focal traumatic abnormalities.

Injuries to muscle include myotendinous or myofascial strain or tear, delayed-onset muscle soreness, contusion, and laceration. Clinical grading is based on the presence of pain, weakness, and/or loss of function. Grade 1 injuries are characterized by pain alone, whereas grade 2 injuries are defined by pain with weakness, without loss of function. Grade 3 injuries include loss of function, reflecting extensive myotendinous damage or complete tearing of the myotendinous junction
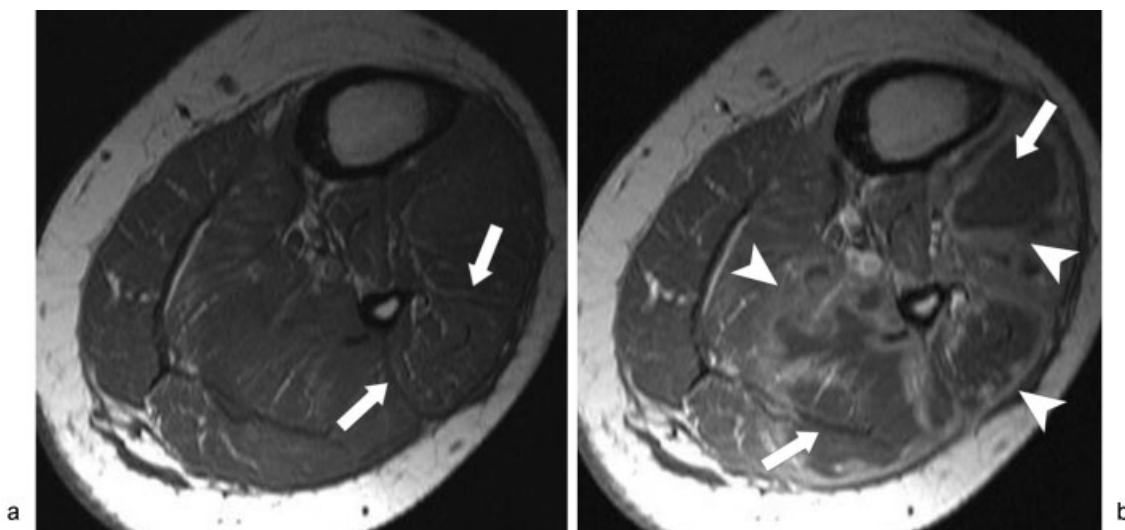

Fig. 4 A 34-year-old woman with a desmoid tumor of the vulva with postoperative swelling of the left lower extremity and peroneal motor deficit. (**a**) Axial T1-weighted MR image of the left leg shows bulging of the anterior and posterior intermuscular septa (arrows) at the lateral compartment due to enlargement of the peroneus longus muscle. (**b**) Axial T1-weighted postcontrast MR image shows extensive fascial enhancement at the periphery of the anterior, lateral, and posterior compartments of the leg (arrowheads) with disordered enhancement of the muscles themselves (arrows) related to multifocal compartment syndrome with myonecrosis.
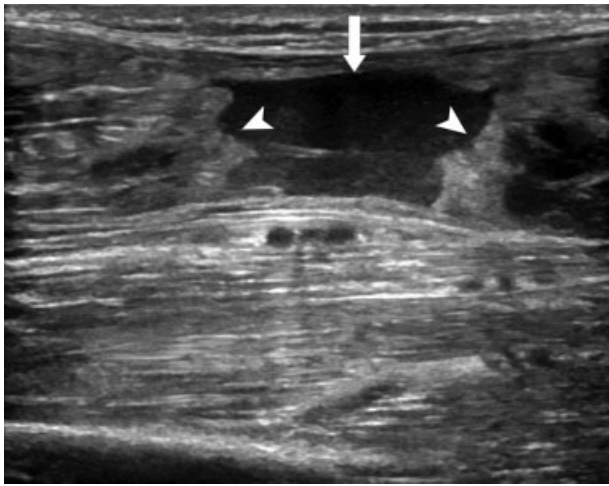
Fig. 5 A 24-year-old man with a visible and palpable defect in the midportion of the upper arm following a direct blow. Longitudinal ultrasound image demonstrates a defect within the biceps brachii muscle (arrow), with interposed fluid and hemorrhage within the defect. The free edges of the torn muscle are easily seen (arrowheads).

(►**Fig. 5**).[9] Newer grading systems of muscle injury use far more stratified and comprehensive grading than the traditional three-grade classification in an attempt to provide more accurate prognostication of recovery time. Current grading systems, which rely on imaging findings on sonography and MRI, are evolving rapidly. Commonly used comprehensive systems include the Munich Consensus Statement, British Athletics Muscle Injury Classification, and FC Barcelona/Aspetar Grading System.[9] All emphasize injury stratification based on etiology, precise anatomical localization, and quantitative assessment of the extent of myotendinous damage.

The most commonly encountered injury in clinical practice is muscle strain, an acute stretching injury of the myotendinous or myofascial junction, typically affecting young adults involved in athletic endeavors. Strain injury of the myofascial junction is less common than at the myotendinous junction. Differentiation of a myofascial junction injury from a myotendinous junction injury can be difficult when these interfaces are in close proximity. Muscles particularly predisposed to strain are long, cross two joints and therefore experience two separate moment arms, perform predominantly eccentric contraction, and have a higher proportion of type 2 fibers.[4] Additionally, pennate muscles generate more force than parallel muscles and are at considerably increased greater risk for muscle strain. Strains in unipennate muscles are associated with prominent fluid along the surface of the muscle where the tendon resides. Bipennate strains have a myotendinous junction located deep within the muscle and exhibit minimal epimysial fluid. In multipennate muscles, injury may be limited to one, a few, or all of its discrete muscle-tendon segments, producing a range of appearances (►**Fig. 6**).

The imaging appearance of muscle strain varies with the severity of the injury. Mild injuries exhibit fluid and hemorrhage tracking along the fasciculi of the muscle, without gaps in the muscle tissue.[4] The injured muscle may appear normal or show only ill-defined increased echogenicity on US.[9] T1-weighted MR images show normal muscle architecture, whereas fluid-sensitive images show a feathery pattern of intramuscular edema centered around a myotendinous junction, either within or at the surface of the muscle depending on the muscle's architecture. In distinction to muscle strain, denervation-related muscle shows uniform muscle signal abnormality without myotendinous localization, absent
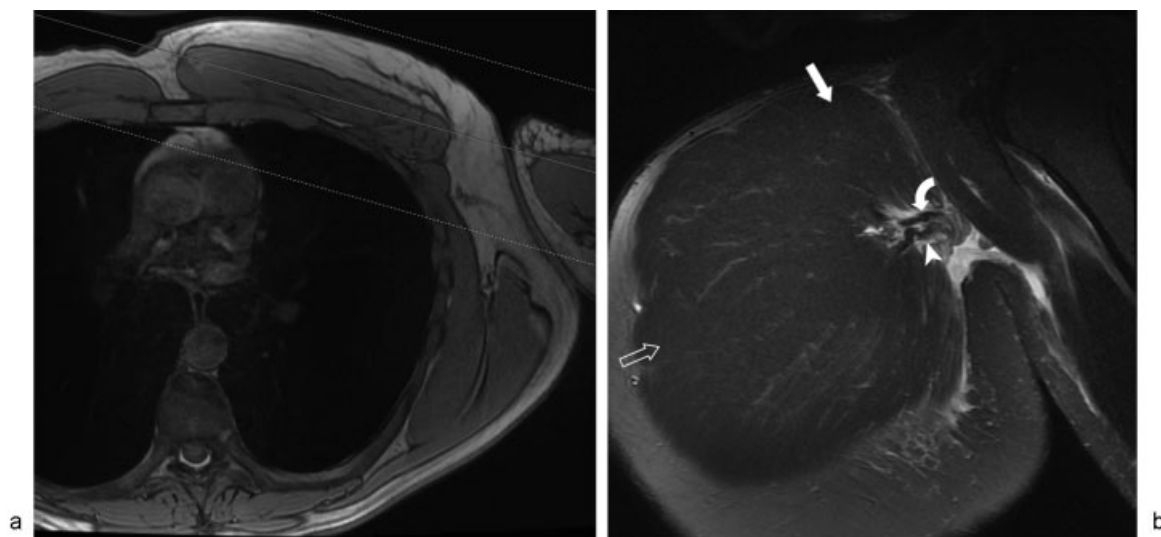

Fig. 6 A 45-year-old man with left shoulder injury after weight lifting. (**a**) Axial gradient-echo localizer MR image of the left chest shows the imaging plane used to obtain oblique coronal MR images. (**b**) Oblique coronal T2-weighted fat-saturated MR image of the left chest shows a high-grade tear of the pectoralis major tendon with torn and retracted tendon fibers from upper (curved arrow) and lower (arrowhead) sternal head segments; corresponding sternal head muscle belly (open arrow) is shown. Some fibers from the clavicular head (solid arrow) remained intact. This patient underwent open repair and had an excellent recovery.

fascial edema, and a distribution corresponding with a particular nerve.

As the injury becomes more severe, architectural distortion becomes apparent, with focal regions of fluid accumulation within the muscle, often associated with irregularity, thinning, and laxity of tendon fibers.[4] Intramuscular hematoma formation may be evident on both MRI and US, particularly in severe injuries with extensive tissue damage (►Fig. 7). In a severe injury resulting in a complete tear of the myotendinous junction, MRI and US show retracted muscle with separated tendon margins, with hemorrhage/fluid separating the torn ends. In general, complete tears of the myotendinous junction result in significant loss of function and may require surgical reconstruction.[21] As the tissues heal, fluid becomes less prominent, and the damaged tissues start to heal or be replaced with fibrofatty tissue, although there is a poor correlation between these healing changes and the clinical return of muscle function. Dynamic muscle contraction evaluation with US may be more helpful to assess healing than assessment of signal alterations following structural myotendinous injury.[9]

Delayed-onset muscle soreness is a milder overuse injury than strain and involves the entire muscle rather than localizing to the myotendinous interface. It is caused by excessive eccentric exercise resulting in direct injury to the sarcomere.[22] Unlike an acute muscle strain, pain tends to develop 1 to 2 days following physical activity.[9] On MRI, diffuse edema is seen in the affected muscle; such signal alterations can persist for weeks. Contusions result from direct trauma to the muscle. These injuries occur at the point of impact and are not localized to the myotendinous junction. The superficial muscles of the lower extremity, particularly the quadriceps, are typically affected. In addition to muscle edema, interstitial hemorrhage, and hematoma formation, ancillary findings may include skin edema and bone contusion.

Tendon

Anatomy and Biomechanics

Normal tendon is the strongest link in the muscle-tendon-enthesis unit, able to tolerate greater loads than muscle and bone. Failure of the normal tendon can result from a single inordinate stress, but tendon failure is far more commonly related to alterations in its biomechanical resilience secondary to disease induced by repetitive overuse. Disordered healing of tendon damage from any cause results in a decrease in the tendon's ability to tolerate load and deformation, allowing macroscopic failure.[6,23]

The biomechanical properties of normal tendon are best expressed as a stress-strain curve (►Fig. 8). Stress or load is defined as the force per unit area applied to a material. Strain or deformation is the percentage change in the shape of the material in response to an external load. The slope of the curve expresses the stiffness of a material. The first portion of the stress-strain curve represents the "toe" phase, where the resting crimp or wavy shape of collagen is straightened. Once crimp is removed, the slope of the stress-strain curve increases, and more force is needed to elongate collagen fibers. It is during this linear phase that microscopic damage occurs following ∼ 4% of fiber elongation. Once a collagen fiber is elongated > 10%, its failure point is reached.[23] Submaximal loading can lead to tendon fatigue and failure because of the tendon's viscoelastic properties, allowing creep (increased strain with constant stress over time) and stress relaxation (decreased stress with constant strain over time).
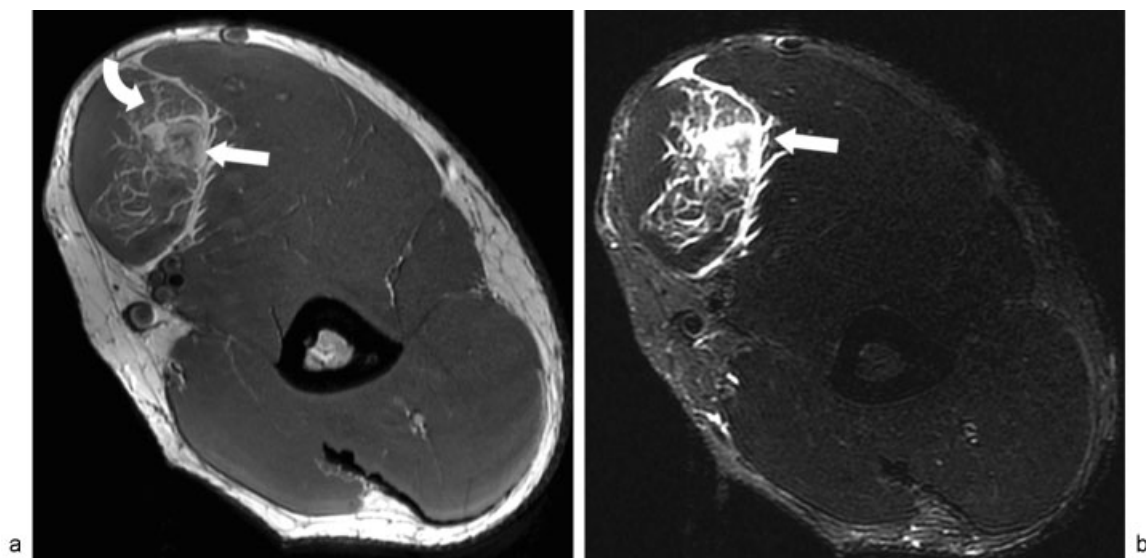

Fig. 7 A 28-year-old professional athlete with right biceps muscle strain. (**a**) Axial T1-weighted MR image of the right arm shows architectural distortion of the lateral head of the biceps brachii muscle (curved arrow) with high signal intensity, consistent with hemorrhage, and a more focal hematoma at the myotendinous junction (straight arrow). (**b**) Axial short tau inversion recovery MR image shows high signal intensity at the myotendinous junction corresponding with the areas of hemorrhage on the T1-weighted MR image, compatible with moderate-grade muscle strain.
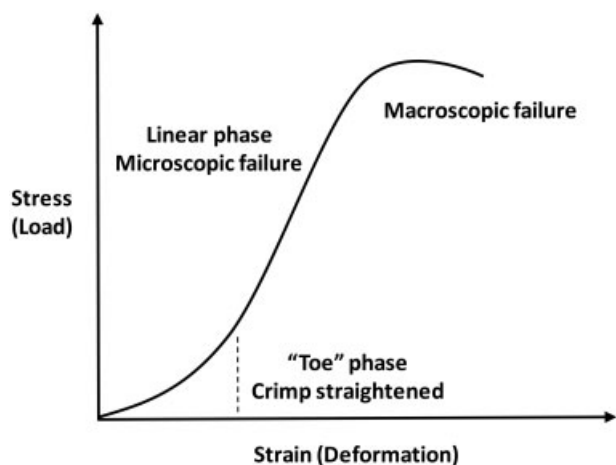
Fig. 8 Stress-strain curve of collagen. The mechanical behavior of collagen within tendons can be understood by graphically depicting its load-deformation relationship. The first region of the curve reflects the "toe" phase when redundancy in collagen fibers is removed. Subsequently, the linear phase shows a steepened slope when collagen fibers are stretched longitudinally. In this second region, some collagen fibers are damaged, resulting in microscopic failure. The peak of the curve corresponds to macroscopic failure when there is significant irreversible macroscopic damage to collagen fibers.

The strength of a tendon is a function of its collagenous organization. The smallest structural unit within tendon is tropocollagen that aggregates to form microfibrils. Microfibrils are aggregated into fibrils, fibrils into fibers, fibers into fiber bundles, and fiber bundles into fasciculi that combine to form the tendon proper.[1] A loose connective tissue, called endotenon, surrounds the fiber bundles and fasciculi, allowing for independent sliding of fasciculi and fibrils, a process aided by a molecule called lubrican.[24] Endotenon is continuous with epitenon, the layer of connective tissue that surrounds an entire tendon.[1]

Tendons are classified into two types based on whether they are enveloped within a tendon sheath or are only partially surrounded by a paratenon.[23] A tendon sheath is a double-layer synovial envelope typically located around tendons where there are bony pulleys or retinaculae.[1] Paratenon, a loose connective tissue that allows for freer tendon movement, is usually found adjacent to a tendon that elongates in a linear fashion. A prototypical paratenon is seen related to the Achilles tendon. When paratenon is combined with epitenon, it is sometimes referred to as peritenon or peritendon.

Tendon vascularity depends on both intrinsic and extrinsic sources. Intrinsic blood supply is derived from the myotendinous junction and enthesis.[25] Extrinsic blood supply varies depending on whether the tendon is invested by a paratenon or tendon sheath. Tendons with a paratenon receive blood supply at several points along the tendon and are considered vascular tendons.[23] Tendons with a synovial sheath are considered avascular because only a few vessels reach the tendon from the sheath via structures called vinculae. Diffusion of substances through synovial fluid is an important source of nutrition to such tendons, particularly when vascularity is impaired.[23] Typical regions of diminished vascularity include sites of friction, compres-

sion, or torsion.[26] Some tendons, such as the supraspinatus and Achilles, exhibit consistent regions of relative avascularity, termed critical zones, although these do not directly correspond to common sites of tendon failure.[27]

Imaging: Normal

On MRI, normal tendons exhibit low signal on conventional clinical sequences. Due to magic angle phenomenon, tendons show increased signal on low echo time (TE) sequences when oriented near 55 degrees to the main magnetic field,[28] simulating tendinosis and tearing, although the tendon caliber and contour remain normal. Signal alterations related to tendinosis and magic angle artifact do not persist on long TE sequences, whereas frank tendon tears typically show persistent fluid-like signal. On US, interfascicular interfaces within the tendon act as specular reflectors, producing an echogenic fibrillar pattern in the long and short axes (►**Fig. 9**).[10] Anisotropy can produce an artifactual appearance of tendinosis or tendon tearing when the angle of insonation is not perpendicular to the tendon, resulting in diminished return of sound energy to the transducer.

Imaging: Pathologic Conditions

Tendon disorders most commonly relate to degeneration and tearing.[23] Tendinosis refers to the array of histologic changes that take place as the tendon degenerates. Common associations with tendinosis include aging, male gender, obesity, overuse, hormone replacement, and oral contraceptives.[29] Tendinopathy is a clinical entity that refers to tendon pain, swelling, and decreased function. Because structural changes of tendinosis may be asymptomatic, the processes of tendinosis and tendinopathy do not necessarily coexist.[10] Unless intratendinous inflammation is present, the term *tendinitis* should be avoided.[30]

Tendinosis is thought to be secondary to high-dose cyclical strain, corresponding with the linear or microtear phase of the tendon's stress-strain curve. Oxidative stress and cartilage gene pathways are upregulated resulting in a decrease in type 1 collagen production, increase in immature cartilaginous matrix, and destruction of the metalloproteinase matrix. During the repair attempt, vascular infiltration and nerve regeneration occurs.[29] Multiple histologic patterns of tendinosis have been
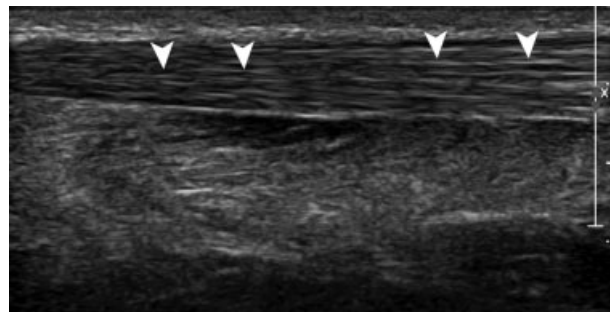

Fig. 9 A 33-year-old man with a normal Achilles tendon. Longitudinal ultrasound image of the left ankle shows uniform echotexture and thickness of the Achilles tendon. The echogenic fibrillar pattern reflects interfascicular interfaces within the tendon (arrowheads). Dynamic and color Doppler assessments were normal.

described including hypoxic, hyaline, mucoid, fibrinoid, lipoid, calcifying, and fibrocartilaginous.[31] These forms often coexist, resulting in varying and overlapping imaging appearances.[32] Of these processes, mucoid degeneration dominates, resulting in tendon thickening and a gentle graying in signal intensity on MRI. Histologic studies showed mucoid degeneration to be slightly hyperintense to normal tendon on T2 and proton-density-weighted fat-suppressed sequences,[32,33] although its T2 signal does not approach that of fluid. On US, tendon degeneration demonstrates thickening with loss of a normal echogenic fibrillar pattern. Neovascularity can be seen as hyperemia on color Doppler (►Fig. 10).[10] Severe tendon degeneration can evolve to form intratendinous ossification[34]

that prototypically involves Achilles, quadriceps, and patellar tendons.

Tendon tears typically show fluid signal within a gap in the tendon that is high signal on fluid-sensitive MR images and anechoic on US. The free edges of the torn tendon usually show preexisting tendinosis (►Fig. 11).[10] The size of the gap, degree of myotendinous retraction, and secondary muscle atrophy are important additional features to describe. Small tears may not result in a significant gap and can be challenging to identify. Dynamic US examination can be helpful to assess torn ends that are apposed to one another. If the tear starts to heal with scarring, it may become obscured and difficult to appreciate because the fluid is replaced by scar and granulation tissue.[35]

The terminology for describing tendon tears lacks consistency, and various descriptors are used at different anatomical regions. At the shoulder, surfacing tendon tears are typically described by the extent of tissue damage involving both the thickness and the width of each rotator cuff tendon. A full-thickness tear perforates through the entire tendon substance from the articular side to the bursal side, whereas a full-width tear involves the entire articular or bursal surface of the tendon. A complete tear affects the entire thickness and width of a tendon. In the case of the rotator cuff, a massive cuff tear is a full-thickness tear that involves the full width of two continuous tendons or a full-thickness tear whose width is > 5 cm. Tears can also be described based on their precise location as affecting the footprint, critical zone, or myotendinous junction.

Tears may be categorized as intrasubstance or surfacing; those that do not surface may not be recognized at
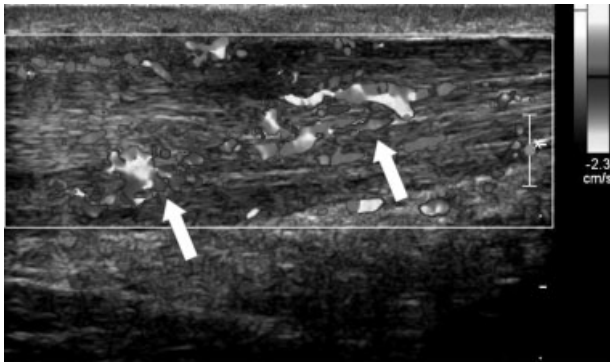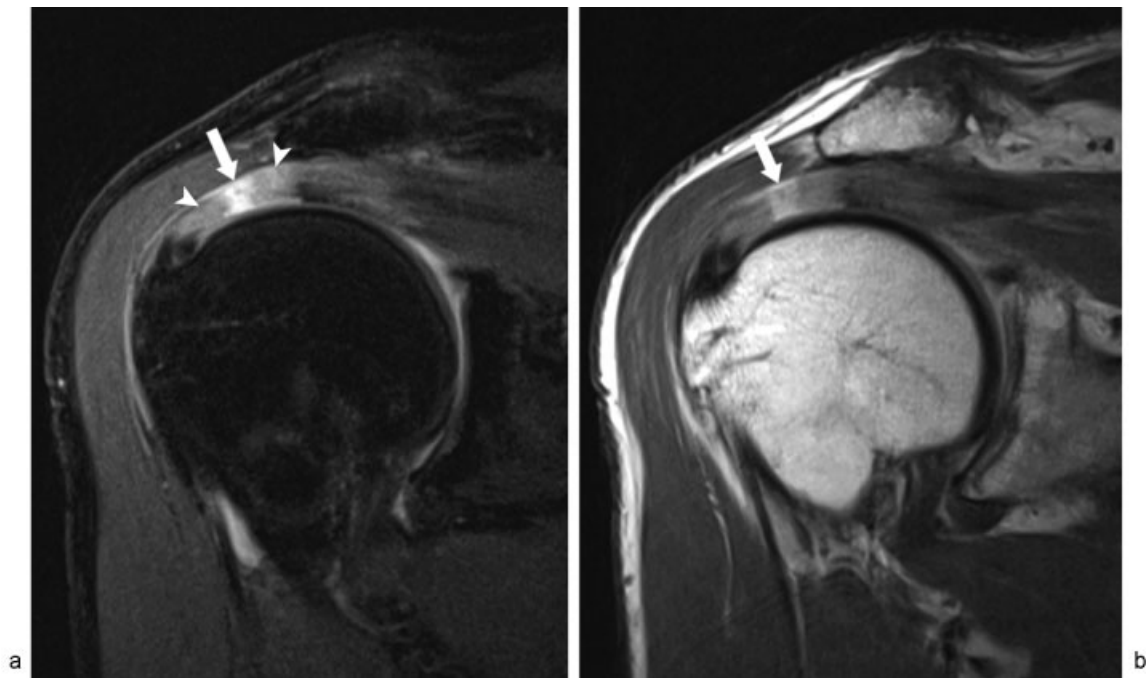

Fig. 10 A 71-year-old man with left Achilles tendinosis. Longitudinal color Doppler US image of the left Achilles tendon shows fusiform thickening of the tendon with marked hyperemia (arrows), indicative of neovascularization. The tendon exhibited multiple focal areas of intrasubstance tearing.

Fig. 11 A 79-year-old man with a supraspinatus tendon tear. (**a**) Coronal T2-weighted fat-saturated MR image of the right shoulder shows a high signal gap (arrow) in the distal supraspinatus tendon with tendinosis of the torn tendon edges (arrowheads), indicating a full-thickness tear. Note the retraction of the myotendinous junction. (**b**) Coronal proton-density MR image shows high signal intensity within the gap at the torn tendon ends that could represent hemorrhage, scarring, or granulation tissue. With ultrasound or MR arthrography, this diagnosis can be differentiated.

arthroscopy. Intrasubstance tears are caused by delamination, splitting the tendon horizontally along the path of least resistance between collagen fasciculi. These appear as focal fluid accumulation within the tendon. Delamination can also take place with surfacing tears, allowing a conventional tear to extend deeply into the muscle, sometimes resulting in the formation of an intramuscular cyst. In the rotator cuff, intramuscular cysts occur most commonly in the infraspinatus, followed by the supraspinatus.[36] Although most intramuscular cysts are related to delaminating tears,[37] cysts without any identifiable rotator cuff tear at MRI and arthroscopy have been described.[36]

Common inflammatory disorders affecting tendons include inflammation of the tendon's synovial covering, resulting in paratenonitis or tenosynovitis, and calcium hydroxyapatite deposition disease. Calcium hydroxyapatite deposition has an unclear pathogenesis. It is most common at the shoulder and can be asymptomatic, result in mechanical symptoms, or produce inflammation as the calcification extrudes from the tendon, inciting inflammation of the adjacent soft tissues, bursae, bones, and/or muscles.[38] On MRI, calcific deposits appear as ovoid foci of intratendinous low signal on all sequences. As they start to extrude, secondary surrounding inflammatory changes become apparent as the calcification itself begins to resorb (►Fig. 12). Calcium deposits are echogenic on US, with surrounding hyperemia during the symptomatic phase.[10]
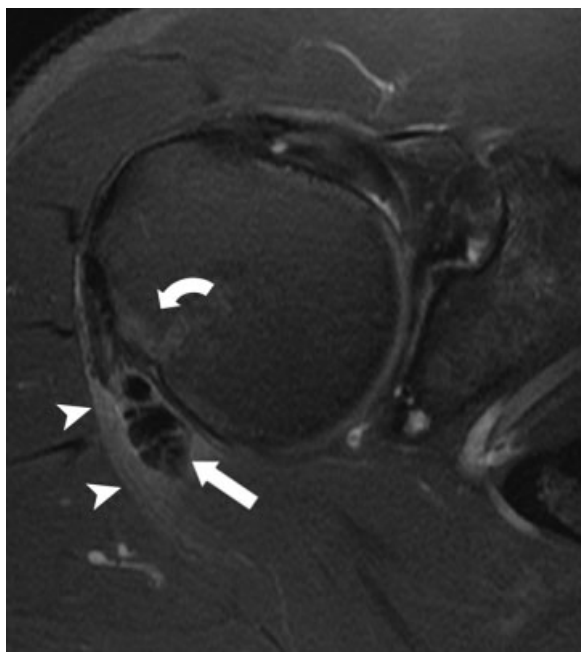

Fig. 12 A 48-year-old man with right shoulder pain. Axial proton-density MR image of the right shoulder shows several large focal calcifications (straight arrow) deep to the insertion of the teres minor tendon. There is associated edema in the soft tissues (arrowheads) and bone marrow edema of the posterior humerus (curved arrow). These findings are consistent with active calcific tendinitis related to extruded calcium hydroxyapatite deposition.

Enthesis

Anatomy and Biomechanics

The enthesis is a specialized zone of fibrous or fibrocartilaginous connective tissues located where tendons, ligaments, and joint capsules attach to bone.[39] In the context of tendon attachments, it functions to transmit contractile forces generated by the muscle to the skeleton.[2] Fibrous entheses are found where a tendon attaches to either periosteum or directly to bone itself, typically over a broad surface of the diaphysis. Fibrous entheses are typically associated with short tendons, and the muscle force is typically transmitted over a large area; such an arrangement is evident at the gluteus maximus insertion at the posterior femur. Fibrocartilaginous entheses occur where tendons insert into apophyses or epiphyses that lack periosteum. These attachments occur through an organized transition from tendon to bone via four zones of tissue:
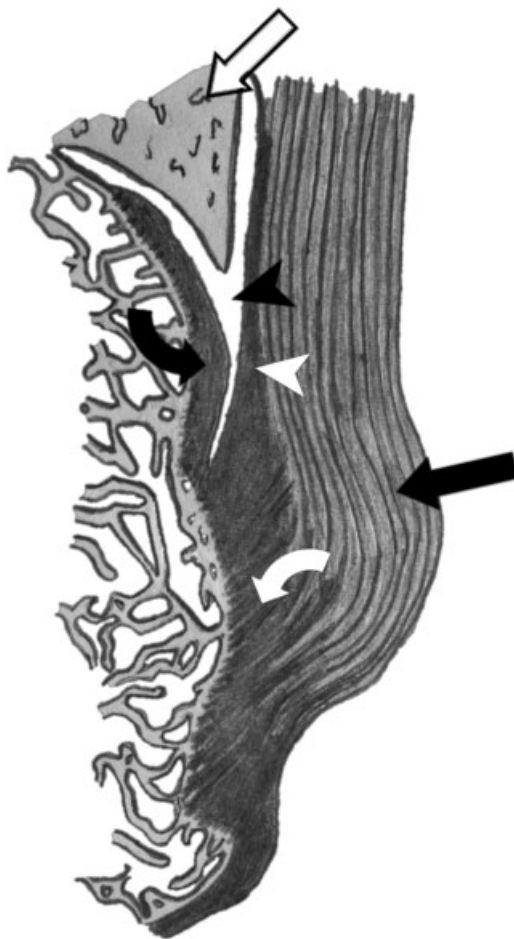

Fig. 13 Diagram of the Achilles enthesis organ. The Achilles tendon (black arrow) insertion is characterized by a fibrocartilaginous enthesis (curved white arrow) along the posterior aspect of the calcaneus. Adjacent to the enthesis, sesamoid fibrocartilage lines the deep surface of the tendon (white arrowhead), and periosteal fibrocartilage lines the superior tuberosity of the calcaneus (curved black arrow). These opposing fibrocartilages are separated by the retrocalcaneal bursa (black arrowhead), covered by synovium. The tip of Kager's fat pad (black border arrow) protrudes into the retrocalcaneal bursa. Collectively, these structures form the enthesis organ and reduce stress concentration at this hard-to-soft tissue interface.

dense fibrous connective tissue, uncalcified fibrocartilage, calcified fibrocartilage, and finally bone itself.[2] This zonal arrangement, typified by the Achilles insertion, reduces stress concentration and decreases risk of failure. Some muscles, such as the deltoid, attach to the skeleton without any discrete tendon via specialized perforating muscle fibers.[2]

Benjamin et al proposed the concept of an "enthesis organ" that encompasses the entire array of anatomical structures that support the dissipation of stress at the enthesis.[40] In addition to the tendon insertional tissues themselves, the enthesis organ includes nearby bone fibrocartilage, zones of tendon fibrocartilage, and supporting soft tissue structures including bursae, fat pads, and synovium. Enthesis organs can be articular or extra-articular; both demonstrate sesamoid and/or periosteal cartilage or fibrocartilage adjacent to a synovial cavity. Articular enthesis organs are found where tendons attach within a joint cavity (e.g., popliteus) or join a joint capsule (digital extensors). Extra-articular enthesis organs show a subtendinous bursa situated between the tendon insertion and bone (e.g., biceps brachii and patellar). The prototypical extra-articular enthesis organ is the Achilles tendon insertion (►**Fig. 13**). The enthesis organ concept has been used to explain the spectrum of abnormalities occurring adjacent to entheses including subtendinous bursitis, periostitis, and synovitis.[40]

Imaging: Normal

The bone at an enthesis should appear smooth, without cortical irregularity, disruption, or proliferation. On MRI, the normal enthesis shows an intact cortex and normal bone marrow signal, and the adjacent tendon is uniform in thickness and signal intensity, without edema in the adjacent perientheseal soft tissues.[41] On US, the bone profile should be smooth without cortical defects or enthesophytes, the adjacent tendon should have normal thickness and fibrillar appearance, and the nearby soft tissues should be free of fluid accumulation, fibrosis, and calcifications.[42]

Imaging: Pathologic Conditions

Enthesopathy refers to any pathologic condition affecting the enthesis including traumatic, degenerative, inflammatory, metabolic, and endocrine disorders.[43] Trauma-related enthesopathy typically presents as an avulsion injury in children and young adults, either related to acute trauma or repetitive overuse, and commonly involve unfused apophyses.[2] Acute apophyseal avulsions most commonly affect the pelvis, whereas typical sites of repetitive microtrauma affect the entheses at the distal patella (Sinding-Larsen-Johansson syndrome) and tibial tuberosity (Osgood-Schlatter disease) (►**Fig. 14**). Separated cartilage and/or osseous fragments can be variable in size, ranging from small fragments to complete apophyseal separation. Because the apophysis is unossified in the child, radiographs may underestimate the degree of injury.[44] In adults, avulsion fractures at the enthesis require significant trauma and are often seen in the setting of underlying disorders such diabetic neuropathy, rheumatoid arthritis, hyperparathyroidism, and steroid use.[2,43]

In degenerative enthesopathy, repetitive biomechanical stress at the enthesis results in tissue degeneration without inflammation. Common sites of enthesopathy related to overuse include the greater trochanter, greater tuberosity,

Fig. 14 A 14-year-old young man with Osgood-Schlatter disease. (**a**) Lateral radiograph of the left knee shows fragmentation of the tibial tubercle (arrowhead), edema at Hoffa's fat pad, and overlying soft tissue swelling (arrow). (**b**) Sagittal T2-weighted fat-saturated MR image shows bone marrow edema within the tibial tubercle (arrowhead) and adjacent ossicles, reactive fluid within the deep infrapatellar bursa (straight arrow), and thickening of the distal patellar tendon (curved arrow).

Fig. 15 A 43-year-old woman with Achilles tendon insertional pain after running a marathon. Sagittal proton-density fat-saturated MR image of the right ankle shows longitudinal intrasubstance tearing of the Achilles tendon at the calcaneal insertion (solid arrow). There is secondary enthesitis with retrocalcaneal bursitis (arrowhead), calcaneal tuberosity edema (curved arrow), and paratenonitis (open arrow).

lateral epicondyle of the humerus, lower pole of the patella, and calcaneal tuberosity.[45] Degeneration of the tendon substance is associated with a proliferative bony response in reaction to altered biomechanical forces, producing enthesophytes. Enthesophytes develop by endochondral ossification of fibrocartilage at the enthesis, comparable with osteophytes that develop adjacent to hyaline cartilage.[46] When a tendon is absent, as seen with fleshy muscle attach-

ments, similar proliferative regions at their insertions are referred to as traction periostitis.[2] Degenerative enthesopathy may incite a secondary inflammatory response involving adjacent synovial tissue (►**Fig. 15**).[47]

When the primary feature of an enthesophyte consists of inflammation rather than degeneration, the condition is referred to as enthesitis, the hallmark of inflammatory spondyloarthropathies.[41] It can produce various articular and extra-articular manifestations.[43] Articular manifestations include erosions, reactive sclerosis, and irregular bone proliferation at synovial articulations including the sacroiliac and apophyseal joints, as well as abnormalities at the collagenous attachments of the annulus fibrosus in the spine and the fibrous attachments at the sacroiliac articulation. In classic spondyloarthropathy, inflammation at these entheses can ultimately result in ankyloses.[43] Extra-articular sites of enthesitis include the iliac crest, calcaneus, femoral trochanter, humeral tuberosity, and anterior patella.[43]

Although the MRI appearance of degenerative enthesopathy and inflammatory enthesitis overlap, inflammatory changes in the para-articular soft tissues and bone marrow edema are typically more pronounced in enthesitis.[41] For example, inflammatory changes are more pronounced at the distal interphalangeal joints of patients with psoriatic arthritis as compared with those with conventional osteoarthritis.[48] MR findings of enthesitis include tendonitis, soft tissue enhancement, bursitis, joint effusion, cartilage damage, synovitis, bone erosions, marrow edema, cortical thickening, and enthesophytes (►**Fig. 16**).[41,48] Several studies showed US to be a reliable tool for the assessment of peripheral enthesitis.[49] US features include thickening and hypoechogenicity of the enthesis, erosions, enthesophytes, calcifications, bursitis, and hypervascularity on power Doppler. Overall, MRI is more accurate for enthesitis involving axial
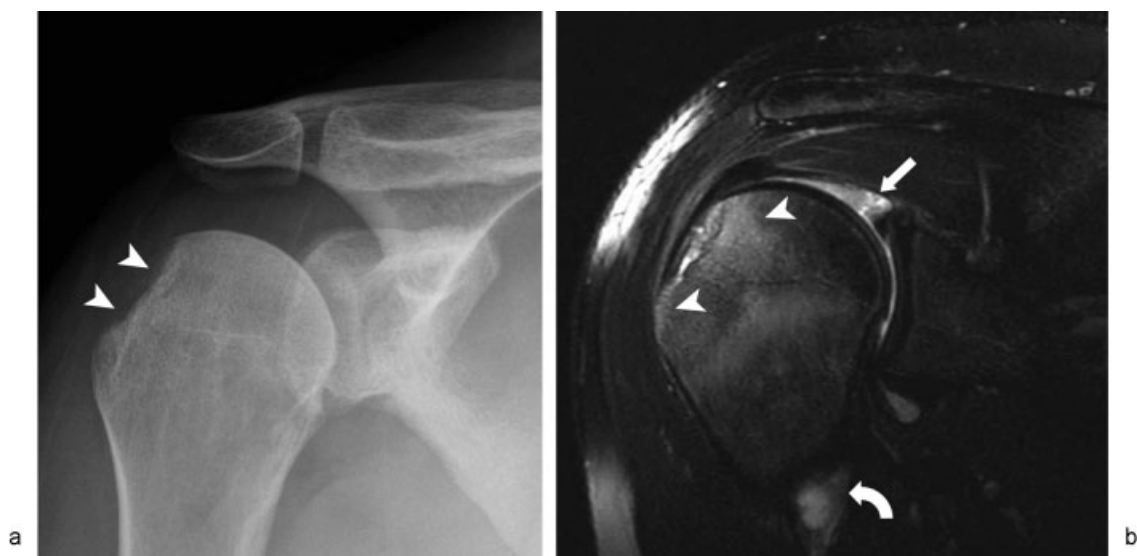

Fig. 16 A 37-year-old man with ankylosing spondylitis and atraumatic right shoulder pain. (**a**) Frontal radiograph of the right shoulder shows ill-defined erosive changes of the greater tuberosity (arrowheads). (**b**) Coronal T2-weighted fat-saturated MR arthrogram image shows reactive bone marrow edema in the greater tuberosity (arrowheads) at the insertion of the rotator cuff tendons, consistent with enthesitis, and should not be confused with a Hill-Sachs fracture. Extensive synovial thickening was observed within the joint (straight arrow) and bicipital tendon sheath (curved arrow), consistent with synovitis.

disease, whereas US may be preferable for peripheral enthesis.[49]

Conclusion

The anatomy and biomechanics of the muscle-tendon-enthesis unit are crucial for understanding its normal and abnormal imaging appearances. Typical patterns of failure vary with age, predominantly affecting the growth plate in children, the myotendinous junction in young adults, and the tendon in the older patient. MRI and US are well suited for diagnosis of the range of pathologic conditions that affect the muscle-tendon-enthesis unit.

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