

Tendon structure, disease, and imaging

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Summary

Tendon imaging plays a critical role in evaluating tendon diseases and injuries including mechanical, degenerative, and overuse disease, inflammatory enthesitis, as well as partial and full thickness tears. Ultrasound and magnetic resonance imaging (MRI), each with unique benefits and limitations, are commonly utilized to assist in diagnosing these diseases and conditions. This review delineates important structural properties of tendon and biochemical changes occurring in tendon pathology. This review also examines commonly injured tendons including tendons of the elbow, tendons of the rotator cuff of the shoulder, hip abductor tendons, patellar tendons, and the Achilles tendon to help clinicians better recognize tendon disease. Finally, this paper introduces several emerging imaging techniques including T₂ mapping, ultra-short echo time MRI, and sonoelastography as ways in which tendon imaging and evaluation may be improved.

KEY WORDS: *imaging, Magnetic Resonance Imaging, tendinopathy, ultrasound.*

Background

Tendons are ubiquitous, dense, regular connective tissue structures in the body and are defined by their anatomical position connecting muscle to bone¹. Ten-

dons consist of cross-linked triple helices of mostly type I collagen and enable the transfer of mechanical load generated by a muscle to bone, resulting in stabilization or movement across a joint^{1,2}. Tendon matrix composition and structure vary considerably based on functional requirements and body position¹. Changes to tendon composition predispose tendinopathy, which includes mechanical, degenerative, and overuse disease, as well as inflammation of the entheses. Tendons may also be fully or partially torn following tendinopathy or trauma. Several advanced imaging modalities play a critical role in comparing normal and abnormal tendon structures. Magnetic resonance imaging (MRI) and ultrasound (US) play a critical role in comparing normal and abnormal tendon structure and in the evaluation of tendon disease². However, new modalities including T₂ mapping, ultra-short echo time MRI, and sonoelastography are emerging as candidates for improving tendon disease imaging.

Normal Tendon Structure

The dry weight of normal tendons generally consists of 65-80% collagen (mostly type I), and includes 1-2% elastin embedded in a proteoglycan-water matrix³. The remainder of the collagen structure includes other types of collagen including type II in cartilaginous zones, types III, IV, and V in the vasculature, and type X in the mineralized fibrocartilage near entheses, among others^{4,5}. The remainder of the tendon structure consists of tenocytes and tenoblasts between collagen fibers and ground substance surrounding the collagen including proteoglycans, glycosaminoglycans, structural glycoproteins, and other small molecules³. Inorganic components form less than 0.2% of dry tendon mass, with calcium representing the most abundant inorganic tendon component⁴. There is increased calcium concentration at the insertion and decreased concentration in the tensional area.

Individual collagen fibrils are organized into collagen fibers, which are subsequently organized into subfascicles. An endotendon sheath encloses subfascicles and fascicles. The number of collagen fibers in each subfascicle may vary considerably from tendon to tendon³. A group of fascicles is organized into tertiary fiber bundles, which are grouped together and enclosed by the epitendon to form a tendon³. Fiber orientation varies depending on location and functional requirements. Collagen fibers are arranged parallel to the axis of the tendon when force is directed along this axis, as in the patellar tendon². Complex fiber patterns, including plait

formation, crossing, and up-tying fibers have been described³. The tendon structure is further complicated when multiple muscles converge on a single tendon, as in the Achilles or quadriceps tendon².

Tendon Pathology

There are two general classifications of tendon disease. Inflammatory enthesitis refers to inflammation of the entheses while tendinopathy refers to mechanical, degenerative, and overuse disease².

Inflammatory Enthesitis

The entheses are the sites of attachment of tendon, ligament, fascia, or joint capsule to bone. The argument has been made that any fibrocartilagineous structures contributing to this role can be considered part of the 'enthesis organ' and may include the entheses, sesamoid, and periosteal fibrocartilage as well as associated bursa and synovial fluid⁶. Inflammatory enthesitis occurs when this area becomes inflamed and is a distinguishing pathological feature of spondyloarthritides⁶. Inflammatory enthesitis may be either painful or asymptomatic and may be visualized as soft tissue swelling. Although obtaining entheses tissue proves difficult, histological changes including macrophage mediated fibrocartilage destruction, lymphocytic infiltration within bone, lymphocyte paucity at the insertion site, and macrophage infiltration have been reported^{6,7}.

Tendinopathy

Historically, pain associated with tendon overuse is referred to as tendinitis. Although the etymology of tendinitis suggests an inflammatory process, research indicates tendons exposed to overuse demonstrate little or no inflammation⁸. Tendinopathy has therefore been supported as a classification of the painful condition occurring in and around tendons accompanying mechanical, degenerative, and overuse disease. Tendinopathy is associated with degeneration and disorganization of the collagen structure and an increase in mucoid, proteoglycan and water content^{2,8-12}. A 10-20 fold calcium concentration increase may also occur⁹. Chemical and molecular changes, including matrix metalloproteinase production, tendon cell apoptosis, chondroid metaplasia of the tendon, and expression of protective factors including insulin-like growth factor 1 and nitric oxide synthetase, have been described in tendon overuse⁹. Macroscopic changes include tendon thickening, loss of mechanical properties, and pain^{2,8,9,12}. Collagen fibril rupture may occur and progress to full-thickness tears. These degenerative changes generally precede a full thickness tear, which does not typically occur in a healthy tendon². Following a tear, there may be ingrowth of vessels into the tendon.

Aging tendons undergo structural and compositional changes including decreased water content and collagenous changes. These changes may predispose both tendinopathy and inflammatory enthesitis¹³. Tendon disease more commonly occurs in hypovascular segments of tendon. Decreased tendon vascularity is associated with aging^{2,14}. These changes play an important role in tendon imaging.

Imaging Techniques

Magnetic Resonance Imaging

MRI is widely used to evaluate tendon disease. MRI is an attractive option as it is non invasive and does not utilize ionizing radiation. Patients with implanted devices have historically been unable to undergo MRI, but as technology improves, MRI is contraindicated in fewer patients¹⁵. Tendon structure plays a critical role in determining appearance in MRI. The water and collagen in tendon are aligned, shortening the T₂ relaxation time to 1-2ms¹⁶⁻¹⁸. The T₁ relaxation time is relatively short at approximately 600ms at 3-T². With conventional MRI techniques, these strong dipole interactions may result in a dark signal void. However, if tendons are examined at 55° to the static magnetic field, this signal becomes detectable. This angle is known colloquially as the magic angle¹⁸⁻²⁰. T₂-weighted imaging sequences have been shown to be more dependent on fiber orientation than T₁²¹. As previously discussed, the orientation of tendon fibers may change along its course, resulting in magic angle distortional effects. This problem may be mitigated by imaging with a sufficiently long echo time². A stronger magnetic field may also be useful in clarifying the signal noise from tendon by improving image resolution and increasing the signal-to-noise ratio²².

An increase in signal intensity on T₂ weighted imaging sequences is often the first sign of tendon abnormality on MRI. Thickening of the tendon may also be seen². MRI of inflammatory enthesitis demonstrates tendon enlargement, loss of the normal flattened hypointense (dark) appearance, focal thickening, and rounded configuration at the insertion site²³.

Although the appearance of a full thickness or partial tear is variable, a hyperintense (white) signal may be seen at the site of the tear or in the surrounding tissue (Fig. 1). Complete rupture of the tendon and displacement of adjacent features may also be seen (Fig. 2). Scarring can obscure this effect and result in an intermediate signal². Fatty tissue infiltration has also been reported following a tendon tear²⁴. Fatty tissue may be differentiated from fluid on T₂ weighted MRI as fluid appears darker than fatty tissue²⁵.

Ultrasound

Much like MRI, US imaging does not utilize ionizing radiations or require an invasive procedure. US examination has the added advantage of allowing ten-



Figure 1. Axial proton-density weighted MRI of severe insertional bicep tendon partial-thickness tear with tendinopathy.



Figure 2. Sagittal proton-density-weighted turbo spin-echo MRI of a full thickness patellar tear with patella alta.



Figure 3. Longitudinal ultrasound of an intact supraspinatus tendon one year after successful repair. The arrow demonstrates the parallel echogenic lines representing the fascicles of the tendon.

don imaging in a weight bearing position and from many angles². However, US is unable to image through bone or gas-containing structures, making visualization of articular or deep-lying tendons difficult. When viewed with a high-frequency transducer, a longitudinal orientation reveals parallel echogenic lines representing the fascicles of a tendon (Fig. 3). Viewed axially, the fascicles appear as echogenic points or lines². Ideal frequency for evaluation depends on tendon depth.

US examination can visualize disruptions in the tendon fascicular pattern. Pathologic changes may include increased space between echogenic fibers and

decreased echogenicity. Similar to MRI, diseased tendon may appear thicker on US². US of inflammatory enthesitis may show loss of normal fibrillarechotexture, blurring of tendon margins, and irregular fusiform thickening²³. Calcification that may occur in tendinopathy can be visualized as bright echogenic regions with US and neovascularization occurring in a tear may be visualized with Power Doppler US²⁶. The imaging timescale is crucial in US as the anechoic fluid buildup that follows an acute tear eventually becomes organized and is difficult to distinguish from associated tendon².

Common Tendon Injuries, Imaging, and Accuracy

Medial and Lateral Epicondylitis

Lateral and medial epicondylitis, known as ‘tennis elbow’ and ‘golfer’s elbow’ respectively, are most commonly seen resulting from repetitive stress²⁷. Lateral epicondylitis occurs when repeated pronation and supination occur in an extended elbow position involving the common extensor tendon while medial epicondylitis occurs following repetitive throwing motions and typically involves the pronator teres and flexor carpi radialis near their condylar insertion². Lateral epicondylitis occurs seven to 10 times more frequently than medial epicondylitis²⁷. On US, medial and lateral epicondylitis appear as tendon enlargement and heterogeneity. Tears may be visualized as hypoechoic regions with adjacent discontinuity. Calcific changes may also be visualized²⁷. MRI findings of tendinopathy for T₁ and T₂ weighted imaging include intermediate signal intensity within the substance of the tendon with or without tendon thicken-

ing²⁷. Partial thickness tears can be visualized as regions of fluid signal intensity within the tendon with tendon thinning while full-thickness tears appear as a fluid filled gap across the substance of the tendon^{2,27}. A fluid signal may also be seen adjacent to both full and partial thickness tears².

In a study by Miller et al. the sensitivity of US for the detection of both lateral and medial epicondylitis ranged from 64 to 82%, while the sensitivity of MRI was between 90 and 100% indicating careful selection of imaging technique and operator is important when detecting epicondylitis²⁸.

Rotator Cuff of the Shoulder

The term “rotator cuff” describes the tendons connecting the infraspinatus, supraspinatus, teres minor, and subscapularis muscles to the humeral head. Traumatic tears of the rotator cuff tend to occur at the tendon-bone junction of the supraspinatus and greater tuberosity of the humerus whereas degenerative tears tend to be seen posteriorly at the junction of the supraspinatus and infraspinatus^{2,29}. Recent evidence strongly suggests that most rotator cuff tears are caused by primary intrinsic degeneration³⁰. Tears of the subscapularis tendon may occur in isolation, however, concomitant tears of the supraspinatus or infraspinatus tendons are more common. Teres minor tendon tears are rare³¹. Partial tears are more common than full thickness tears and tears are more commonly located on the articular surface compared to bursal or intrasubstance locations³².

On MRI, partial tears appear with low/intermediate signal on T₁ weighted images and may be difficult to differentiate from tendinosis². T₂ weighted imaging with fat suppression results in a high signal intensity and is most useful for diagnosing tears³³. The tendon surface may also present with thickening or thinning. Additional signs of a rotator cuff tear include fluid filled defects in the tendon superstructure and fluid superiorly, anteriorly, or inferiorly to a supraspinatus tear. Tendon retraction may or may not occur (Fig. 4)². Hyperdense calcification and bursitis may also be present (Fig. 5)³⁴. On US, full thickness tears may present as focal defects,

however, visualization of the tendon may be difficult or impossible if retraction has occurred (Fig. 6). Partial thickness rotator cuff tears may be hypoechoic or contain heterogeneous echogenicity tendon signals which may be also accompanied by structural flattening of the bursal surface^{2,35}.

A recent meta-analysis examining 65 rotator cuff tear studies in which full and partial thickness tears were evaluated determined the sensitivity for diagnosing a full thickness tear with MRI and US to be 92.1% and 92.3% respectively. The specificity for diagnosing a full thickness tear on MRI and US is 92.9% and 94.4% respectively. This sensitivity of MRI and US for diagnosing a partial thickness tear is considerably lower than a full thickness tear at 63.6% and 66.7% respectively. The specificity for diagnosing a partial tear in MRI and US is 91.7% and 93.5% respectively³⁶. These data suggest that MR and US are comparable in diagnosing full and partial thickness rotator cuff tears.

The accuracy of detecting tendinopathy on MRI and US has also been described. The sensitivity of MRI and US was found to be 55% and 66.7% respectively while the specificity was determined to be 92.7% and 88.4% respectively. Although these data suggest US may be more sensitive and less specific than MRI, more high quality studies are needed.

Gluteal Tendons

The gluteus medius and gluteus minimus tendons insert on the greater trochanter of the hip and have been referred to as “the rotator cuff of the hip”. Both tendons have been implicated in Greater Trochanteric Pain Syndrome (GTPS)^{37,38}. GTPS may mimic other serious conditions including a vascular necrosis and stress fracture. Therefore, accurate imaging and evaluation of these tendinous structures is critical to obtaining an accurate diagnosis³⁹. On MRI, gluteal tendinopathy may be seen as tendon thickening or increased tendon signal intensity on T₂-weighted images without tendon discontinuity or thinning.

MRI permits tendon tear visualization as well. Tendon thinning on T₁ and T₂ weighted images in conjunction

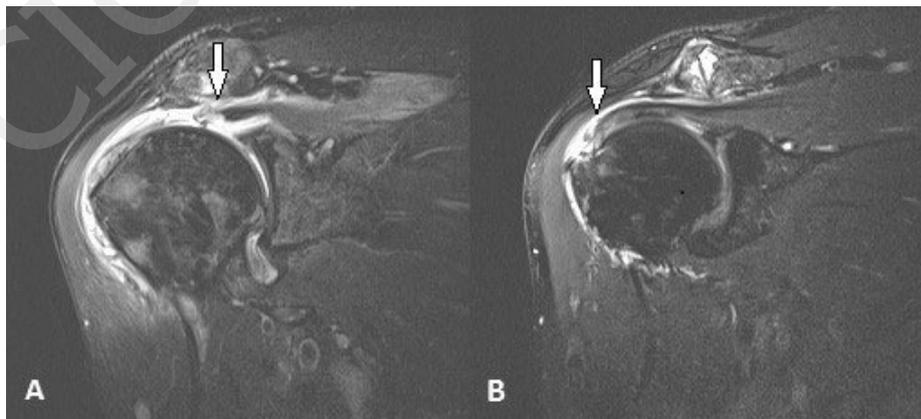


Figure 4. A) Coronal T₂ weighted MRI of rotator cuff demonstrating full thickness supraspinatus tendon tear with retraction to the glenoid. B) Coronal T₂ weighted MRI of the rotator cuff demonstrating a full thickness supraspinatus tendon tear without retraction.

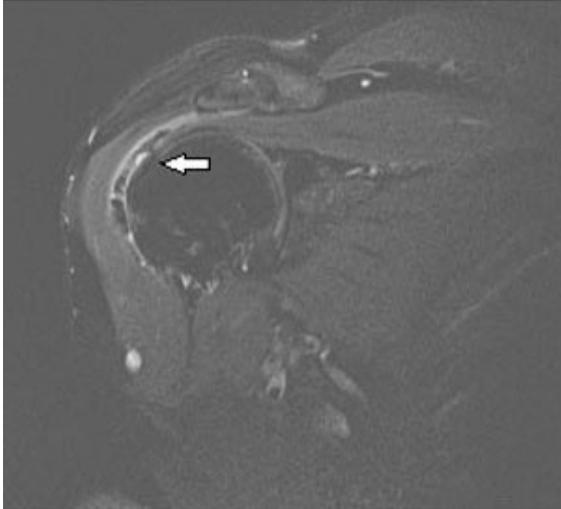


Figure 5. Coronal T₂ weighted MRI of supraspinatus tendosius with reactive subacromial subdeltoid bursitis, intra-substance tear, and hydroxyapatite calcification.

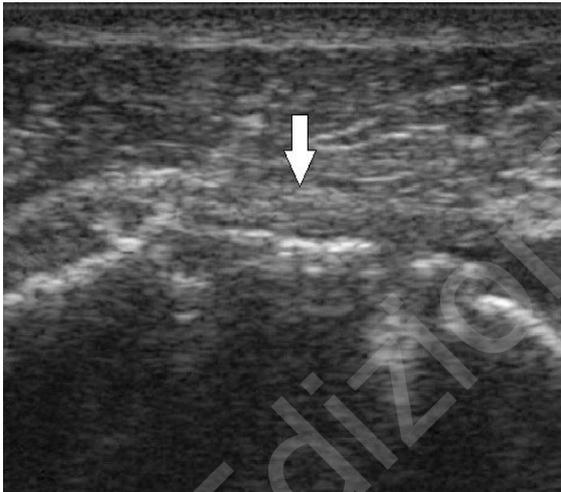


Figure 6. Longitudinal ultrasound of a torn supraspinatus tendon three months after surgery. Due to retraction, the echogenic tendon fascicles cannot be visualized. Arrow indicates where the tendon would normally be visualized.

with high signal intensity in the tendon on T₂ weighted sequences is indicative of a partial thickness tear. Discontinuity of tendon fibers with a high signal intensity on T₂-weighted images are suggestive of full thickness tears³⁸. MRI may also reveal bursal fluid and hypointensity caused by calcification in areas of tendinopathy². Using US, gluteal tendinosis may present as decreased or heterogeneous echogenicity, often with tendon thickening. Tendon tears may manifest as tendon thinning or as anechoic defects within the tendon⁴⁰. Calcification may appear as hyperechogenic foci in the tendon².

A systemic review by Westacott et al. reported that MRI had a sensitivity of 33-100% and a specificity of 92-100% when detecting gluteal tendon tears. In the

same review, US had a sensitivity of 79-100% and positive predictive value of 95-100%³⁷. This data suggests that US may be the preferred imaging technique, although additional well-designed studies are required. Additionally, the sensitivity and specificity of US to detect tendinous pathology is highly dependent on the administering technician³⁷.

Patellar Tendon

Patellar tendon disease is associated with instability and over use, especially accompanying jumping sports and is seen in the deep, medial, or central part of the tendon near its insertion². Normal tendon appears with low signal intensity on MRI, although the patellar enthesis may be hyperintense (white) and US demonstrates the typical echoic fibrillar structure⁴¹. In tendon disease, US demonstrates disruption of this fibrillar structure, hypoechogenicity, and swelling². However, it should be noted that a hypoechoic signal may be seen in asymptomatic patients and clinical assessment is important in the appropriate management of patellar tendon disease⁴². On MRI, focal thickening of the tendon may be seen in conjunction with increased signal intensity². In cases of complete tendon rupture, the tendon appears discontinuous on MRI and may be accompanied by patella alta (Fig. 2). In a study by Warden et al., MRI and US had equivalent specificity (82%) for detecting patellar. Tendinopathy while the sensitivity of US was greater than that of MRI and 87% and 57% respectively^{43,44}.

Achilles Tendon

The combined insertions of the soleus and gastrocnemius muscles on the calcaneus tuberosity form the Achilles tendon. The Achilles tendon produces a low signal on all conventional MRI sequences^{2,45}. On US, normal Achilles tendon appears as a ribbon-like, hypoechoic, tightly packed fibrillar structure^{2,46}. In tendonopathy, the tendon may appear thickened with convexity at the anterior border⁴⁷. MRI may reveal an intermediate signal in T₁ weighted images, and a high signal in or around the tendon in fluid-sensitive MRI sequences, especially in the retrocalcaneal bursa, or as bony edema (Fig. 7)². If the tendon has been completely ruptured, a hyperdense fluid signal may be seen in the tendon space (Fig. 8). US may reveal hypoechogenicity with separation of the fibrillar structure or neovascularization by Doppler flow². Additionally, mechanical tendon disease, visualized as microtearing on US in asymptomatic patients, generally affects the mid-section of a tendon⁴⁸. Obesity has been shown to be an important risk factor for Achilles tendinopathy. Running is also associated with physiologic hypertrophy of the Achilles tendon and overweight runners may precociously develop tendon abnormalities⁴⁹.

Although US has been shown to be both sensitive and specific for diagnosing partial and full-thickness



Figure 7. Sagittal T₂ weight edinsertional Achilles tendinopathy with reactive bony edema pattern (lower arrow) in the calcaneus and retrocalcaneal bursitis (upper arrow).



Figure 8. Sagittal T₂ weighted MRI of the Achilles tendon demonstrating a full thickness tear with fluid noted by arrow.

Achilles tendon tears, MRI is believed to be more sensitive for detecting partial tears based on studies comparing the sensitivity of US and MRI^{11,50}.

Emerging Techniques

Ultrashort Echo Time MRI

In conventional MRI one is limited when it comes to imaging tissues with a T₂ signal that is significantly shorter than 10 microseconds². Using ultrashort echo

time MRI techniques, it is possible to acquire images with echo times as low as a few microseconds. This technique will allow new options to characterize these tissues, including tendon entheses⁵¹.

Sonoelastography

Sonoelastography estimates the hardness or softness of a tissue by measuring the strain caused by tissue compression⁵². It is a noninvasive estimation of tissue stiffness based upon the fact that soft tissue has greater tissue displacement than hard tissue when externally compressed⁵³. This is useful when disease occurs, as there are changes to the biomechanical properties of tendon⁵⁴. Sonoelastography allows calculation and comparison of tissue displacement before and after tissue compression with conventional US equipment but modified software, making widespread implementation more convenient⁵⁵.

T₂ Mapping

T₂ mapping was developed to exploit the sensitivity of MRI to the biophysical properties of cartilage⁵⁶. Unlike anatomic imaging, T₂ mapping is sensitive to changes in the chemical composition and structure of cartilage⁵⁷. T₂ mapping is capable of evaluating the condition of articular cartilage in orthopaedics⁵⁸. Although primarily used to evaluate cartilage, intervertebral discs, and bony structures, the biochemical changes, especially water content changes in tendons that predispose or indicate tendinopathy, could be a future target for T₂ mapping^{2,9,10,59-61}.

Conclusion

Tendon evaluation using ultrasound and magnetic resonance imaging modalities play a crucial role in the identification of normal and abnormal tendon structure. Knowledge of the capabilities and limitations of these methods is important for clinicians regarding diagnosis and prognosis. Clinicians should also be aware of the emerging imaging technologies that may someday assist them to successfully diagnose and treat tendon disease.

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