[Imaging]

Imaging of Tendons

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Both magnetic resonance imaging (MRI) and sonography are well suited to tendon imaging. A normal tendon on MRI demonstrates low signal intensity and on sonography, an echogenic fibrillar pattern. MRI is considered the imaging gold standard, providing an anatomic overview and excellent soft tissue contrast. Sonography is a more rapidly performed examination; it has greater resolution than that of MRI; it allows dynamic evaluation of tendons and muscles; and it can guide percutaneous therapeutic procedures. Moreover, the advent of sonographic extended-field-of-view imaging allows the demonstration of the entire length of a tendon, matching MRI's ability to display a large anatomic region. Sonography should best be considered a focused examination, concentrating on the area of pain and clinical suspicion of pathology, whereas MRI can provide a global assessment of the region of concern. Both modalities demonstrate high accuracy for abnormalities of various tendons. This article reviews normal tendon anatomy and its imaging appearance, as well as the imaging appearances of tendon degeneration and tear.

Keywords: tendon; tendinopathy; magnetic resonance imaging; sonography

Tendon injury is a common sports-related problem that affects both the upper and the lower extremities, and it often has eponyms that describe the offending activity, such as *jumper's knee* and *tennis elbow* . These injuries are the result of repetitive overuse rather than acute trauma; in fact, an estimated 30% to 50% of sports injuries in the United States are related to overuse.³⁰ For example, elite runners have a 57% prevalence of Achilles tendinopathy,³⁷ compared to a 6% incidence in sedentary people.⁵⁰ In addition, overuse injuries can be exacerbated by intrinsic factors, such as muscle imbalance and skeletal malalignments (eg, limb length discrepancies), as well as extrinsic factors, such as training errors and poor equipment.^{29-31,44,50}

Both magnetic resonance imaging (MRI) and sonography are well suited to tendon imaging. MRI is considered the imaging gold standard, providing an anatomic overview and excellent soft tissue contrast. However, sonography is a more rapidly performed examination; it has greater spatial resolution than that of MRI⁵⁵; it allows dynamic evaluation of tendons and muscles; and it can guide percutaneous therapeutic procedures. Moreover, the advent of sonographic extended-field-of-view imaging allows the demonstration of the entire length of a tendon, matching MRI's ability to display a large anatomic region. Sonography should best be considered a focused examination, concentrating on the area of pain and clinical suspicion of pathology, whereas MRI can provide a global assessment of the region of concern. Both modalities demonstrate high accuracy for abnormalities of various tendons.^{2,19,23,25,54}

ANATOMY

Tendons are composed of type 1 and, to a lesser degree, types 3 and 4 collagen fibers that are oriented longitudinally into fascicles and surrounded by dense fibrous connective tissue with little cellular stroma.¹⁴ Tropocollagen, the building block upon which tendons are constructed, is made by specialized fibroblasts called *tenocytes*, and consists of 3 polypeptide chains arranged in a triple-helical formation, aligned end to end and in parallel fashion, to form microfibrils.^{5,17} Collagen microfibrils are then organized into fibrils, the bundles of which constitute fibers that are embedded in an amorphous matrix of proteoglycans. Fiber aggregates make up fascicles, which are the major unit of the tendon, with each fascicle enveloped in its own endotenon. Fascicles of collagen fibers are arranged into primary to quaternary bundles and are contained within a peritenon, consisting of loose connective tissue that invaginates between the fiber bundles.

In addition to comprising the peritenon, some tendons are covered by a synovial sheath in areas of mechanical stress or in areas requiring tendon constraint. Those tendons not covered by a synovial sheath are surrounded by a paratenon, consisting of loose connective tissue. Blood supply to tendons is scant, as opposed to that of muscle or bone, and it comes from the myotendinous junction, the tendon sheath or paratenon, and the enthesis. Peritendinous bursae occur at sites of potential tendon friction or compression (eg, where a tendon changes

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Figure 1. Longitudinal sonographic image of a normal Achilles tendon (arrow) showing uniform thickness and an echogenic fibrillar appearance.

course, where a tendon may be compressed against a bone or another tendon).¹⁸

SONOGRAPHY

The normal tendon has an echogenic fibrillar pattern in both short and long axis because sound waves reflect back to the transducer from the collagen bundles, whose interfascicular interfaces act as specular reflectors (Figure 1). Caution should be exercised when scanning tendons, to make sure that the sonographic beam is perpendicular to the tendinous structure in whatever plane is being imaged; a nonorthogonal sound beam may make the tendon look artifactually hypoechoic, thereby mimicking tendinosis or tear, due to anisotropy. Anisotropy is the property of highly ordered structures, such as tendons, ligaments, and nerves, to vary in their reflective echogenicity depending on the angle of insonation of the interrogating sound beam. At an angle of insonation directly perpendicular to the tendon, the reflected sound energy is at its greatest as it is returned to the transducer, thus giving the tendon its typical echogenic appearance. At any other angle of incidence, however, the sound energy returning to the transducer is decreased, yielding diminished echogenicity and an artifactual appearance that can be misinterpreted as tendinosis (Figure 2).¹⁴

MAGNETIC RESONANCE IMAGING

On all conventional pulse sequences, normal tendons should be homogeneously low signal intensity (Figure 3). However, when a tendon is oriented approximately 55° to the main magnetic field, as it may when it changes course, there is an increase in signal intensity, which occurs on short echo sequences and is a form of anisotropic prolongation of relaxation time, referred to as the magic angle phenomenon.¹⁷ It is the highly ordered organization of predominantly collagen type 1 fibers that results in this structural anisotropy. Magic angle phenomenon is less apparent when the echo time is greater than 37 milliseconds⁴⁶; thus, signal hyperintensity attributed to magic angle is not discernable on T2-weighted images while signal hyperintensity owing to tendinosis persists (Figure 4).

The densely arranged and highly organized microstructure of tendons is thought to restrict the rotational motion of water, accounting for the uniformly low signal intensity on conventional

Figure 2. Sonographic anisotropy. A, when the transducer is perpendicular to the tendon, the sound waves (arrows) are maximally reflected back and the tendon has an echogenic appearance; B, when the transducer is oblique to the tendon, the sound waves (arrows) are not reflected back to the transducer and the tendon has a hypoechoic appearance. Tr, transducer.

Figure 3. Sagittal T1-weighted magnetic resonance image of the hindfoot showing a normal Achilles tendon (arrow) with uniform thickness and uniform low signal intensity.

MRI.¹⁷ In addition, the densely packed and ordered collagen bundles generate a static, local magnetic field that results in increased spin-spin interaction, which in turn leads to tendon T2 relaxation times on the order of microseconds. The tendon signal decays faster than it can be detected using conventional pulse sequences, which image on the order of milliseconds⁴⁶; thus, the tendon appears as low signal intensity. However, advances in gradient strength have enabled ultra-short echo time imaging (ie, in microseconds), and preliminary investigations have shown tendon ultrastructure not visible on conventional sequence.^{4,48}

TENDON INJURY

Tendon injury is a spectrum of abnormalities, ranging from degeneration to tears. *Tendinosis* refers to degeneration, which is caused by repetitive cyclical loading with resultant microtears of the tendon and which leads to crimping and disorganization of collagen fibers, tenocyte death, chondroid

Figure 4. Magnetic resonance anisotropy. A, axial T1-weighted image, with an echo time of 20 milliseconds through the hindfoot, showing high signal intensity in the posterior tibial tendon (arrow); B, axial T2-weighted image, with an echo time of 85 milliseconds at the same location, showing normal low signal intensity in the tendon (arrow).

metaplasia of tenocytes, increase in mucoid ground substance, and occasionally, angiofibroblastic proliferation due to vascular endothelial growth factor.^{32,34,51,53} The term *tendinitis* is a misnomer because there is no evidence of acute or chronic inflammatory infiltrate on histologic inspection.³⁴ Tendinosis may also be a function of senescence; for example, the aging supraspinatus tendon demonstrates decreased mucopolysaccharides, decreased cellularity, increased collagen cross-linking (which makes the tendon more brittle), and an increased amount of type 3 collagen (which is weaker than type 1 collagen, found in younger tendons).^{8,47}

Figure 5. Sagittal proton density magnetic resonance image of the hindfoot showing tendinosis of the Achilles tendon (arrow), manifest as fusiform thickening.

On MRI, the degenerated tendon may be thickened and may have signal heterogeneity, instead of the usual low signal intensity (Figures 5, 6, and 7). Sonographically, a degenerated tendon will be thickened and hypoechogenic, with loss of the normal echogenic fibrillar pattern (Figures 8 and 9). Power or color Doppler imaging may demonstrate hyperemia because of the neovascularity of angiofibroblastic proliferation (Figure 10).

Whereas *tendinosis* refers to the structural changes of degeneration seen histologically or with imaging,⁴⁴ *tendinopathy* is a clinical term describing tendon pain, swelling, and resultant decreased athletic capability^{45,56}; as such, these 2 processes can be mutually exclusive. In 83 patients with clinical tendinopathy of the Achilles, tendinotic changes were seen sonographically in only 35% of the cases, with the remaining symptomatic cases showing no abnormality; conversely, 18 asymptomatic tendons showed sonographic abnormalities.⁴⁵ Similarly, in a study of 45 patients with Achilles tendinopathy, 20 of the 57 clinically abnormal tendons were sonographically normal, whereas 9 of the 28 clinically normal tendons were sonographically abnormal.³⁶ Although some studies have shown that symptomatic patients with normal sonographic results have a significantly better clinical outcome than that of those with abnormal sonographic results,^{1,41} Khan et al found that

Figure 6. Sagittal proton density magnetic resonance image of the knee showing tendinosis of the proximal patellar tendon, or jumper's knee (arrow), with focal thickening and interstitial signal hyperintensity.

the severity of the US findings in the Achilles tendon at baseline was not associated with clinical outcome 1 year later.³⁶ Moreover, the gray scale sonographic appearances may resolve, persist, or enlarge, without any relation to symptoms.^{11,35}

Tendon subluxation may also be a cause of tendinosis, owing to altered tensile stress from altered biomechanics or repeated frictional forces. Tendon subluxation, whether dynamic or static, is usually due to failure of the tendon's soft tissue or osseous restraints. Sonography has the capability of dynamically assessing subluxation.^{10,42}

The presence of neovascularity in the degenerated tendon has been suggested as a cause of pain,^{12,13} but this finding may not be the entire cause of symptoms, given that 8 of 20 symptomatic foci in one study lacked vascularity on power Doppler imaging²¹ and 10 asymptomatic tendons in another study had gray scale and Doppler abnormalities.²² Moreover, Boesen et al have demonstrated increased vascularity in the Achilles tendon

Figure 7. Sagittal proton density magnetic resonance image of the knee showing tendinosis of the patellar tendon (arrow) with generalized thickening and signal heterogeneity.

Figure 8. Longitudinal extended-field-of-view sonographic image of Achilles tendinosis (arrows) showing fusiform thickening and hypoechogenicity.

after exercise, even in asymptomatic people.^{6,7} Nonetheless, several studies have reported therapeutic relief of pain after sclerotherapy of the intratendinous vascular regions.^{40,43,58}

Figure 9. Patellar tendinosis. A, longitudinal sonographic image of patellar tendinosis showing a thickened and heterogeneous hypoechoic tendon (arrows) extending from the patella to the tibial tubercle; B, longitudinal sonographic image of a normal patellar tendon (arrows) showing an echogenic fibrillar appearance with straight, parallel superficial and deep surfaces. P, patella; T, tibial tubercle.

Calcium hydroxyapatite may precipitate in a tendon because of tendon degeneration with local tissue hypoxia and decreased oxygen tension²⁸ or because of an endocrine disturbance,²⁶ with a general prevalence of approximately 3% to 22%.²⁶ On MRI, the calcium typically appears as an oval focus of low signal intensity, but small or amorphous foci may be difficult to identify against the background of the low signal intensity tendon (Figure 11). Sonographically, the calcific deposit has an echogenic surface and may or may not have posterior acoustic shadowing, depending on its concentration (Miller et al, unpublished data) (Figure 12). One study showed that hyperemia around the calcific deposit correlated with symptomatology, possibly representing inflammation during the resorptive phase of the process.⁹

A trace amount of fluid within the tendon sheath is normal, but circumferential presence of fluid or thickening of the tendon sheath indicates tenosynovitis. However, if the tendon sheath normally communicates with a joint, such as in the case of the long head of the biceps in the shoulder and the flexor hallucis longus tendon at the ankle, fluid may track into the sheath from an associated joint effusion.

Continued repetitive tensile stress and micro-tearing can lead to macro-tear of the tendon, which may be purely interstitial or may involve the tendon surface. Rosenberg et al⁴⁹ first described the imaging classification of tendon tear: grade 1 (low grade)

Figure 10. Longitudinal sonographic power Doppler image of proximal patellar tendonosis showing hyperemia (red color) in a thickened and hypoechoic proximal tendon. P, patella.

Figure 12. Longitudinal sonographic image of supraspinatus calcific tendonitis showing an echogenic surface of the calcium deposit (large arrow) within the tendon, as well as focal posterior acoustic shadowing (small arrow) partially obscuring the underlying humeral cortex.

Figure 11. Oblique coronal proton density magnetic resonance image of supraspinatus calcific tendonitis showing low signal intensity (arrow) within the tendon.

appears as tendon thickening; grade 2 (high grade) appears as thinning and attenuation of the tendon; grade 3 (rupture) manifests as tendinous discontinuity, with or without retraction.

Notwithstanding this generic model of tendon tear, some tendons exhibit additional patterns of tear. For example, the peroneal tendons may develop longitudinal split tears.^{3,15,16,42,52} The peroneus brevis tendon is more often involved than the longus tendon because of its anterior location in the fibular groove; in the setting of superior peroneal retinacular insufficiency, repetitive subluxation of the brevis tendon, as pressed against the malleolus by the longus tendon, leads to compressive and shear forces with the eventual longitudinal split tear of this

Figure 13. Sagittal T2-weighted magnetic resonance image of Achilles tendon rupture showing thickening and signal heterogeneity within the retracted tendon edges (white arrows), indicating underlying tendinosis, with high signal intensity edema/hemorrhage within the tendon gap (black arrow).

Figure 14. Longitudinal sonographic image of Achilles tendon rupture showing thickening and loss of the fibrillar architecture within the retracted tendon edges (white arrows), indicating underlying tendinosis, with echogenic hemorrhage within the tendon gap (black arrow).

tendon. The supraspinatus tendon may undergo delamination and retraction of its articular surface, partly owing to different shear forces and tensile forces as experienced by the bursal and articular surfaces.^{33,57}

Degeneration of the tendon is a risk factor for rupture. Gibbon et al found 3 or more microtears in athletes with partial-thickness Achilles tendon tears, compared to normal tendons or tendons without partial-thickness tear.²⁰ A histological study of normal, tendinotic, and ruptured Achilles tendons showed a significantly greater amount of degeneration in ruptured tendons compared to that of tendinotic or normal tendons.⁵³ Similarly, a longitudinal study of patients with achillodynia found 7 ruptured tendons in patients with sonographically abnormal tendons at the beginning of the study and none in patients with initially normal appearing tendons.⁴¹ Systemic diseases, such as diabetes, chronic renal failure, rheumatoid arthritis, and chronic steroid therapy, are also risk factors for tendon rupture.²⁴

Tendon ruptures are characterized on MRI and ultrasound as discontinuity of the tendon fibers, with or without retraction and resultant tendon gap (Figures 13 and 14). The gap can be filled with fluid, fat, or scar tissue.³⁹ The free edges of the ruptured tendon are typically thickened and heterogeneous in their echogenicity or signal intensity, as a result of underlying tendon degeneration. An advantage of MRI for evaluating suspected tendon rupture is the large field of view and global evaluation of any other anatomic abnormalities. An advantage of sonography is the ability to perform a dynamic examination; this can be helpful in cases of tendon tear in which the torn ends are apposed to one another, thus making clinical distinction between partial tear and rupture difficult.^{27,38}

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