Abstract

In the past decades the incidence of acute and chronic disorders of the Achilles tendon associated with sport-induced overuse has steadily increased. Besides acute complete or partial ruptures, achillodynia (Achilles tendon pain syndrome), which is often associated with tendon degeneration, represents the most challenging entity regarding clinical diagnostics and therapy. Therefore, the use of imaging techniques to differentiate tendon disorders and even characterize structure alterations is of growing interest. This review article discusses the potential of different imaging techniques with respect to the diagnosis of acute and chronic tendon disorders. In this context, the most commonly used imaging techniques are magnetic resonance imaging (MRI), B-mode ultrasound, and color-coded Doppler ultrasound (US). These modalities allow the detection of acute tendon ruptures and advanced chronic tendon disorders. However, the main disadvantages are still the low capabilities in the detection of early-stage degeneration and difficulties in the assessment of treatment responses during follow-up examinations. Furthermore, differentiation between chronic partial ruptures and degeneration remains challenging. The automatic contour detection and texture analysis may allow a more objective and quantitative interpretation, which might be helpful in the monitoring of tendon diseases during follow-up examinations. Other techniques to quantify tendon-specific MR properties, e.g. based on ultrashort echo time (UTE) sequences, seem to have great potential with respect to the precise detection of degenerative tendon disorders and their differentiation at a very early stage.

Key Points:

▶ For radiological imaging in the clinical routine, different clinical presentations of acute or chronic overuse injuries of the Achilles tendon must be considered.
▶ In addition to qualitative morphological imaging criteria, supplementary quantitative criteria (planimetry/volumetry) of the Achilles tendon seem to be significant with respect to the differentiation between symptomatic and asymptomatic Achilles tendons.
▶ Other techniques to quantify tendon-specific MR properties, e.g. based on ultrashort echo time (UTE) sequences, seem to have great potential with respect to the precise detection of degenerative tendon disorders and their differentiation at a very early stage.

Citation Format:


Zusammenfassung

Innerhalb der letzten Jahrzehnte hat die Inzidenz von akuten und chronischen Überlastungsschäden der Achillessehne stetig zugenommen. Die deutliche Zunahme in den letzten Dekaden lässt sich in erster Linie durch die erhöhte Freizeitaktivität in der Bevölkerung begründen. Neben den akuten Teil- oder Komplettrupturen der Achillessehne stellt die Achillodynie (Scherzersyndrom der Achillodynie), welche häufig mit einer Sehnendegeneration einhergeht, eine Herausforderung für Diagnostik und Therapie dar. In diesem Zusammenhang hat der Einsatz von bildgeben-
Übersicht

Introduction

The incidence of acute and chronic pathologies of the Achilles tendon has increased considerably in the last decades. These diseases are often caused by overuse during sports, in particular sports involving running or a ball, or increased occupational strain [1]. Both external influencing factors (e.g., training conditions and equipment) as well as internal influencing factors (e.g., anatomical conditions or systemic diseases) can trigger the development of acute or chronic pathologies [2]. The use of imaging methods for such cases has increased. In addition to the primarily qualitative imaging methods typically used in the clinical routine (ultrasound, MRI), methods for quantitatively analyzing and characterizing pathologies of the Achilles tendon are increasingly being developed.

In the clinical routine consideration of the different clinical presentations of acute and chronic pathologies of the Achilles tendon that need to be differentiated in relation to morphology and location and require corresponding diagnostic methods is relevant for the selection of the suitable imaging method.

Imaging methods for evaluating the Achilles tendon

Due to their good soft tissue contrast, magnetic resonance imaging (MRI) and ultrasound have become the standard for diagnosing acute and chronic overuse injuries of the Achilles tendon [3, 4]. Conventional radiography and computed tomography (CT) play only a secondary role, for example, in the evaluation of possible bone involvement in an avulsion fracture at the calcaneus or in regard to the imaging of tendon calcification or bone spurs [5, 6]. Conventional radiography is also still valuable for diagnosing Haglund’s exostosis [7].

Ultrasound

Due to its broad availability, good sensitivity for Achilles tendon pathologies, and comparatively low costs, ultrasound is very frequently used as the primary method for diagnosing acute and chronic overuse injuries of the Achilles tendon [8]. However, the disadvantages of this method include examiner dependence and limited reproducibility [9]. To improve the detail resolution, high-frequency probes with a frequency of 7.5 MHz or higher are used [10, 11]. B-mode ultrasound is regularly used for visualizing anatomical structures and power Doppler is used for evaluating the vascularization of tendons and paratendinous tissue.

The patient is positioned in a prone position on the examination table. The ankle joint is positioned so that it protrudes past the rear edge of the examination table and should be held in the neutral-zero position. Transverse and longitudinal scans of the entire tendon are acquired. The longitudinal scans should always be at a 90 degree angle with respect to the widest part of the Achilles tendon to prevent possible artificial echogenicities in healthy tendons from being incorrectly evaluated as pathological [12]. In the longitudinal scan, healthy tendon tissue has a relatively homogeneous echo pattern with parallel linear echoes that show the fibrillar structure (Fig. 1) [10]. The boundaries (paratenon) are parallel to one another and can also be delimited. The important diagnostic parameter, the so-called true tendon thickness, should also be measured in this scan orientation [13, 14]. The standard value for healthy tendons is approx. 4 – 5 mm. The standard measurement is performed using a longitudinal scan in the center of the Achilles tendon approx. 2 – 4 cm from the bony insertion at the calcaneus [9, 14]. The smallest neovascularizations in the Achilles tendon can be visualized with power Doppler or color duplex sonography [15]. Power Doppler is superior to color duplex sonography with respect to diagnostic sensitivity in this regard [16].

MRI

The field strengths typically used in the clinical routine for the diagnosis of pathologies of the Achilles tendon are 1.5 T and 3 T. However, due to the increasing availability of high-field MRI units, MR examinations of the ankle joint and the surrounding tendons are increasingly being performed at a field strength of 3 T in recent years. The main advantage of the higher field strength is the almost linear increase of the signal-to-noise ratio (SNR) in relation to the field strength. The greater SNR compared to at 1.5 T can be used to either improve the spatial resolution or to reduce the measurement time with fewer averaging operations. Moreover, the frequency-specific fat suppression in the region of the extremities is faster and more robust at 3 T. This is mainly due to the greater chemical shift between fat and water (approx. 220 Hz at 1.5 T and approx. 440 Hz at 3 T) [17]. When comparing examination protocols for 1.5 T and 3 T, it must be taken into consideration that the T1 relaxation times at 3 T are approximately 15 – 20% longer depending on the type of tissue while the T2 relaxation times at 3 T are only slightly shorter [18]. The main disadvantages of the higher field strength are increasing field inhomogeneity, more significant artifacts in the area of metal implants, and regional image shading caused by B1 field inhomogeneities [17].

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standard sequences at 1.5 T and 3 T are summarized in Table 1.

A healthy tendon has a low signal appearance due to its ordered structure and the limited intratendinous mobility of free water in T1, T2 and proton density (PD) weighting. It is uniformly narrow and extends for approx. 15 cm from the calcaneal insertion to the myotendinous junction. Fluid-sensitive sequences show the retrocalcaneal bursa in the form of a small hyperintense band of fluid on the ventral side above the calcaneal tuberosity. It is bordered apically and ventrally by the fat-isointense Kager’s fat pad (Fig. 1).

Solitary focal intratendinous changes (usually point-shaped or longitudinally oriented) do not have any pathological value since they correspond to small connective tissue septa or small intratendinous vessels [19].

### Table 1

<table>
<thead>
<tr>
<th>sequence</th>
<th>TE (ms)</th>
<th>TR (ms)</th>
<th>field strength (T)</th>
<th>FOV (mm)</th>
<th>resolution (mm³)</th>
<th>scan duration (min)</th>
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<tbody>
<tr>
<td><strong>standard protocol at 1.5 T</strong></td>
<td></td>
<td></td>
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<tr>
<td>T1 TSE sagittal</td>
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<td>220</td>
<td>0.5 × 0.5 × 3</td>
<td>3:21</td>
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<tr>
<td>T2 TSE axial</td>
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<td>6640</td>
<td>1.5</td>
<td>160</td>
<td>0.8 × 0.8 × 3</td>
<td>3:58</td>
</tr>
<tr>
<td>PD TSE fs sagittal</td>
<td>29</td>
<td>4000</td>
<td>1.5</td>
<td>220</td>
<td>0.7 × 0.7 × 3</td>
<td>5:30</td>
</tr>
<tr>
<td>TIRM sagittal</td>
<td>32</td>
<td>4000</td>
<td>1.5</td>
<td>220</td>
<td>0.7 × 0.7 × 3</td>
<td>5:26</td>
</tr>
<tr>
<td><strong>standard protocol at 3 T</strong></td>
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<td></td>
<td></td>
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<tr>
<td>T1 TSE sagittal</td>
<td>12</td>
<td>600</td>
<td>3</td>
<td>220</td>
<td>0.3 × 0.3 × 3</td>
<td>3:20</td>
</tr>
<tr>
<td>T2 TSE axial</td>
<td>45</td>
<td>6000</td>
<td>3</td>
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<td>PD TSE fs sagittal</td>
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<td>3</td>
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<td>3800</td>
<td>3</td>
<td>220</td>
<td>0.5 × 0.5 × 3</td>
<td>4:01</td>
</tr>
</tbody>
</table>

TE = echo time; TR = repetition time; FOV = field of view; TSE = turbo spin echo; PD = proton density; TIRM = turbo inversion magnitude; fs = fat saturation.
Pathologies of the Achilles tendon

The most important intratendinous and peritendinous changes visualized by imaging used in the clinical setting will first be discussed and then their significance in the general clinical and histological context will be explained in the following.

Intratendinous changes

Tendinosis

Tendinosis refers to a pathological change of the tendon due to degeneration. With approx. 60% of cases, it is the most common form of tendinopathy of the Achilles tendon [1]. The often used term tendinitis is misleading since it suggests inflammation for which there is not histopathological evidence [20]. According to the current theory, both repetitive overuse as well as defective reparative processes on a cellular level can result in destruction of the fiber structure. Repetitive microtraumas (so-called tendinosis cycling) cause increasing fiber fatigue resulting in tendinosis [21]. In principle, there are four types of degeneration but they overlap and the individual types cannot always be precisely differentiated. Therefore, hypoxic, mucoid, lipomatous, and calcifying ossifying degeneration is described in the literature. While hypoxic and mucoid degeneration are often the result of chronic overuse and have the highest total prevalence, lipomatous degeneration is primarily the product of aging [22]. However, calcifications within the Achilles tendon as the result of an ongoing degeneration process are rather rare. According to histopathological studies, the different types of Achilles tendon degeneration are present in 90% of symptomatic tendons and in up to 30% of asymptomatic tendons [1, 20, 22].

In MRI as well as ultrasound, hypoxic degeneration is visualized as a spindle-shaped thickening of the Achilles tendon [10, 22, 23]. In the further course, the fibrillar echo pattern is lost and there is generalized hypoechogenicity of the tendon [10]. MRI sometimes shows slight signal enhancement in the T2 weighting [22, 24] (Fig. 2a).

In MRI, mucoid degeneration is visualized with focal interstitial signal alterations and interruptions (hyperintensities in the T2 weighting and at times milder hyperintensity in the T1 weighting) [22]. Sonography frequently shows focal hypoechogenicities and microtears [10]. The Achilles tendon...
don thickness in the ventrodorsal diameter shows only a moderate increase to approx. 6 – 7 mm with the standard measurement being performed in the sagittal plane 2 – 4 cm from the calcaneal insertion under consideration of the abovementioned “true tendon thickness” concept [13, 14] (Fig. 2). Paratendinous neovascularizations with diffuse ingrowing of capillaries and arterioles are often seen histopathologically in the case of degenerated tendons [1]. In the case of achillodynia (pain syndrome of the Achilles tendon), Doppler sonography typically shows intratendinous neovascularizations but these can also be visualized in asymptomatic tendons [15] (Fig. 2). The percentage of asymptomatic tendons with intratendinous neovascularizations is up to 35 % according to the literature [25]. The degree of neovascularization can be classified according to the Öhberg score (0 through 4+) with 0 indicating the absence of neovascularization, 1+ the presence of neovascularization in the anterior tendon segment and 2+ through 4+ indicating varying degrees of irregular neovascularization [26]. The modified Öhberg score is as follows: 0 (no vessels), 1+ (1 vessel in the anterior segment, 2+ (2 vessels in the entire tendon), 3+ (3 vessels), and 4+ (> 3 vessels) [27]. The existence of a correlation between the occurrence of pain and the degree of neovascularizations is a topic of debate in the literature due to the relatively high prevalence of neovascularizations in asymptomatic tendons [15, 28]. Since mucoid and hypoxic degeneration can coexist, a qualitative differentiation is often difficult [29]. Lipomatous degeneration can be diffuse or focal. Diffuse structure changes or hypoechogenic zones are often seen in ultrasound as a sign of local xanthomas [30]. MRI shows point-shaped hyperintensities in the T2 and T1 weighting. The tendon can also appear thickened in the case of lipomatous atrophy. Differentiation from other types of tendinopathy can be difficult [31]. Calcifying tendinosis often includes focal calcifications of the tendon which appear extremely hyperechogenic with a lack of dorsal through-transmission in sonography. In MRI, calcifications may exhibit a signal behavior corresponding to the bone marrow of the tibia in the T1 weighting [6].

**Partial rupture**

On the whole, the diagnosis of partial ruptures via ultrasound is limited with the differentiation from focal degenerative changes being particularly difficult [32]. Ultrasound shows partial ruptures of the Achilles tendon as wavy irregularities of the fibrillar echo pattern [33]. Focal hypoechogenic areas and thickening of the affected tendon segment are often seen [32]. Alfredson et al. reported that the superficial dorsal tendon boundary also has an interruption and accompanying hyperperfusion can be visualized with power Doppler [33]. However, sonographic diagnosis of a partial rupture seems to be limited in particular in the myotendinous junction and in the proximal third of the Achilles tendon. Supplementary MRI is usually necessary in this case [34].

In the case of a partial rupture, MRI shows apposition of the ruptured fibers and local signal enhancement in particular in T1 and T2-weighted imaging [23]. Moreover, partial ruptures exhibit significant focal thickening of the Achilles tendon to 10 – 18 mm, which is often greater than the thickening associated with degenerative tendinosis [35]. However, the differentiation between a small partial rupture and focal degenerative changes remains a challenge both with sonography and MRI [36] (Fig. 2).

**Complete rupture**

In the case of a complete rupture, a differentiation can be made between an acute complete rupture and a chronic rupture with scar formation [24]. Acute as well as chronic rupturing of the Achilles tendons often shows concurrent degenerative changes [35]. Ruptures usually occur in the
avascular area 2 – 6 cm proximal to the calcaneal tendon insertion. Moreover, atypical insertional or proximal Achilles tendon ruptures can occur in rare cases. Changes near the insertion often occur as part of an advanced insertional tendinopathy, while proximal ruptures usually correspond to a rupture of the muscle-tendon complex [6] (Fig. 3). Complete rupture of the Achilles tendon is visualized sonographically as dehiscence of the fibers with a frequently inhomogeneous hypoechoic hematoma between the tendon stumps [32, 37]. In the chronic stage, fiber discontinuity occurs with structure resolution which exhibits variable echogenicity depending on the chronicity of the process [38]. A hyperechoic signal indicates scar formation (fibrosis) [39]. The interruption of the tendon structure and variable dislocation of the tendon stumps can be effectively visualized with MRI [24]. A concomitant hematoma with edema formation and very high signal intensity in T2 weighting is often seen [6, 23]. Chronic ruptures are typically low signal in T1 weighting, and discontinuity of the tendon with horizontal hyperintensity is often seen in T2 weighting [24, 38].

Insertional tendinopathy

Insertional tendinopathy which accounts for only approx. 20% of all tendinopathies of the Achilles tendon is rarer than tendinosis [1]. Insertional tendinopathy is visualized in ultrasound as tendinous thickening in the region of the tendon insertion. Hook-shaped calcifications with a lack of dorsal through-transmission or pathological bone spur formation (dorsal calcaneal spur) can often be delineated. T2-weighted MRI shows fine longitudinal hyperintense lines corresponding to focal microtears [24] (Fig. 4). Moreover, concomitant changes, such as bone marrow edema of the calcaneus (8%) or a thickened retrocalcaneal bursa (19%), can be imaged [40, 41]. MRI has proven to be very useful for insertional tendinopathy treatment planning and monitoring in a number of studies [42, 43].

Peritendinous changes

Paratenonitis

Paratenonitis and peritendinitis are often used synonymously and refer to the inflammatory change of the paratenon which surrounds the Achilles tendon and separates it from the crural fascia [1]. The paratenon differs from the peritendineum of other tendons in that it is not a real tendon sheath. It does not contain any synovial tissue but does supply blood to the tendon, which is the basis for inflammatory changes [6]. Therefore, the term paratenonitis better describes the inflammatory reaction of the tissue around the actual tendon.

Due to the increased fluid content in the inflamed tissue, T2-weighted MRI shows paratenonitis as a bright hyperintense band that typically incompletely surrounds the Achilles tendon [22, 24]. In contrast, ultrasound shows paratenonitis as a dark hypoechoic peritendinous halo due to the reduced echogenicity. Once the condition has become chronic, irregularities of the boundary zone of the paratenon can additionally occur and result in significantly increasing hypervascularization in power Doppler [44, 45] (Fig. 2).

Retrocalcaneal bursitis

The retrocalcaneal bursa is located between the calcaneal tuberosity and the Achilles tendon insertion and is surrounded by Kager’s fat pad. It is directly related to the paratenon and is often also affected in the case of degenerative tendon changes [46]. In these cases, the bursa often exhibits histological degeneration of the bursal wall – at times with calcification or hypertrophy – and intraluminal fluid collection. In sonography, the fluid-filled bursa appears homogeneously hypoechoic, while it appears hyperintense in T2-weighted MRI [45]. However, focal hyperintensity of the bursa is not to be primarily evaluated as pathological. The bursa in asymptomatic subjects reaches an average size of 1 × 6 × 3 mm while the symptomatic bursa is usually significantly larger at approx. 4 × 9 × 4 mm [46]. In addition to the retrocalcaneal bursa, the subcutaneous calcaneal bursa, which is directly subcutaneous and posterolateral to the bony insertion, is frequently also affected. In a pathological state, it appears as an elongated hypoechoic structure in sonography and as a hyperintense structure in T2-weighted MRI [45] (Fig. 4).

Haglund’s exostosis

Haglund’s deformity is caused by a pathological impingement of the retrocalcaneal bursa and the insertional Achilles tendon. The most important influencing factors in this context are an inherent prominence of the posterolateral portion of the calcaneus and the wearing of high-heeled shoes (“pump bump”). In the case of repetitive injuries to the insertion region, hypertrophy of the calcaneal tuberosity, frequently with concomitant bursitis and tendinopathy, occurs. Signs of bursitis and insertional tendinosis and a prominent, cranially extended posterior tuberosity are typically visualized [6, 22, 47] (Fig. 4).

Clinical radiological tendinopathy classifications

For radiological imaging in the clinical routine, different clinical presentations of acute or chronic overuse injuries of the Achilles tendon must be considered. Schweitzer und Karasick differentiate between 7 clinically relevant main groups including a total of 11 subgroups with different imaging characteristics [22]. The individual groups differ with respect to their location (insertion, peritendinous and intratendinous changes), morphology (rupture, calcification, hypoxic and mucoid degeneration), and chronicity (acute versus chronic). In a recently published study, Weber et al. reduced these to 4 clinically relevant groups based on morphological imaging criteria (1) peritendinous changes (paratenonitis), (2) increase in the size of the Achilles tendon (hypoxic degeneration) with focal intratendinous changes, (3) visible morphological changes in the case of mucoid degeneration (e.g. signal amplification in T2 weighting), and (4) ruptures. Moreover, the differentiation between insertional and non-insertional tendinopathies (mid-portion tendinosis) seems to be important [1, 22, 24]. Viewed with other clinical and morphological imaging criteria, the following points therefore seem relevant:

- **Location:** Peritendinous, intratendinous insertional, or intratendinous non-insertional
Morphology: Increase in caliber (hypoxic degeneration) vs. generalized structural changes (mucoid degeneration)

Chronicity: Acute versus chronic tendinopathy

In comparison to the above-described classification according to Schweitzer and Karasick, other classification systems (e.g. according to Weinstabl et al. or Pomeranz et al.) are based on the extent of the visible morphological changes [43, 48, 49]. To summarize, we find the following classification useful for the clinical routine:

1. Solitary paratenonitis: Strict peritendinous changes without intratendinous involvement.
2. Tendinosis without or with only mild visible morphological intratendinous structural changes: Thickening of the tendon with only focal signal alterations of the tendon.

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Fig. 4 a shows an insertional tendinopathy with retrocalcaneal bursitis in a 55-year-old patient. The Achilles tendon shows an insertional thickening with focal hypoechoic areas (white arrow). The black arrow marks the concomitant hyperemia of the retrocalcaneal bursa. b shows a PD weighted sagittal image of an insertional tendinopathy in a 75 years old patient, where focal hyperintensities are present (white arrow). c Sagittal PD-weighted image with fat saturation in a 26-year-old patient with acute retrocalcaneal bursitis (black arrow). The bursa is enlarged (18 × 9 mm) and shows a hyperintense signal (fluid) in the PD-weighted image. Accompanying bone bruise within the proximal part of the calcaneal tuberosity is also present (white arrow). A plain radiograph d of a symptomatic Haglund’s deformity (black arrow) in a 50-year-old female patient with accompanying swelling of the adjacent subcutaneous tissue (white arrow).
don corresponding to hypoxic degeneration, possibly with concomitant paratenonitis.

3. Tendinosis with visible morphological intratendinous structural changes: Resolution of the fibrillar echo pattern, signal enhancement in T2 weighting corresponding to mucoid degeneration ("silent tendonitis") according to Karasek et al.

4. Ruptures: Complete and partial rupture.

5. Insertional tendinopathy: This is often considered an independent entity and is often accompanied by focal calcifications [1].

In contrast to chronic changes, paratenonitis, bone marrow edema at the calcaneal insertion, edema of Kager's fat pad, and concomitant retrocalcaneal bursitis are often present in the case of an acute tendinopathy [22, 24]. The use of power Doppler ultrasonography which shows increased vascularity of the peritendinous structures in the case of an acute tendinopathy seems useful in this regard.

Despite the above criteria and classifications of Achilles tendon changes, definitive diagnosis of pathologies on a purely morphological basis remains difficult. This is due to the fact that types of hypoxic and mucoid degeneration can be histologically present in both asymptomatic and symptomatic tendons and the line between borderline normal changes and borderline pathological changes is not clearly defined [29]. Therefore, Weber et al. recently used MRI images to evaluate various quantitative parameters in addition to visible morphological characteristics for the diagnosis of pathologies in the region of the Achilles tendon. Using 3 parameters (anteroposterior diameter of Achilles tendon 3 cm above the calcaneus, cranio-caudal size of the retrocalcaneal bursa, elliptical tendon cross-section from maximum anteroposterior and mediolateral diameter), it was possible to differentiate between healthy (i.e., asymptomatic) and pathological (i.e., symptomatic) changes in the Achilles tendon with a specificity of 91% and a sensitivity of 97% [24]. However, the degree of intratendinous and peritendinous changes was not applied to the binary logistic regression analysis. Other studies using comparable parameters for differentiating between symptomatic and asymptomatic tendons (tendon volume in MRI [50, 51] and average tendon thickness 2-3 cm from the calcaneus in the longitudinal ultrasound image [14]) yielded similar results. However, the relevance of the parameters in the evaluation of clinical course, especially the persistence of visible morphological changes after acute symptoms have subsided, remains unclear [52]. Quantitative parameters for characterizing the intratendinous structure could respond earlier to changes than only the determination of tendon diameter or cross-sectional area and therefore prove to be more sensitive for follow-up during treatment or for the diagnosis of the early stages of degeneration. With respect to these remaining diagnostic shortcomings, a number of parameters have been evaluated in recent years with varying degrees of success. The most important methods in current research regarding this topic are described briefly in the following and evaluated with respect to their clinical relevance.

New possibilities for imaging the Achilles tendon

Contrast-enhanced MRI

In addition to the routine use of native T1, T2, or PD-weighted sequences in MRI, intravenously administered contrast agents can also be used in special cases. However, the results of different studies regarding this topic are controversial. While Movin et al. postulated an improved detection of symptomatic tendons compared to asymptomatic tendons, Gardin et al. did not find a benefit with respect to native imaging [36, 53]. They achieved the best differentiation regarding the intratendinous signal behavior in a native T1-weighted gradient echo sequence. Both studies are comparable only on a limited basis with respect to the study protocol and are only of limited significance due to the small number of patients (20 and 25 patients, respectively). The asymptomatic tendons of the patients served as the control in each case [36, 53]. Shalabi et al. examined the contrast agent dynamics in 15 patients with symptomatic tendinopathy of the Achilles tendon. The healthy tendons on the opposite side served as the control group (n = 10). The symptomatic tendons exhibited early contrast uptake with-
out washout in the late phases while the control group only showed uptake in the late phase [54]. A disadvantage of intravenous contrast-enhanced MRI is the potential risk for nephrogenic systemic fibrosis (NSF) in the case of significantly limited kidney function and the potential allergenic effect, thus making large studies with healthy subjects problematic. According to the current data, there is no clear clinically relevant benefit of the use of intravenous contrast agent. Therefore, routine application should be viewed with reservation. Intravenous application of contrast agent should only be used for evaluating the Achilles tendon when indicated by a concomitant disease (e.g. rheumatoid arthritis, Bechterew's disease).

**Automated contour recognition and texture analysis**

An important clinical parameter in the diagnosis of tendinosis, i.e., the maximum thickness of the Achilles tendon, is typically determined via a manual point-wise measurement both in ultrasound and MRI images [13, 55]. However, in particular in regard to the measurement of small tendon changes, point-wise manual measurement is only usable on a conditional basis due to its limited reproducibility (variation coefficient approx. 5–7 %) [13, 14, 55]. Automated contour recognition methods improve reproducibility by up to 50 % in both B-mode ultrasound and MRI [14, 50, 51]. Moreover, the maximum thickness as well as the tendon volume can be reliably calculated in an automated manner via so-called seed growing or snake algorithms in MRI images (variation coefficient approx. 1–5 %) [50, 51]. The automated methods could have particular advantages with respect to the detection of the smallest tendon changes, e.g. with respect to minor training or therapy effects. A disadvantage is that some methods are still dependent on image quality and require manual corrections by an experienced examiner in individual cases [14, 50, 51]. In addition to surface area and volume calculation, the automated methods also allow more comprehensive computed-aided analysis of the internal tendon structure. A possible parameter with good reproducibility is the average signal intensity of the Achilles tendon in MRI images [50]. Another study showed a good correlation between intratendinous signal enhancement and the pain symptoms experienced by the patients with the pain symptoms correlating better with the signal intensity than the tendon volume [53].

Bashford et al. developed an ultrasound-based method for automated differentiation of degenerative and healthy Achilles tendons. Quadratic regions of interest (ROIs) were split into their spatial frequency spectrums and 8 different spatial frequency parameters were extracted. Up to 84.9 % of patients with a tendinopathy were able to be correctly classified in discriminant analysis [56]. In contrast, Van Schie et al. used an automated ultrasound tissue characterization (UTC) to examine four different, predefined echo patterns based on stability and distribution (1: very stable, 2: average stability, 3: highly variable, 4: low intensity with variable distribution). It was shown that symptomatic Achilles tendons with tendinosis have a significantly higher percentage of variable unstable echo patterns compared to a healthy control collective [57].

Because of the low number of examined patients, automated texture analysis must still be considered in terms of experimental feasibility studies and has not become established in routine imaging due to the questionable therapeutic consequence. This does not apply to automated contour recognition which is based on already clinically established measurement methods (Achilles tendon thickness and volume) and significantly reduces the examiner-dependent variability so that it could become a valuable part of the clinical routine particularly for follow-up examinations.

**Elasticity analysis**

Real-time sonoelastography of the Achilles tendon has been a topic of research for a number of years [58, 59]. The different degrees of tissue hardness and elasticity are visualized using a color scale in the corresponding B-mode image [58]. De Zordo et al. showed that, in contrast to healthy tendons which have a very hard structure (93 % hard), tendon degeneration is accompanied by increasing softening of the structure (57 % soft) [60]. In opposition to the study by Zordo, Sconfienza et al. were able to show that symptomatic Achilles tendons of athletes have a higher degree of tendon structure hardness but the data were also only evaluated qualitatively in this study [61]. Drakonaki et al. attempted to quantify the tendon structure elasticity and introduced the so-called strain index which is the ratio between the tendon tissue hardness and the hardness of the surrounding fat tissue [59]. However, with a variation coefficient of approx. 30 % for different examiners, the reproducibility is too low for clinical diagnostics. Velocity-encoded phase-contrast MRI (VE-PC-MRI) in combination with an MR-compatible interferometer is another option for the in vivo quantification of force-dependent length changes and stiffness properties of the Achilles tendon. In this way Shin et al. were able to determine the force-length curve of the Achilles tendon in the case of isometric contractions [62]. For the reproducibility of the transition point (change of the tendon length in mm at 40 N), there was good reproducibility with a variation coefficient of approx. 4 % (intraday variability). However, the variation coefficient was significantly higher at approx. 17 % (intraday variability) for the stiffness measurement [62]. Since there is currently no data regarding pathologically altered tendons with respect to VE-PC-MRI, a final conclusion about clinical applicability cannot be made.

Due to the very small number of test subjects in the studies regarding measurement with both sonoelasticity and VE-PC-MRI, the applicability of the data is still limited. Clinically relevant use is not in sight at this time.

**“Magic angle” and ultrashort echo time (UTE) imaging**

In conventional MR sequences, the healthy Achilles tendon appears largely homogeneously signal-free. This is due to the extremely short T2 relaxation time of approx. 1 ms which causes almost complete dephasing of the transverse magnetization prior to the actual data acquisition [63]. The short T2 times are primarily based on the dipole-dipole interactions between the highly organized collagen fibers and the interstitial water molecules embedded between them. The strength of the dipole-dipole interaction depends on the orientation (angle θ) with respect to the B0 field and is defined by the formula (3 * cos2θ – 1). At an angle of 54.74°, the so-called magic angle, the strength of the dipole-dipole interaction is minimal, resulting in an exten-
tion of the T2 relaxation by a factor of approximately 100. As a result, the tendon tissue with a fiber orientation of approx. 55° with respect to the B0 field is visualized as signal intense [63, 64]. In a number of clinical studies, it was shown that degenerative and acute changes of the Achilles tendon can be better delineated with non-contrast-enhanced MR imaging with the help of the “magic angle” effect [65, 66]. Moreover, Oatridge et al. and Marshall et al. showed that a pathological contrast agent uptake in symptomatic tendons that was not able to be visualized with parallel orientation to the B0 field can be visualized with the help of the “magic angle” effect [66, 67]. However, in the case of minimal random samples, the applicability of the above studies is limited. The utilization of the “magic angle” effect is a simple means of intratendinous signal enhancement but the positioning of the patient’s lower leg at an angle of approx. 55° with respect to the B0 field when using a conventional tube-shaped MR scanner and conventional extremity coils is a challenge.

In addition to “magic angle” imaging, the introduction of so-called ultrashort echo time (UTE) sequences also made it possible to visualize quickly relaxing tissues such as ligaments, tendons, or cortical bones, with positive contrast. In contrast to conventional MR sequences with echo times of approx. TE > 1 ms, echo times of approx. TE = 0.05 ms can be achieved with UTE sequences. To achieve such a reduction of the minimum echo time, different methods, such as shortening of the HF pulse time or the use of unconventional read-out samplings, are combined [68]. On the one hand, UTE imaging with its ultrashort echo times provides an amplified intratendinous signal and improved visualization of pathologies both in the native imaging method and in contrast-enhanced sequences [69]. On the other hand, UTE sequences also allow quantitative evaluation of different MR-specific tissue characteristics. Essentially, known methods have been applied to UTE imaging so that in particular T1, T1rho, T2*, and magnetization transfer (MT) effects can be evaluated [70–75].

Fig. 6 shows sagittal 3D UTE images with (off-resonance frequency 1 kHz) and without off-resonance saturation pulse (M0) and the respective subtraction image. Pixel-wise calculated MTR parameter maps are also shown. 

a shows the images of a healthy Achilles tendon of a 31-year-old volunteer. In contrast, b shows images of a 44-year-old patient with mid-portion tendinosis and focally decreased MTR values.
In a number of studies, the effective transverse relaxation time ($T2^*$) of healthy and degenerative tendons was examined. Differences in the $T2^*$ relaxation time were seen both in healthy and degenerated tendons depending on the tendon region (e.g. insertion, muscle-tendon complex, mid-portion) [73, 74]. In studies assuming a monoexponential signal decay, the $T2^*$ time at 3 T in the mid-portion of a healthy Achilles tendon in vivo was approx. 1.5 ms [74, 76]. However, more recent studies regarding the $T2^*$ effect assume a biexponential signal decay from which a long and short $T2^*$ component can be derived [70, 73]. In healthy tendons, the fractional portion of the short $T2^*$ component at 3 T (0.5 – 1.3 ms depending on the region and study) is approximately 50 – 80 % and that of the long $T2^*$ component (7.9 – 31.8 ms) is 20 – 50 % [70, 73]. The longitudinal relaxation time of the Achilles tendon was also determined in vivo via UTE sequences and is approx. 700 ms [75]. So-called T1rho effects were evaluated as a further parameter by Du et al. using spinlock pulses [71, 77]. However, further studies regarding T1, $T2^*$, and T1rho effects at 3 T for degenerative Achilles tendons in vivo are currently not available. Therefore, their clinical significance is uncertain.

Another well known effect is based on the so-called magnetization transfer (MT) which occurs in tissues with a high percentage of macromolecules. This can be determined in tissues with very short transverse relaxation times with the help of UTE sequences. Using select and varied parameters, both the amount of free and bound water and the exchange constants can be quantified and used as absolute parameters. A simpler possibility that is much more suitable for the clinical routine is the pixel-wise calculation of a so-called MT quotient from the images with and without an MT preparation pulse [72, 74]. Tendons with symptomatic tendinosis have significantly lower MT quotients than healthy or asymptomatic tendons [72] (Fig. 6). Moreover, it is interesting that tendons with asymptomatic tendinosis and pure paratenonitis also have slightly reduced MT ratios indicating a concomitant tendon injury [72].

Possibilities of imaging at 7 T

In the case of the MR sequences used in the clinical routine, significantly better SNR and contrast-to-noise ratio (CNR) were achieved at 7 T compared to 3 T due to the improved resolution in the ultrahigh field at 7 T [78] (Fig. 7). Juras

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**Fig. 7** shows sagittal MR images of an Achilles insertional tendinopathy in a 39-year-old patient at 3 T a and 7 T b. Images with different echo times and a corresponding subtraction image are presented (courtesy of Prof. Trattnig and co-workers, Vienna, Austria).
et al. used a 3 D UTE sequence to examine the biexponential T2* signal decay in different portions of the Achilles tendon (insertion, mid-portion, muscle-tendon complex) in healthy and degenerated tendons at 3 T and 7 T. Significant differences in the T2* signal decay were seen depending on the examined region or with respect to healthy and degenerated tendons [73]. In addition to the 1H-MRI used in the clinical routine, other atomic nuclei such as 23Na can be used with restrictions for imaging in scientific studies. There was a good correlation to the water and proteoglycan content of the examined ex vivo preparations for the SNR in the histological comparison [79]. Moreover, initial in vivo

Table 2  Overview of typical imaging features of common intra- and peritendinous pathologies associated with overuse.

<table>
<thead>
<tr>
<th>diagnosis pathology</th>
<th>B-mode ultrasound</th>
<th>Power Doppler/color Doppler</th>
<th>MRI</th>
<th>CE MRI</th>
<th>UTE</th>
<th>elasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>intratendinous changes</td>
<td>thickening of the tendon (6 – 7 mm) focal hypoechochogenicity</td>
<td>neovascularization (initially in the anterior portion)</td>
<td>thickening with increase in volume alteration with focal hyperintensities in T2w</td>
<td>increased contrast enhancement; significant increase in contrast agent dynamics</td>
<td>focal extension of short T2* components significantly reduced MT ratio (1 – 5 kHz)</td>
<td>controversial: increasing vs. decreasing hardness</td>
</tr>
<tr>
<td>partial rupture</td>
<td>thickening of the tendon (10 – 18 mm) interrupted fibrillar echo texture</td>
<td>concomitant focal hypoperfusion</td>
<td>apposition of the fibers, increased intensity in T1w, significantly increased intensity in T2w</td>
<td>n.a.</td>
<td>n.a.</td>
<td>n.a.</td>
</tr>
<tr>
<td>chronic rupture</td>
<td>scar formation with interruption of the echo texture, focal hypoechochogenicity as well as hyperechochogenicity</td>
<td>neovascularization</td>
<td>horizontal focal signal enhancement in T2w, No increased signal intensity in T1w</td>
<td>n.a.</td>
<td>n.a.</td>
<td>n.a.</td>
</tr>
<tr>
<td>acute rupture</td>
<td>complete discontinuity of the fibers, intratendinous hematoma (hypoechoic to inhomogeneously echoic)</td>
<td>concomitant focal hypoperfusion</td>
<td>apposition and dehiscence of the fibers over the entire tendon width, increased intensity in T1w, significantly increased intensity in T2w</td>
<td>n.a.</td>
<td>n.a.</td>
<td>n.a.</td>
</tr>
<tr>
<td>insertional tendinopathy</td>
<td>insertional thickening with irregular echo pattern, focal calcifications</td>
<td>neovascularization</td>
<td>signal alterations in T2w, insertional thickening</td>
<td>n.a.</td>
<td>n.a.</td>
<td>n.a.</td>
</tr>
<tr>
<td>peritendinous changes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>paratenonitis</td>
<td>partial ring-shaped peritendinous hypoechochogenicity</td>
<td>increased vascularity</td>
<td>partial ring-shaped hyperintensity in T2w and TIRM</td>
<td>n.a.</td>
<td>moderately reduced MT ratio (1 – 5 kHz)</td>
<td>n.a.</td>
</tr>
<tr>
<td>retrocalcaneal bursitis</td>
<td>focal hypoechochogenicity cranial to retrocalcaneal tuberosity</td>
<td>concomitant focal hypertensity in T2w and TIRM</td>
<td>significant focal hypertensity in T1w</td>
<td>n.a.</td>
<td>n.a.</td>
<td>n.a.</td>
</tr>
</tbody>
</table>

MRI = magnetic resonance imaging; CE = contrast-enhanced; UTE = ultra short echo time; T2w = T2 weighting; TIRM = turbo inversion magnitude; n.a. = not available; MT = magnetization transfer.

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results show a good correlation with the proteoglycan content of healthy and degenerated Achilles tendons and should be further evaluated in larger clinical studies [80] (Fig. 8). Due to the highly limited clinical availability of MRI units with a basic field strength of 7 T and the not yet fully developed ultrahigh field examination technology, use in the clinical routine is currently not foreseeable [78].

**Conclusion**

For the clinical routine, an evaluation of the exact location of visible morphological changes seems to be relevant for the differentiation between peritendinous changes (paratenonitis) and intratendinous changes (tendinosis and insertion tendinopathy) (Table 2). For further evaluation, sonography as well as MRI can be used to differentiate between different types of degeneration, mainly hypoxic degeneration (with thickening of the tendon and minor focal structure changes) and mucoid degeneration (with changed echo texture and pathological MR signal behavior) but the line between the two is not clearly defined. Concomitant peritendinous changes such as paratenonitis or retrocalcaneal bursitis as well as bone marrow edema of the calcaneus make it possible to make a qualitative statement about acuteness. These peritendinous changes can also be reliably diagnosed via MRI or via ultrasound plus power Doppler except for in the case of bone marrow edema. Acute and chronic complete ruptures can also be effectively and reliably diagnosed with both methods. However, the diagnosis of partial ruptures which can sometimes be missed in morphological imaging diagnostics depending on location (proximal in particular in this case) and degree is currently still problematic. MRI seems to be superior to ultrasound in this regard.

In addition to qualitative morphological imaging criteria, supplementary quantitative criteria (Achilles tendon thickness 2–3 cm above the calcaneus) or the planimetry/volumetry of the Achilles tendon seem to be significant with respect to the differentiation between symptomatic and asymptomatic Achilles tendons. In addition, automation of the corresponding measurement methods allows simple and reliable examiner-independent evaluation also for follow-up. However, it is still unclear whether these measurement variables are suitable for the evaluation of therapy effectiveness or the diagnosis of the onset of degenerative changes. This diagnostic gap could be filled by newer, quantitatively determinable parameters of the intratendinous structure. However, larger clinical studies with corresponding follow-up are needed to be able to come to a clear conclusion about clinical relevance in this case.

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