

Tendons and Tendon Sheaths of the Hand – An Update on MRI

Sehnen und Sehnenscheiden an der Hand – Befunde in der MRT

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ABSTRACT

Background The tendons of the hand run in close proximity to each other and within retinacular tunnels adjacent to articular joints, while forming intersections in characteristic locations. The enclosing tendon sheaths are often sites of systemic or infectious inflammation.

Method This review article outlines the different entities of tendon and tendon sheath pathology and their manifestation in the hands. Diagnostic findings in tendon and tendon sheath disorders are illustrated using MRI imaging and discussed in context with the current literature.

Results and Conclusion Overuse may cause stenosis in the fibrous outer layer of the retinacula and the A1 annular ligaments as well as tendinosis. In contrast, proliferative tenosynovitis is a disease of the synovial inner layer of the tendon sheath with tendon infiltration and tendinitis. Pyogenic tenosynovitis favors the flexor compartments. Because of the narrow spaces in the hand, a high-resolution MRI technique must be used.

Key Points:

- Diseases of the tendons and tendon sheaths may have a mechanical, degenerative, metabolic, systemic inflammatory, or infectious etiology.
- Fibrous tunnels and bony prominences in close proximity to crossing tendons predispose to mechanical tendon irritation at typical sites of the hand.
- Stenosing tenosynovitis occurs in the fibrous layer of the extensor retinaculum or the A1 annular pulleys. The most frequent manifestations are the “trigger finger” and de Quervain disease.
- Proliferative tenosynovitis affects the synovial layer of the tendon sheaths before infiltrating the tendons. The classic representative is rheumatoid arthritis.

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ZUSAMMENFASSUNG

Hintergrund Die Sehnen der Hand verlaufen eng benachbart, teils überkreuzend und an artikulären Umlenkorten innerhalb von retinakulären Tunnelsystemen. Die umhüllenden Sehnenscheiden sind häufig Manifestationsorte systemischer oder infektiöser Entzündungen.

Methode In der Übersichtsarbeit werden die Krankheitsentitäten der Sehnen- und Sehnenscheiden mit der Pathophysiologie und den klinischen Erscheinungsbildern beschrieben. Die Diagnostik der Sehnenerkrankungen wird anhand der MRT-Bildgebung erläutert und mit der aktuellen Literatur diskutiert.

Ergebnisse und Schlussfolgerung Überlastungen können zu Stenosen in den fibrösen Außenschichten der Retinacula und der A1-Ringbänder sowie zu Tendinosen führen. Dagegen ist die proliferative Tenosynovialitis eine Erkrankung der synovialen Innenschicht der Sehnenscheide mit Sehneninfiltration und Tendinitis. Die purulente Tenosynovialitis bevorzugt die Flexorenkompartimente. Wegen der engen Raumverhältnisse an der Hand müssen hochauflösende MRT-Techniken verwendet werden.

Kernaussagen:

- Erkrankungen der Sehnen und Sehnenscheiden können mechanische, degenerative, metabolische, systemische, entzündliche oder infektiöse Ursachen haben.

- An der Hand prädisponieren Tunnelengen, Knochenvorsprünge sowie eng benachbarte und kreuzende Sehnen zur mechanischen und topografischen Sehnenirritation.
- Stenosierende Tendovaginosen spielen sich im Stratum fibrosum des Retinaculum extensorum oder der A1-Ringbänder ab. Häufigste Vertreter sind der „Trigger-Finger“ und die Tenovaginitis de Quervain.
- Proliferative Tenosynovialitiden betreffen das Stratum synoviale, bevor sie im Sinne einer sekundären Tendinitis in die Sehnen infiltrieren. Klassischer Vertreter ist die rheumatoide Arthritis.

Introduction

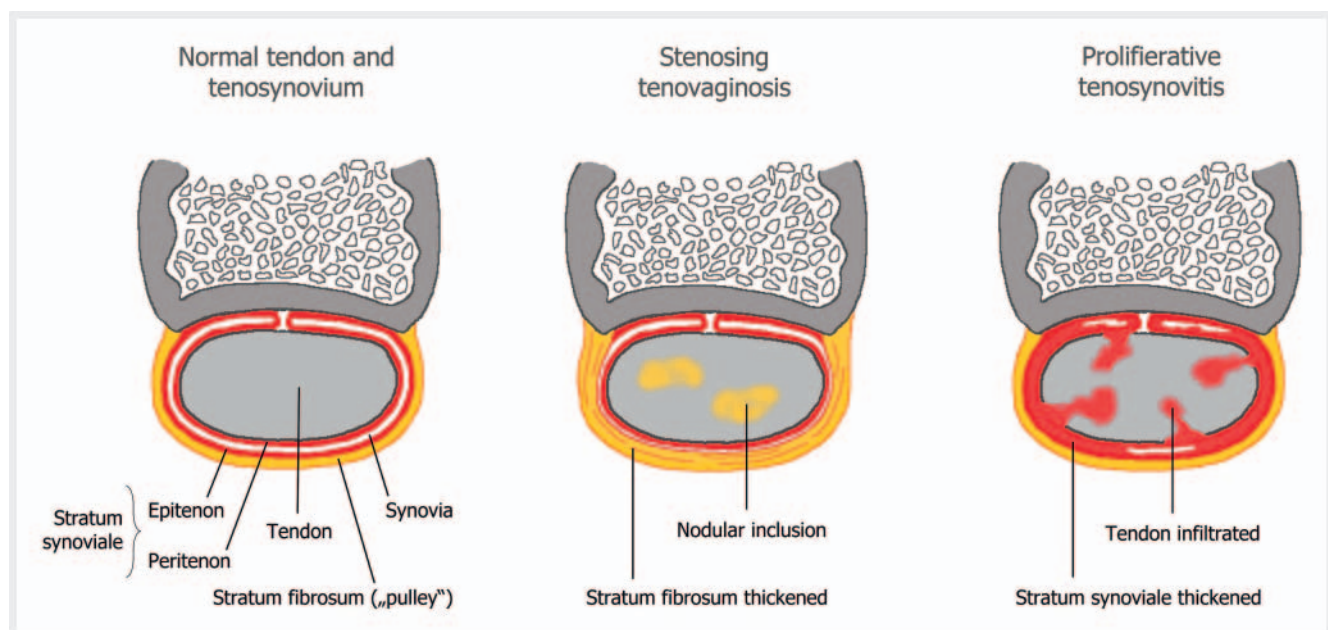
After fractures, tendon disorders are the second most common complaint evaluated by hand care professionals [1]. Besides the extensive use of the hands in everyday life, anatomical conditions predispose us to the high incidence of tendon pathologies, for example, the close proximity of the tendons to each other in the carpal tunnel. Moreover, the extrinsic muscles of the hand are located on the forearm, from where the extrinsic tendons for the wrist and fingers cross the carpus before inserting at the midhand or the fingers [2]. This tendon topography makes the carpus an “intercalated” segment. Finally and most importantly, many tendon segments of the hand are surrounded by protective tendon sheaths in proximity to joints or bony prominences [3].

As a result, not only lesions of the tendons but also of the tendon sheaths (“tenosynovium”) and combined pathologies can arise. Potential causes of tendon and/or tendon sheath disorders are acute injuries, chronic overuse, systemic and infectious inflammation of the tenosynovium, and rare tumors of the tendon sheath.

Anatomy of the tendon and tenosynovium

A **tendon** is the connective link between a muscle and bone. It consists of tenocytes and an intercellular substance with type I collagen fibers embedded [3]. Multiple interlinked collagen molecules form tendon fibers and tendon fiber bundles. The poor regenerative capability of tendons is mainly due to their poor vascularization. Two different sheath structures are to be distinguished:

Tendon sheaths (synonym: **tenosynovium**) enclose segments where the tendons pass over joints or run around bony prominences [4]. Consisting of an outer **parietal layer** (stratum fibrosum) and an inner **visceral layer** (stratum synoviale), the sheath protects the tendon and reduces its friction. The parietal layer is a strong fibrous mesh, which is reinforced either by annular pulleys or fibrous tracts and septa. The visceral layer is composed of synoviocytes and forms a double lamella: The outer synovial sheet (**epitenon**) is connected to the fibrous parietal layer and the inner synovial sheet (**peritenon**) is in close proximity to the tendon surface (► Fig. 1a). Synovial fluid (**synovia**) fills the space between the epitenon and peritenon.



► **Fig. 1** Schematic display of tendons and tendon sheath anatomy. Left: Normal anatomy. The tendon is surrounded by an inner synovial layer and an outer fibrous layer. Middle: Stenosing tenovaginitis. The stenosing stratum fibrosum is thickened with nodular fibrous inclusions in the tendon. Right: Proliferative tenosynovitis of rheumatoid arthritis. The synovial layer is massively thickened, and tendon infiltration is present.

The **paratenon** surrounds extra-articular tendon segments where the tendon sheath is absent. There, the tendon is enclosed by a membrane-like layer consisting of loose connective tissue.

Pathology of the tendon and tenosynovium

The term “tendinopathy” is used for a broad spectrum of non-rupture pathologies of the tendons in acute and chronic conditions [3].

Tendinosis

A non-inflammatory lesion of a tendon is called “tendinosis”. Common causes include age-related tendon degeneration and tendon overuse [5]. Rarely, tendon impingement, systemic glucocorticoid medication or dyslipidemia in xanthomatosis are responsible. Tendinosis is a chronic remodeling caused by micro-ruptures [5, 6]. Constriction of the venules leads to increased blood flow, plasma leaks, and edematous swelling of the injured tendon. Tendon remodeling slowly progresses over time, since injured collagen fibers are replaced by less tear-resistant ones. The affected tendon segment appears thickened and is potentially at risk of rupture.

Tendinitis

The term tendinitis is often used incorrectly in clinical practice and is mistaken for the chronic remodeling process of tendinosis. Histologically, tendinitis is an inflammatory reaction of the tendon to an external noxious agent, either to immunologically mediated antibodies in rheumatic diseases or to a pathogen in bacterial infections. The tendon substance is infiltrated by leukocytes, lymphocytes, and others [7]. In rheumatoid arthritis, inflammatory invasion of the tenosynovium results in tendon thinning, ischemia, and rupture [8, 9], while attrition occurs adjacent to bony erosions, e. g., at the ulnar head, the Lister tubercle, or at the distal scaphoid pole.

Paratendinitis

Paratendinitis is defined as non-specific inflammation and potentially fibrosis of the paratenon, i. e., the soft tissues surrounding a sheathless tendon segment. Paratendinitis is less common in the hand.

Reactive tenovaginitis

Reactive tenovaginitis is caused by a preceding strain [10], such as repetitive flexion-extension and pro-supination, for example, in racket athletes and craftsmen. Mechanical stress leads to hyperemia in the synovial cell layer with stimulation of synoviocytes to increasingly produce synovial fluid. Most often, reactive tenovaginitis is found in the ECU tendon sheath. The term “reactive tenosynovitis” should be avoided since it implies inflammation instead of overuse as the causative agent.

Stenosing tenovaginitis

This condition is caused by narrowing of a tendon’s retinacular sheath at osteo-fibrous tunnels, resulting in entrapment of the encased tendon [6]. The term “stenosing tenovaginitis” is the

accurate description because the outer fibrous layer of the tendon sheath is mainly involved in this non-inflammatory pathology [11]. Osteo-fibrous channels predispose to stenoses at sites where the tendon changes direction. This anatomical feature is found at the A1 annular pulleys and at the extensor retinaculum [12]. The fibrous layer of the flexor tendon sheaths of the thumb and fingers is interconnected to annular and cruciate pulleys. These reinforcing ligaments are lattice-like interwoven collagen fiber bundles. With similar histological composition, the extensor retinaculum forms six channels for the extensor tendons at the wrist. In the case of discrepancy between the diameters of the tendon sheath and the tendon, functional stenosis may occur with aggravation in chronic overuse. Chronic irritation of the gliding layers results in fibrocartilaginous metaplasia, fibrovascular proliferation, and thickening of the retinacular pulley with increasing loss of tendon function (► **Fig. 1b**) [13].

Proliferative tenosynovitis

True tenosynovitis develops in about 64 % to 95 % of patients suffering from **rheumatoid arthritis**. Proliferative synovitis of the tendon sheaths occurs in a similar manner to articular involvement [14]. However, invasion of the tenosynovium may begin months before joint manifestations are discernible [15]. In the early stages of rheumatoid arthritis, synovial tissue is sparse, and the tendon sheath is filled with effusion and small fibrinoid “rice bodies”. As the disease progresses, the synovium thickens (► **Fig. 1c**). The inflammatory tissue infiltrates the tendon substance and proliferating synovitis results in destruction of the tendon and the tenosynovium. Eventually, tendon rupture may occur due to tendon weakening and synovial adhesions. The three most common sites for rheumatoid tendon sheath involvement are located at the dorsal and palmar aspects of the wrist as well as the palmar aspect of the fingers. Extrinsic factors (sharp-edged protuberances of the radius, ulnar head and scaphoid), and vascular factors (reduced perfusion at the retinacula and pulleys) also play a role [8]. These factors can lead to compromising deformities, such as ulnar deviation as well as swan neck and Boutonniere deformities of the fingers.

Rarely, inflammatory tenosynovitis occurs in metabolic diseases, and in pyogenic or atypical tenosynovitis.

- In **gouty tenosynovitis**, acute inflammation is triggered by crystalline deposition of monosodium urate in the tendon sheath, followed by phagocytosis and lysosomal release [16].
- **Hydroxylapatite dihydrate disease (HADD)** is induced by the release of hydroxylapatite bodies into the tenosynovial sheaths [17]. The cause is unknown.
- **Calcium pyrophosphate dihydrate (CPPD disease, pseudo-gout)** can precipitate within the carpal tunnel and cause acute inflammatory tenosynovitis.
- In **amyloidosis**, β_2 -microglobulin is accumulated in bones, tendon sheaths, and other soft tissues of patients with renal insufficiency requiring dialysis.
- **Pyogenic tenosynovitis** is a closed-space infection of the flexor tendon sheath mostly caused by staphylococcal or streptococcal species, less commonly by neisserial, gonococcal, or mycobacterial pathogens [18].

► **Table 1** MRI sequence recommendations for tendon pathologies of the distal forearm and hand [21].

Area		Slice orientation	Sequence type	Thickness (mm)	i. v. Gad.
Forearm and wrist	As standard	Coronal	PD FSE fatsat	2.5	No
		Transaxial	PD FSE fatsat	3.0	No
		Transaxial	T1 FSE	3.0	No
		Transaxial	T1 FSE fatsat	3.0	Yes
		Sagittal	T1 FSE	2.5	Yes
	As an option	Coronal	T1 FSE fatsat	2.5	Yes
		Coronal	3 D GRE / PD	0.5	Yes
Midhand and fingers	As standard	Transaxial	PD FSE fatsat	2.5	No
		Sagittal	PD FSE fatsat	1.5	No
		Sagittal	T1 FSE	1.5	No
		Sagittal	T1 FSE fatsat	1.5	Yes
		Coronal	T1 FSE	1.5	Yes
	As an option	Transaxial	T1 FSE fatsat	2.5	Yes
		Coronal	3 D GRE / PD	0.5	Yes

Imaging of the tendons and tenosynovium

Ultrasound (US) and magnetic resonance imaging (MRI) are the primary means of tendon examination. US is undoubtedly the first-line modality because it offers the advantages of dynamic analysis and Doppler function for assessing inflammation [19]. However, MRI is the method of choice in the case of inconclusive US imaging and for preoperative planning due to its excellent overview capability [20].

With the tendons being aligned parallel to the forearm bones, thin-slice axial stacks are helpful for the analysis of their internal structure and the tendon sheaths. Long-axis views depict the tendons in their entirety, with the coronal plane being most valuable at the level of the wrist and the sagittal plane at the level of the fingers. 3D MRI is beneficial for oblique or curved image reconstruction in complex tendon anatomy, for example, when examining the EPL tendon. If available, 3 Tesla scanners should be preferred as they ensure higher signal intensity. The use of multi-channel phased-array coils (8- or 16-channel coils) is crucial for parallel imaging. In-plane resolution should be about 0.3 mm, which is realized with a field of view of 80 mm with a matrix of 320 × 320. A protocol recommendation [21] is provided in ► **Table 1**.

Tendon appearance should not differ between T1- and T2-weighted sequences. A pitfall in tendon assessment is the magic angle effect [22]. The EPL and FPL tendons are particularly affected by this phenomenon, but either using sequences with a longer echo time or repositioning the hand provides a solution. Tendons can also have an increased internal signal in asymptomatic individuals as seen in the higher density of fascicles of the APL and ECU tendons, or in preexisting tendon degeneration with mucoid inclusions.

Early MRI signs of tendinosis include moderate tendon thickening, contour blurring, and a focal or diffuse signal intensity increase [23].

As tendinosis progresses, tendon thickening and signal changes increase. MRI signs of tendinosis can also be found in asymptomatic individuals. Chronic enthesopathy is often associated with bone marrow edema and enthesopathic bone proliferation at the tendon insertion.

In tenovaginitis, MRI shows a non-specific synovial effusion, which should normally not exceed 2 mm in thickness [24]. Furthermore, the tendon sheath is distended with edge blurring at the site of stenosis, mostly at the extensor retinaculum or the A1 annular pulleys. These structures may appear hyperintense in T2-weighted sequences.

Tenosynovitis is characterized by an increased quantity of synovial fluid surrounding the tendon, and by areas of thickened or irregular wavy lines within the tendon sheath. Edematous and hyperemic soft-tissue alterations are frequently encountered in acute stages as well as contrast enhancement of varying intensity, which depends on the underlying disease and stage.

Disorders of the extensor mechanism

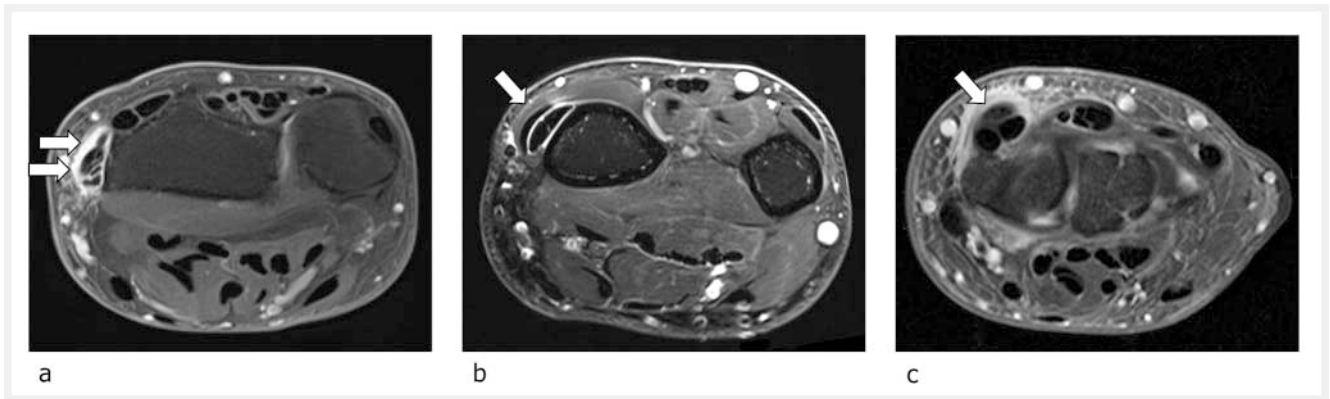
In the cross-sectional view, the extensor retinaculum surrounds the tendons circularly with supra-tendinous and infra-tendinous fibrous tracts running horizontally and the septa running perpendicularly to them, thereby defining six fibro-osseous tendon compartments [12, 25]. The number of extensor tendons is based on their anatomical positions at the wrist from radial to ulnar:

I = abductor pollicis longus (APL) and extensor pollicis brevis (EPB) tendons,

II = extensor carpi radialis longus (ECRL) and extensor carpi radialis brevis (ECRB) tendons,

III = extensor pollicis longus (EPL) tendon,

IV = extensor digitorum communis (EDC) and extensor indicis proprius (EIP) tendons,



► **Fig. 2** Stenosing tenovaginitis of the radial extensor tendons. **a** de Quervain's disease: The APL and EPB tendons (arrows) are enclosed within a thickened tendon sheath. **b** Proximal intersection syndrome: Friction at the musculotendinous intersection where the APL (arrow) crosses the ECRL and ECRB tendons. **c** Distal intersection syndrome: Friction at the musculotendinous intersection where the EPL tendon (arrow) crosses the ECRL and ECRB tendons (all fat-saturated axial T1 FSE after intravenous application of gadolinium).

V = extensor digiti minimi (EDM) tendon and
VI = extensor carpi ulnaris (ECU) tendon.

The pathologies of the extensor tendons are explained below in this order, with distally a slightly modified order due to tendon crossings.

Stenosing APL and EPB tenovaginitis (de Quervain disease)

De Quervain disease is the second most common entrapment tendinopathy of the hand. Secretarial and nursing workers and also mothers due to lifting their infants are primarily affected [26]. Friction of the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) tendon is caused by overuse through repetitive abduction of the thumb with simultaneous ulnar abduction of the wrist. The extensor retinaculum shows increased vascularization, fibrosis, and myxoid degeneration leading to shrinkage of the retinaculum and thus to tendon entrapment (► Fig. 2a). Signs of tendinosis, subcutaneous edema, or periosteal reaction at the radial styloid are present [23, 25], but there are no signs of inflammation [27]. A longitudinal split of the APL tendon is frequently seen. An inter-tendinous septum with a sub-compartment for the EPB tendon must be ruled out before surgical therapy.

Stenosing ECRL and ECRB tenovaginitis (proximal intersection syndrome)

Proximal intersection syndrome (“oarsmen’s wrist”) occurs at the crossing of the APL and extensor pollicis brevis (EPB) myotendinous junctions with the extensor carpi radialis longus (ECRL) and the extensor carpi radialis brevis (ECRB) tendons. Friction is usually induced by overuse of the forearm muscles during sporting and work activities [28] as well as by stenotic alterations of the tendon sheaths [29]. Due to the location of the intersection 4–6 cm proximal to Lister’s tubercle, the field of view must be shifted proximally on MRI. Diagnostic signs include synovial sheath fluid in the first and second extensor compartments, extra-synovial fluid collection at the intersection and muscle edema (► Fig. 2b) [23, 30].

Stenosing EPL tenovaginitis (distal intersection syndrome)

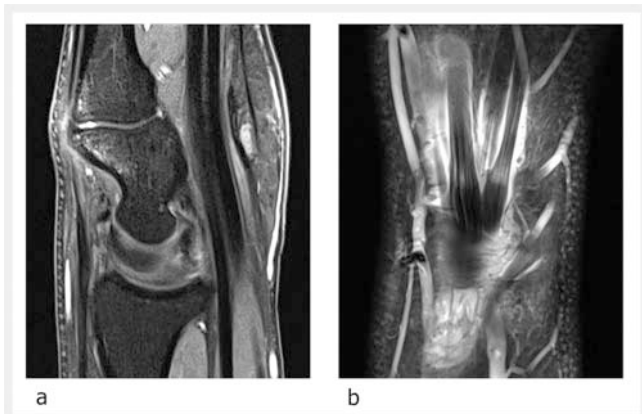
Distal intersection syndrome is much less common than its proximal counterpart. It refers to the crossing of the extensor pollicis longus (EPL) tendon over the ECRL and ECRB tendons [31]. Several mechanisms can cause EPL tenovaginitis: In chronic overuse, the EPL tendon is abraded by Lister’s tubercle, which increases the risk of tendon rupture (drummer boy’s palsy). In addition to overuse, primary blunt trauma to the EPL tendon, compression by the antebrachial fascia (if thickened), or friction from osteophytes in scapholunate advanced collapse deformity (SLAC wrist) or scaphoid fractures may cause tendon laceration. Synovial effusion, peritendinous edema, and contrast enhancement around the affected tendons are seen on MRI (► Fig. 2c). Inter-compartmental inflammation is maintained by a foramen connecting the EPL and ECRB tendon sheaths.

Rupture of the extensor pollicis longus (EPL) tendon

The EPL tendon is the most commonly torn extensor tendon of the wrist, partly due to a hypovascularized zone near Lister’s tubercle, where the tendon is only supplied by synovial fluid. Tendon ruptures may occur in distal radius fractures including an avulsed Lister tubercle or in protrusion of osteosynthesis material [32]. Also, rheumatoid arthritis, lupus erythematosus, and gout can cause tendon attrition [23]. Tendon sheath effusion is only an indirect rupture sign. The retracted EPL ends may appear tendinopathic [33]. Due to the curved course of the EPL tendon, the best imaging results can be achieved with curved or double-angled reconstructions of a 3 D MRI sequence.

Reactive EDC tenovaginitis (including carpal boss syndrome)

After overuse or straining of the extensor digitorum communis (EDC) muscles, reactive tenovaginitis may occur in the fourth extensor tendon compartment. This overuse form is reversible after immobilization [10]. The “carpal boss” is a bony prominence on the dorsal side of the carpometacarpal joint, formed either by



► **Fig. 3** Stenosing tenovaginitis of the extensor tendons of the fingers. **a** Carpal boss syndrome: Enthesopathic protrusions at the capitate bone and the base of the metacarpal III lead to synovial irritation of the tendons in the fourth extensor compartment. The extensor retinaculum is massively thickened resulting in tendinosis of the extensor tendons (both fat-saturated coronal T1 FSE after intravenous application of gadolinium).

an enthesopathic protuberance at the base of metacarpal II or III or by a normal variant, the *os capitatum secundarium*. In sagittal and axial MRI slices, the carpal boss hump becomes evident. In symptomatic patients, local bone marrow edema and tenovaginitis of the adjacent ECRB or EDC III tendons are suggestive (► **Fig. 3a**) [34, 35].

Stenosing EDC tenovaginitis

Stenosis of the fourth compartment is very rare, occurring occasionally in the presence of malunited radius fractures and osteoarthritis of the distal radioulnar joint [36]. A case series highlighting the ultrasound findings in 18 patients with overuse-related stenoses was published recently [37]. The index and little fingers are most frequently affected because their extensor tendons take an oblique course at the distal end of the fourth extensor compartment [36]. MRI findings have not been described in the literature thus far. From our experience, thickened tendon sheaths with mild edema and moderate contrast enhancement occur (► **Fig. 3b**). A hyperintense tendon signal in T1- and T2-weighted sequences indicates tendinosis.

Stenosing EIP tenovaginitis (EIP syndrome)

Isolated tenovaginitis may be caused by congenital anomalies of the extensor indicis proprius (EIP) muscle. Abnormal myotendinous junctions and an accessory extensor indicis brevis muscle have previously been identified [38]. These variants lead to hypertrophy of the EIP muscle, potentially resulting in EIP tenovaginitis [25].

Proximal rheumatoid EDC and EDM ruptures (Vaughan-Jackson syndrome)

With up to 64%, the EDC tendons and the EDM tendon are most frequently affected in rheumatoid arthritis due to their close

proximity to the ulnar head, which serves as a predilection site for inflammatory manifestations [14]. Tendon ruptures occur in a sequence from ulnar to radial [9, 39]. The extension deficit of the little finger depends on whether the EDM tendon is also torn. Simultaneously, ulnopalmar dislocation of the MCP joints occurs. As a result, the extensor tendons are displaced into the grooves between the metacarpal heads, resulting in further loss of finger extension. In most cases, articular synovitis and tenosynovitis precedes ulnar dislocation and tendon ruptures [20]. It is important for surgical therapy to determine the location of the tendon rupture and the exact extent of tendon retraction.

Distal rheumatoid EDC rupture

Rarely, rheumatoid arthritis manifests further distally at the extensor tendon. Then, synovial inflammatory tissue is found in the dorsal recess of the PIP joint beneath the central slip. Focal synovitis weakens the central slip and causes a rupture with ensuing Boutonnière deformity [40]. With the use of sagittal MRI planes, both an extensor tendon rupture and synovial inflammatory tissue can be visualized (► **Fig. 7c**).

Injury of the extensor hood

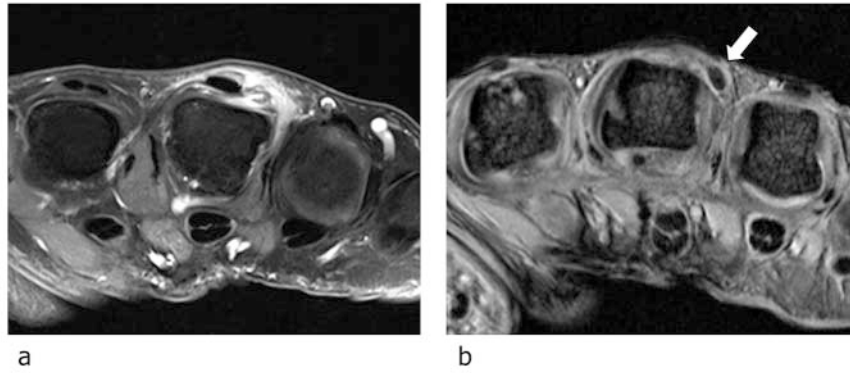
At the MCP joints, the EDC tendons stretch out to form the extensor hoods whose main components are the two lateral bands and the median slip [2]. Insufficiency or rupture of the extensor hood results in decentralization of the entire extensor apparatus at the level of the MCP joint, resulting in inability to extend the finger actively [41]. Etiology may be congenital laxity of the connective tissue, direct trauma to the dorsum of the MCP joint (“boxer’s knuckle”), a forced flexion injury, or rheumatoid synovitis. Extensor hood lesions are best depicted on axial MR images with asymmetry and blurring of the extensor tendon and hood (► **Fig. 4a, b**) [42]. In most cases, the radial lateral band is injured, and the extensor hood is dislocated to the ulnar side [43]. The interosseus and/or lumbrical tendons are dehiscent at the injury site. The vascularization of the extensor apparatus predisposes to adhesions [43].

Insertional tear of the central EDC slip

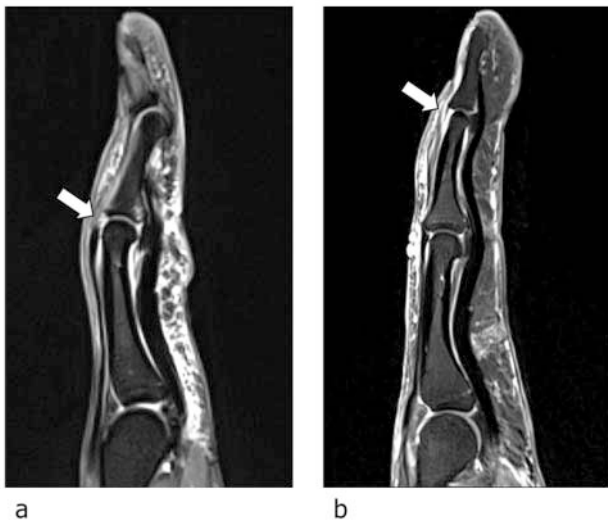
Rupture of the central slip at the base of the middle phalanx (extensor zone 3) results from forced flexion of the PIP joint while the extensor tendon is firmly tense [44]. Less frequently, a fracture of the central slip attachment can occur. Clinically, this injury type is difficult to discern because intact lateral bands can compensate for the extension deficit. Untreated rupture of the central slip leads to the “Boutonnière deformity” which is characterized by PIP flexion and compensatory DIP hyperextension due to palmar dislocation of the lateral bands [40]. Tears of the central EDC slip are visualized with high-resolution sagittal MR images (► **Fig. 5a**) [43].

Insertional tear of lateral EDC bands (Mallet finger)

At the dorsal base of the distal phalanx (extensor zone 1), rupture of the extensor tendon results either from forced flexion of the distal phalanx when the extensor apparatus is tense or from the



► **Fig. 4** Injuries of the extensor hood. **a** Traumatic rupture on the ulnar side of the middle finger's extensor hood. Only minor radial dislocation of the tendon is visible (fat-saturated axial T1 FSE after intravenous application of gadolinium). **b** Chronic ulnar dislocation of the extensor tendon after untreated finger injury in childhood (axial T2* GRE).



► **Fig. 5** Tears of the extensor apparatus. **a** Tear of the central slip (arrow) at the base of the middle phalanx (fat-saturated sagittal PD FSE). **b** Tear of the terminal lateral bands (arrow) at the base of the distal phalanx (fat-saturated sagittal T1 FSE after intravenous application of gadolinium).

impact of an axial force [44]. Ligamentous tears and osteoligamentous avulsion fractures must be differentiated. If left untreated, a mallet deformity frequently progresses to a swan-neck deformity (flexion deformity of the DIP joint and hyperextension of the PIP joint). Sagittal MRI planes can be used to determine the degree of tendon retraction and articular surface involvement (► **Fig. 5b**). If an avulsion fragment measures more than 25% of the sagittal articular surface, DIP joint instability may be suspected [45].

Reactive and stenosing ECU tenovaginitis

This is the second most common tendinopathy of the wrist. Reactive ECU tenovaginitis is caused by repetitive wrist flexion-extension and pro-supination motion, e. g., in racket sports or crafts-

men [23]. The high incidence of ECU tenovaginitis can be explained by the ulnar angulation of the tendon and its lateral translation during pro-supination. An ECU tendon sheath effusion may be seen. The rare stenosing ECU tenovaginitis is caused by repetitive distortion that leads to ECU tendon strain and entrapment. MRI is characterized by a thickened and mildly hyperintense ECU tendon sheath.

Instability of the ECU tendon

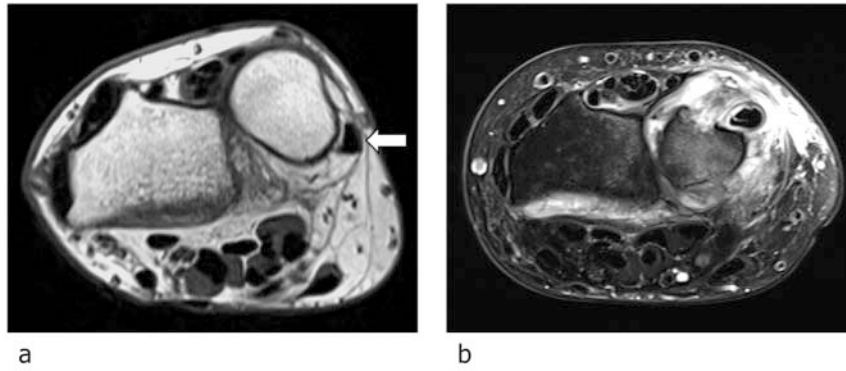
The extensor retinaculum, which passes over the ECU tendon, has no retaining function [46]. Tendon instability results from chronic subsheath overuse or sudden rotational movement, as seen, for example, during backhand tennis swings (supinated forearm, wrist in flexion, and ulnar deviation) [47]. Lesions comprise medial and lateral tears as well as periosteal delamination of the ECU subsheath [48]. Dysfunction ranges from discrete tendon subluxation to gross dislocation. On axial MR images, the unstable ECU tendon is located outside the shallow cavity of the ulnar head (► **Fig. 6a, b**) and the torn subsheath is also visualized.

Rheumatoid ECU tenosynovitis

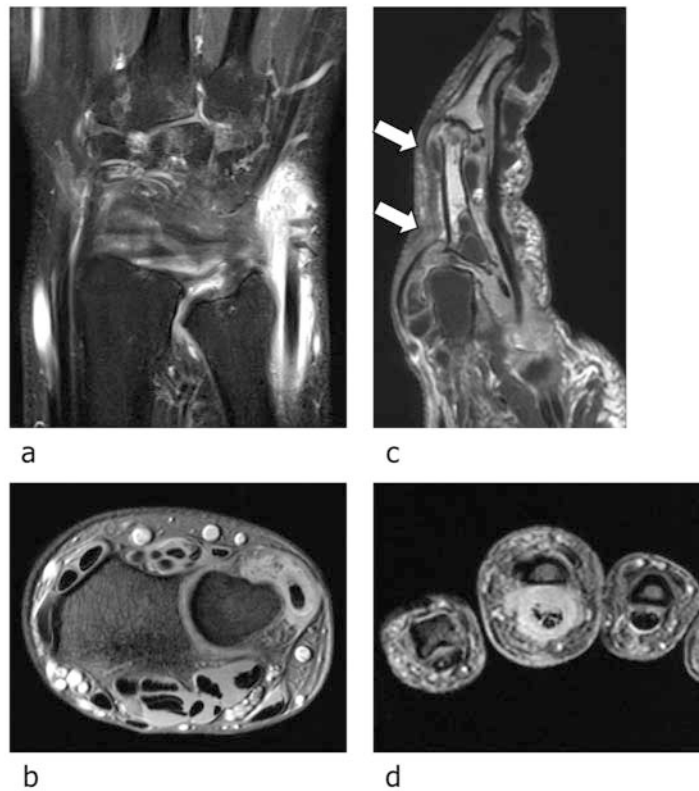
Rheumatoid arthritis manifests early and frequently at the synovial layers of the ulnocarpal compartment, involving the ECU synovial sheath, the tip of the ulnar styloid process, and the distal radioulnar joint [15]. MRI shows effusion and thickening of the ECU synovial sheath with edema and contrast enhancement, while signs of ECU tendinitis are also seen in many cases (► **Fig. 7a, b**).

Rupture of the ECU tendon

ECU tendon tears are almost exclusively seen in advanced rheumatoid arthritis [15]. Ulno-palmar subluxation of the ECU tendon and inflammatory alterations of the TFCC are important prognostic factors for impending tendon rupture. In non-rheumatoid patients, rupture of the ECU tendon is a rare condition [49]. In MRI, the degree of tendon retraction must be determined. The



► **Fig. 6** Dislocation of the ECU tendon. **a** Chronic instability of the distal radioulnar joint with secondary ECU tendon dislocation (arrow) in ulno-palmar direction. The extensor retinaculum is intact (axial T2 FSE). **b** Combined flexion-inclination injury with osteoligamentous avulsion of the ECU subsheath and ulnar ECU tendon dislocation. Surrounding edema and hematoma are present (fat-saturated axial PD FSE).



► **Fig. 7** Proliferating tenosynovitis in rheumatoid arthritis. **a** and **b** Massive synovitis of the ECU tendon which is circularly surrounded by pannus tissue with multiple rice body inclusions. Signs of inflammation are also visible in the ulnocarpal compartment (**a**: fat-saturated coronal T1 FSE after intravenous application of gadolinium; **b**: axial T2* GRE). **c** and **d** Tenosynovial involvement of the middle finger in long-standing rheumatoid arthritis. The central slip of the extensor apparatus is ruptured at its insertion and retracted (arrows). Circular synovitis around the flexor tendons and tendinitis are evident, as well as palmar subluxation in the MCP III joint (**c**: sagittal T1 FSE; **d**: axial T2* GRE).

ECU tendon should be examined for a longitudinal split tear, which must be distinguished from a bifurcated tendon [50].

Disorders of the flexor mechanism

The flexor tendons for the thumb (FPL) and fingers (FDP and FDS) run beneath the flexor retinaculum within the carpal tunnel, while



► **Fig. 8** Flexor tendon lesions. **a** Acute FCR tenovaginitis with synovial effusion affecting the tendon segments adjacent to the distal forearm, distal scaphoid pole, trapezium, and metacarpal bone II. **b** Sub-acute tear of the FDP tendon of the middle finger. The proximal tendon stump (arrow) is considerably retracted. Distally, the retinacular tendon sheath remains visible (both fat-saturated sagittal PD FSE).

the flexor tendons for the wrist (FCR and FCU) course superficially to the carpal tunnel [2].

Stenosing FCR tenovaginitis (FCR tunnel syndrome)

The FCR tendon runs around the distal scaphoid pole, which acts as a hypomochlion, before coursing through the narrow fibrous tunnel of the trapezium. The most common cause of FCR tenovaginitis is triscaphe (STT) osteoarthritis [49–52]. Osteophytes lead to attrition of the FCR tendon and tenovaginitis, which further constrains the trapezium. Due to the tendon’s course through osseous surroundings, MRI is better than ultrasound in diagnosing FCR tunnel syndrome. FCR tendon thickening, and synovial effusion are always present (► **Fig. 8a**) [51]. Bone marrow edema and cystic erosions can be seen at the distal scaphoid pole and trapezium bone. Symptoms at the proximal thumb are often erroneously attributed to STT or trapezio-metacarpal arthritis.

Tear of the FCR tendon

Osteophytes associated with the STT osteoarthritis [52], a narrow trapezium, and rheumatoid arthritis significantly contribute to traumatic rupture of the FCR tendon. In MRI, the discontinuity of the FCR tendon is usually filled with hematoma or scar tissue with additional findings of stenosing tenovaginitis [51].

FCU insertional tendinosis

FCU tendinosis at the pisiforme insertion is the result of repetitive flexion-extension load. While early stages are reversible, angio-fibroplastic hyperplasia of the sheathless tendon and peritendinitis develop in the chronic stage [53]. On MRI, a thickened FCU ten-

don directly proximal to the pisiforme bone and peritendinous soft tissue edema are seen.

Calcific FCU peritendinitis (HADD)

Manifestations of acute inflammatory hydroxyapatite deposition disease (HADD), also referred to as “calcific peritendinitis”, are often located in close proximity to the FCU tendon [54]. While the tendon substance is not involved primarily, it may be affected by paratendinous inflammation. T2-weighted sequences depict tendon enlargement, tendon edema, and contrast enhancement as well as peritendinitis. Radiographs are helpful in detecting fluffy calcifications.

Stenosing FDP and FDS tenovaginitis (carpal tunnel syndrome)

The tendons of the carpal tunnel (flexor zone 4) are enclosed by synovial bursae. Compression of the median nerve is caused either by a mass (e. g., ganglion cyst) or, most frequently, by synovial hypertrophy of the flexor tendon sheaths due to overuse-induced tenovaginitis [55]. On axial T2-weighted images, the flexor tendons are surrounded by a hyperintense rim that is composed of synovial effusion and edematous synovitis. Since circular thickening of the flexor tendon sheaths is non-specific, additional signs are required to diagnose carpal tunnel syndrome on MRI: edema and increased cross-sectional area of the median nerve > 15 mm² proximal and distal to the carpal tunnel, nerve flattening within the carpal tunnel, and palmar bowing of the flexor retinaculum [56].

Pyogenic FDP, FDS and FPL tenosynovitis

Direct internalization of pathogens from penetrating injuries is the most frequent cause of pyogenic tenosynovitis, whereas transmission from adjacent soft tissue infection and hematogenous dissemination is uncommon [18]. The most common site of entry is the PIP joint of the middle finger. There is a gradual infection process consisting of initial exudative effusion, florid tenosynovitis with pus, and finally tissue necrosis and tendon rupture [57]. The hand has many anatomic compartments, and pyogenic tenosynovitis can spread from adjacent anatomic spaces. From our experience, the frequently described “V-phlegmons” rarely play a role, but infections that spread from the thumb into the carpal canal and the Parona space may induce the development of acute carpal tunnel syndrome. Predisposing factors are diabetes mellitus and atherosclerosis. MRI features include voluminous effusion within a thickened and contrast-enhancing synovial sheath. Debris and occasionally foreign bodies may be present within the synovial fluid, while rice bodies are characteristic in tuberculous tenosynovitis [58]. MRI can be used to determine the exact extent of a potential abscess formation, which is important before surgical treatment [59].

Rheumatoid FDP and FDS tenosynovitis

Proliferative tenosynovitis may be present in the early stages of rheumatoid and seronegative arthritis, occasionally as the very first symptom [60]. When synovial proliferation surrounds the

flexor tendon sheaths of the wrist in circular fashion, compression of the median nerve leads to clinical symptoms of carpal tunnel syndrome [15, 23]. Proliferative synovitis may cause a similar effect at the fibro-osseous pulleys of the finger flexor tendons. Because these pulleys are strong and not extensible, even mild synovial proliferation or rheumatoid nodules impair finger function. On MRI, the tenosynovium is thickened by inflammatory pannus tissue, which shows edema and strong contrast enhancement (► Fig. 7 d). In long-term rheumatoid arthritis, the tendons themselves are also inflamed and appear thickened and hyperintense. Tears of the flexor tendons are less common than ruptures of the extensor tendons [8]. Rupture of one FDP tendon can be compensated fairly well by an intact FDS tendon.

Injury of the third lumbrical muscle

Due to its bipennate connection to FDP tendons III and IV, the third lumbrical muscle is at risk of rupture if affected by increased transverse tension, for example in free climbers [61, 62]. The muscle rupture can be visualized on high-resolution T2-weighted images by means of muscle edema and fiber discontinuity.

Rupture of the FPL tendon (Mannerfelt syndrome)

Tears of the FPL tendon occur predominantly in rheumatoid arthritis of the wrist, with almost all ruptures being in women [8]. Ruptures due to attrition are caused by bony spurs of the scaphoid bone and lead to flexion loss of the distal phalanx. On MRI, the usual rupture criteria are seen, as described below for the FDP tendons [63].

Proximal tear of the FDP tendon

After osteosynthesis in distal radius fractures, the distal edge of an inserted volar osteosynthesis plate can erode and damage the FDP tendons adjacent to the radius. This complication usually occurs if the volar plate is implanted distally from the watershed line of the radius. MRI can be used to visualize the rupture site of the FDP tendon and the gap between the tendon ends [63]. However, metallic artifacts may reduce image quality. Therefore, ultrasound should be used as the primary imaging method [64].

Distal injury of the FDP tendon (Jersey finger)

Rarely, the FDP tendon tears off directly at its insertion (flexor zone I) or in the form of an osteoligamentous avulsion fracture at the palmar aspect of the distal phalanx [44]. The injury is caused by forced hyperextension of the DIP joint with the finger being simultaneously flexed. Tendon damage due to focal inflammation or loss of connective tissue may be present. FDP avulsion injuries are graded according to Leddy and Packer [65]. In type I FDP tendon rupture, MRI is particularly useful because it can aid in determining the exact extent of tendon retraction. In contrast, there is no indication for MRI in injuries of types II and III because the fragment (with FDP tendon attached) is directly visualized on X-ray scans.

Open injury of the FDS and FDP tendons

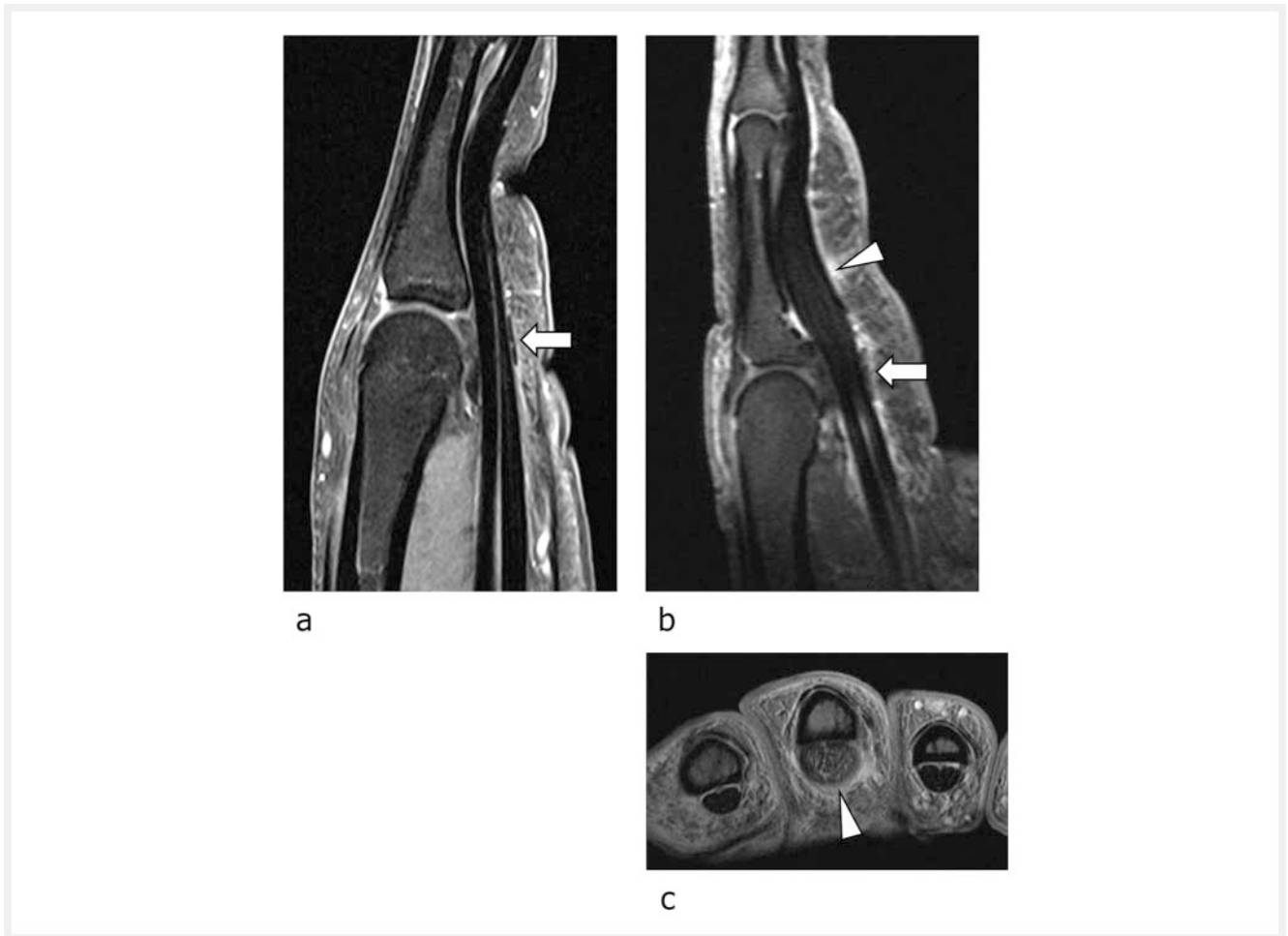
Injuries of the flexor tendons of fingers II-V are frequently open lacerations, whereby the index finger is most often affected. The tendons are mostly damaged in flexor zone 2 [66, 67]. MRI can help to uncover the precise location of a tear and determine whether the tendon is complete or partially torn [68]. In complete ruptures, the tendon stumps appear thickened and a gap between the proximal and distal portion is visible. The extent of the tendon retraction should be reported.

Stenosing FDS tenovaginitis (trigger finger)

At the level of the MCP joints (flexor zone 1), the A1 annular pulley closely surrounds the FDP and FDS tendons palmarly and binds them to the finger skeleton while maintaining their synovial gliding ability (► Fig. 9a). With overuse being responsible for pulley thickening, the so-called “trigger finger” is the most common entrapment tendinopathy of the hand [6, 13]. Osteo-fibrous tunnel narrowing at the level of the A1 pulley leads to the finger being transiently locked in a flexion position. When trying to straighten the finger, considerable resistance is initially present before abrupt extension occurs in combination with a painful snapping effect. Secretaries, craftsmen, and diabetics are most often affected. In a vicious circle, gliding of the thickened FDS tendon causes further irritation of the annular pulley, which reacts with fibrillation, collagen hypertrophy, and cystic degeneration [13]. On high-resolution MRI, thickening of the A1 pulley with blurring and moderate contrast enhancement is a direct sign of a trigger finger [69]. Localized synovial sheath effusion adjacent to the A1 pulley and a thickened and hyperintense FDS tendon segment are only indirect signs (► Fig. 9b, c).

Disruption of the annular pulleys

The pulley system of the fingers consists of five strong annular ligaments and three loose cruciate ligaments, which are located in the stratum fibrosum of the flexor tendon sheath. The odd-numbered annular pulleys A1, A3, and A5 are in close proximity to the MCP, PIP, and DIP joints, while the even-numbered annular pulleys A2 and A4 are firmly attached to the periosteum of the proximal and middle phalanx shafts. Normal pulleys have a thickness of 0.3 to 0.5 mm and show low signal intensity on all MRI sequences. The main function of the annular pulleys is to bind the tendon sheaths to the bony skeleton with a tendon-bone distance (TBD) of 1 to 2 mm, while stabilizing the tendons during finger flexion [69]. Tears of the annular pulleys result from intense contraction of the flexor tendons, as can be seen in rock climbers adopting the “full crimp position” (maximum flexion at the PIP joint and slight hyperextension at the DIP joint). Tears of the distal A2 pulley occur rather early, followed by involvement of the A3 and A4 pulleys [70]. The ring and middle finger are most commonly affected [67]. If an annular pulley is torn, the “bowstring effect” sets in during finger flexion. Sagittal MR images are best to assess tendon bowstringing and measure TBD, whereas axial images allow for direct visualization of the detached pulley (► Fig. 10a) [69]. A tear must be suspected on MRI if a fluid- or hematoma-filled defect is visible within the annular pulley itself.



► **Fig. 9** Stenosing tenovaginitis at the level of the A1 annular pulley. **a** Normal appearance for comparison. The thin A1 annular pulley (arrow) is sharply delineated. Signs of tenosynovitis are not present (fat-saturated sagittal PD FSE). **b** and **c** Stenosis of the A1 annular pulley, which appears slightly thickened and blurry (arrow). Tenovaginitis and tendinitis (arrowheads) of the flexor tendons are visible distal to the stenosis (**b**: fat-saturated sagittal T1 FSE after intravenous application of gadolinium; **c**: axial T2* GRE).

Direct visualization of a torn annular pulley requires a magnetic field strength of 3 Tesla (even better 7 Tesla) and dedicated receiving coils, particularly for imaging the very fine A3 and A4 pulleys [71, 72]. An indirect sign of a pulley rupture is the enlarged TBD [23]. During finger extension, a TBD >2 mm at the level of the A2 and A4 annular pulleys is suspicious for a tear [73, 74]. MRI in flexion position is more sensitive for assessing a pulley tear. Bowstringing with a TBD of 3 mm during flexion indicates a single rupture of the A2 or A4 annular pulley, while a TBD of 4–5 mm during flexion is suspicious for a multiple pulley rupture including the A3 pulley [74, 75].

Tumors of the tendon sheath

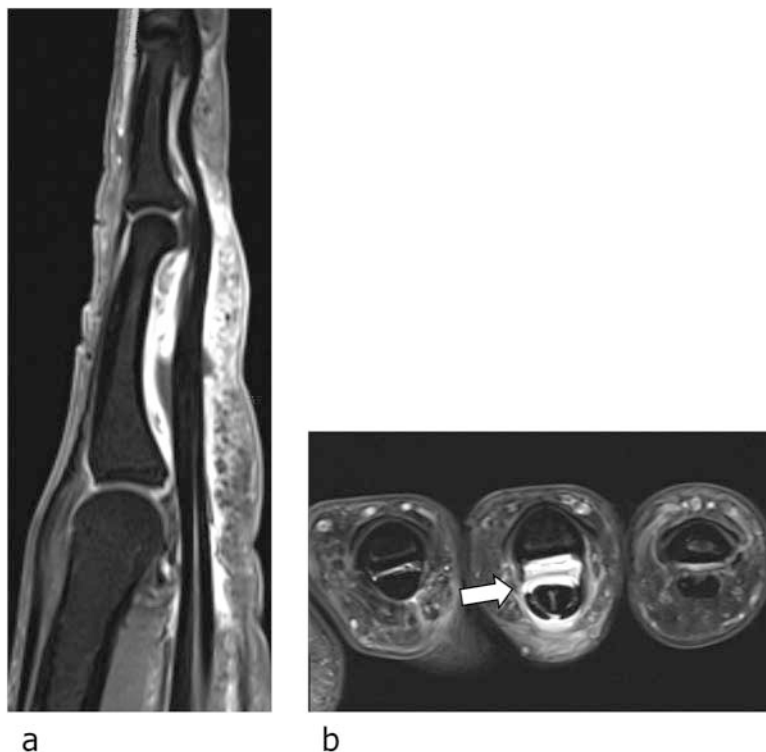
The majority of tenosynovial tumors are benign and have a distinctive appearance on MRI.

Tenosynovial ganglion cyst

Ganglion cysts are the most common masses found on hand imaging. Ganglia are fluid-filled tumors that mostly develop from the joint synovium (articular ganglion cyst), to which they maintain a pedicle. Ganglion cysts arising from the synovial sheath of tendons are rare in comparison (tenosynovial ganglion cyst). Common origins are the flexor tendons of the fingers at the level of the annular pulleys [76, 77]. On MRI, ganglion cysts usually appear as water-filled and thin-walled cavities that often include septa. However, signal intensity can vary depending on the proteinaceous content of a cyst.

Tenosynovial giant cell tumor

Previously known as “pigmented villonodular tumors of the tendon sheath” (PVNTS), giant cell tumors (GCT) of the tendon sheath are firm, indolent masses characterized by aggressive growth and recurrence tendency despite complete excision [78]. In most cases, the origin of GCT is the tenosynovium of the FDP and FDS tendons. The tumors typically grow around the flexor



► **Fig. 10** Tear of the A1 annular pulley. **a** At the level of the proximal phalanx, the tendon-bone distance is 4 mm with a bowstring phenomenon at the level of the A2 annular pulley (fat-saturated sagittal T1 FSE after intravenous application of gadolinium). **b** The rupture site is visible on the radial side of the A2 annular pulley (fat-saturated axial PD FSE).

tendons, often interposed between the FDP tendon and the finger skeleton. Both local and diffuse forms exist. On MRI, tenosynovial GCT appears as a mass adjacent to or surrounding a tendon (► **Fig. 11a, b**). In T2-weighted FSE sequences, and especially in T2*-GRE, low signal intensity and blooming artifacts can be seen if hemosiderin depositions are present within the mass resulting from intra-tumoral micro-hemorrhage [79]. Contrast enhancement is moderate.

Fibroma of the tendon sheath

Fibromas are uncommon proliferative tumors of benign nature. They are located adjacent to tendon sheaths similar to PVNTS [80]. Due to the similar imaging characteristics of both entities, pathological analysis is often needed for differentiation. However, gradient-echo susceptibility artifacts are characteristic for PVNTS but not for fibroma. On MRI, fibromas appear as ovoid, well-encapsulated masses with an isointense signal to skeletal muscle in T1- and T2-weighted sequences [81].

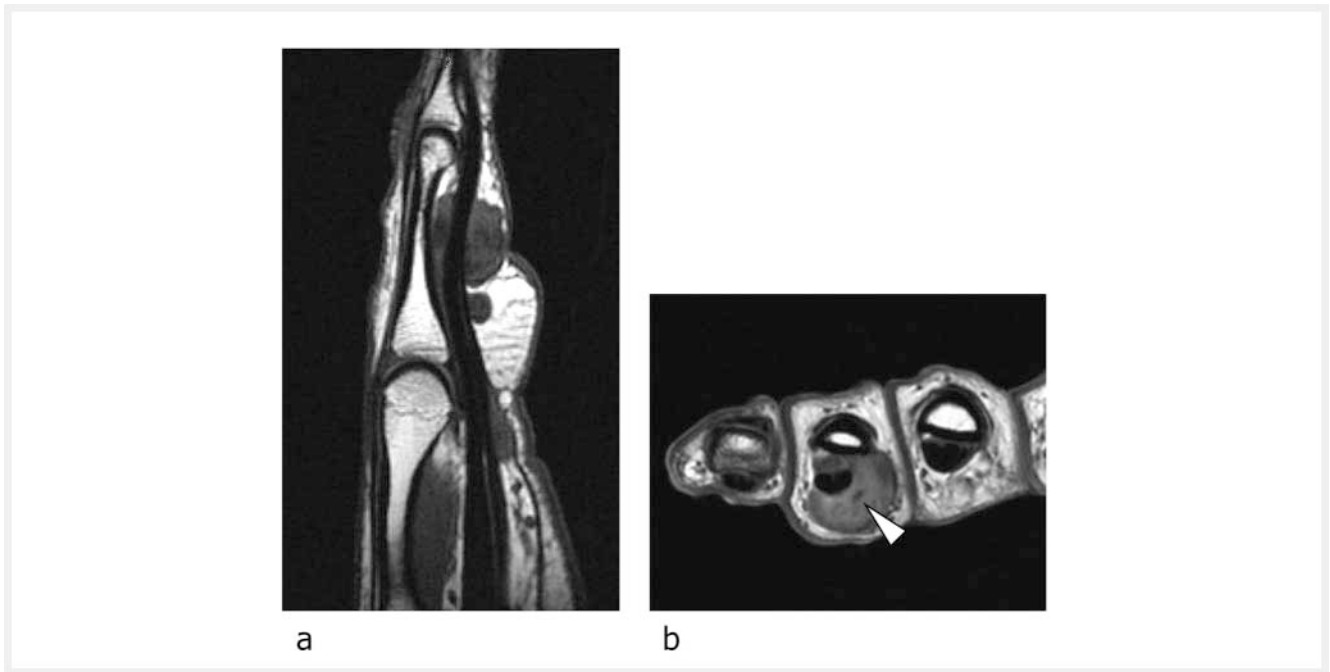
Conclusion

Recapitulating hand anatomy, numerous tendons run closely together in small compartments, sometimes crossing, branching, or uniting. At narrow passages or during curved paths around bony prominences, the tendons are protected by tendon sheaths,

which are reinforced by additional retinacular ligaments near the radiocarpal and finger joints [2]. Among others, injuries, mechanical overload, infections, and systemic or metabolic inflammation may result in different forms of tendinopathy. In clinical practice, tendinopathies are often incorrectly referred to as tendinitis, tenosynovitis, or tenovaginitis, although they do not possess an inflammatory etiology. Since indecisive terminology may lead to wrongful assumptions about a patient's condition, the authors of this review recommend the clear distinction of tendinosis vs. tendinitis and tenovaginitis vs. tenosynovitis. Within this context, it is important to emphasize that stenosing tenovaginitis is a fibrous remodeling process in the outer parietal layer of the tendon [3], whereas proliferating tenosynovitis is caused by inflammatory thickening of the inner visceral layer, often infiltrating the tendon substance [15]. In the pathoanatomical chronicity of stenosis, nodular tendinosis can be both the preexisting cause and the direct result of stenosis of the pulleys or retinacula.

In this review, it became evident that dedicated examination techniques are required for the best MRI results of tendon pathology. These requirements include a scanner with a field strength of 3 Tesla, MRI protocols and sequences adapted to the clinical question, particular slice angulations or 3D sequences if necessary, and finally the targeted use of intravenous contrast agent.

To ascertain the diagnosis of tendon and tendon sheath disorders, the radiologist must be familiar with the complex anatomy of the hand and its common disease patterns. As shown in this



► **Fig. 11** Tenosynovial giant cell tumor of the ring finger. **a** Partially nodular, partially tubular mass alongside the flexor tendons between the metacarpal head and the distal shaft of the proximal phalanx (sagittal T1 FSE). **b** The tumor encases the flexor tendons almost entirely and depicts characteristic hypointense spots (arrowhead; axial T1 FSE).

review, a multitude of tendon disorders exists on both sides of the hand concerning both the extensor and flexor tendons. With the aim of correctly identifying the various forms of tendinopathy on MRI, some general prerequisites need to be fulfilled. First, the examiner must be fully aware of the patient's clinical symptoms. Second, a clearly defined imaging task is required for the MRI examination and should be provided by the referring physician. Third, the radiologist must be sub-specialized in musculoskeletal radiology. He should have profound knowledge of anatomy and pathology of the musculoskeletal system, particularly of disorders in hand surgery for proper diagnosis of hand and finger pathologies. Therefore, musculoskeletal sub-specialization should be implemented and practiced in the daily clinical routine, which is already the case in many European and Anglo-American countries [82, 83]. The authors strongly advocate for a similar approach in Germany in the near future.

Conflict of Interest

The authors declare that they have no conflict of interest.

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