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Tendon injuries of the foot and ankle in athletes

Summary

Sport is a rising lifestyle and subsequent injuries related to sport are more commonly presented to the clinician. In this context tendon injuries of the foot and ankle are often oligo-symptomatic and may be misdiagnosed. Chronic pain and disability can be the long term result. In contrast patients have high demands to return back to their old level of activity after injury. Restoring work ability only is frequently judged as unsuccessful.

The following review focuses on sport related injuries of the anterior and posterior tibial tendons, the peroneal tendons, the extensor and flexor hallucis and digitorum longus tendons. On the one hand the typical clinical presentations of each specific tendon injury are presented with their corresponding diagnostic and therapeutic options, on the other hand the most sport at risk is pointed out and explanations for its disposition are tried to give.

Key words:

Tendon, injury, ankle, sport

Zusammenfassung

Mit zunehmender sportlicher Aktivität der Bevölkerung steigt auch die dem praktizierenden Arzt präsentierte Anzahl von Sportverletzungen. In diesem Zusammenhang sind Verletzungen der Sehnen am Sprunggelenk anfangs oft oligo-symptomatisch und werden leicht unterschätzt. Chronische Schmerzen bis hin zur Invalidität können Spätfolgen sein. Im Gegensatz dazu steht die hohe Erwartungshaltung der Patienten, nach dem Unfall wieder ihr altes Aktivitätsniveau zu erreichen. Mit dem blossen Wiedererlangen der Arbeitsfähigkeit sind die meisten Patienten nicht zufrieden. Der folgende Übersichtsartikel konzentriert sich auf Sportverletzungen der Tibialis-anterior- und -posterior-Sehne, der Peroneal-Sehnen, der langen Extensor- und Flexorsehnen der Gross- und Kleinzehen. Zum einen werden die typischen klinischen Zeichen dieser Sehnenpathologien mit ihren diagnostischen und therapeutischen Optionen aufgezeigt, zum anderen werden die mit der Verletzung assoziierten Sportarten vorgestellt und es wird versucht, eine Erklärung für ihre Disposition zu geben.

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Introduction

The following review focuses on sport related tendon injuries about the foot and ankle. On the one hand the typical clinical presentations of each specific tendon injury are presented with their corresponding diagnostic and therapeutic options, on the other hand the most sports at risk are pointed out and explanations for their disposition are tried to give.

Tendons get hurt in three different ways:

1. Acute tendon distraction, which exceeds tendon elasticity.
2. Chronic tendon overuse, with repetitive micro trauma and reactive synovial inflammation or tendon degeneration; which can progress to spontaneous tendon rupture.
3. Exogenous trauma with laceration or compression of the tendon.

Tendon injuries have grossly the following clinical presentations:

Painful function – reduced function – no function.

Treatment options are injury dependent:

Overuse conditions usually do well with conservative treatment with local and/or systemic anti-inflammatory medications combined with temporary immobilization and/or a subsequent stretching, proprioceptive and strengthening program. If surgery is indicated objectives can be decompression of tendon due to idiopathic stricture of tendon course, repair of tendon lacerations or tears and reconstruction of functional tendon defect.

Sport related injuries of the peroneal tendons

Pathophysiology and related sports

The peroneal tendons are at special risk during skiing [19, 25, 72, 75] and horse riding [54, 97, 103, 139] but are basically injured during ankle distortion [67, 83, 119, 124, 129, 139]. The major pathologies are associated with chronic destabilization of the peroneal tendon complex [26, 136, 139]. Reasons for peroneal tendon instability are thought to be on one hand congenital sulcus abnormalities and on the other hand posttraumatic laxity of the superior peroneal retinaculum (SPR) [26, 83, 113, 162].

To understand the reasons for a specific athletic disposition a thorough understanding of the function and involved anatomy of the peroneal tendons is crucial.

Function: Voluntary muscle contraction of the peroneal muscles leads to ankle eversion. But in addition the muscles contract involuntarily in passive dorsal extension. During this movement the tendons act as dynamic ankle stabilizers [136].

Anatomy: From proximal to distal the tendons run around the posterolateral aspect of the ankle in a common sheath surrounded by a fibro-osseous channel. This peroneal sulcus is formed laterally and posteriorly by the SPR, anteriorly by the concave shaped posterior aspect of the fibula and medially by the posterior inferior tibiofibular-, the posterior talofibular- and the calcaneofibular ligaments. The SPR originates from the periosteum of the posterolateral rim of the fibula. Medially it blends with the posterolateral ankle ligaments and next to their attachments at the calcaneus.

Congenital disposition to tendon dislocation is shown to be dependent on the shape of the posterior surface of the fibula [26, 83, 113, 162]. Cadaver studies documented in 82% a 2–3 mm deep

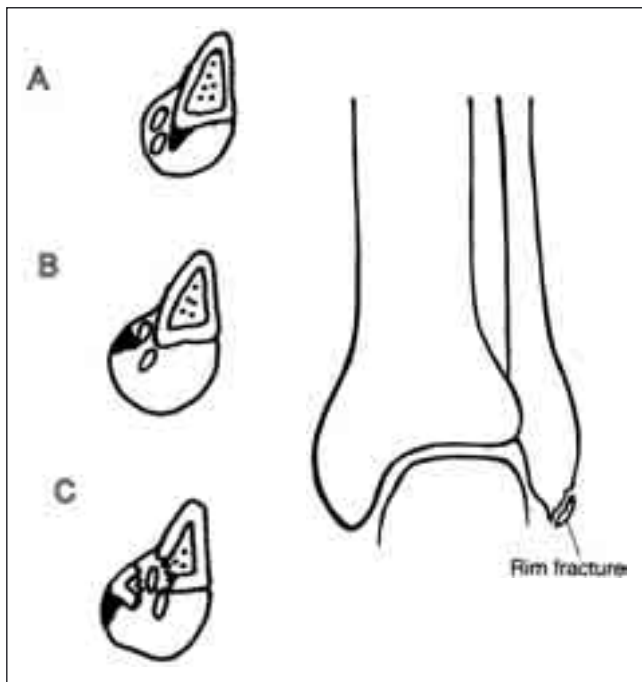


Figure 1: A: Instead of a complete SPR rupture Eckert and Davies [27] found the SPR avulsed but in continuity with the periosteum from the lateral malleolus in 51% of 73 cases (grade I). B: In 33% of cases the fibrous ridge was stripped of as well with the periosteum (grade II). This injury remained them to Bankert lesions of the shoulder. C: In grade III injury (16% of cases) a thin cortical rim was avulsed from the lateral tip of the fibula. This can be seen typically in the mortise view of the ankle.

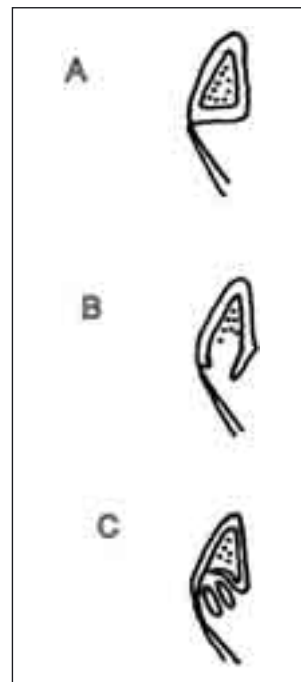


Figure 2: Zoellner and Clancy [162] osteotomized the posterior surface of the fibula at the posterolateral site (A). An osteo-periosteal flap is elevated and left attached at the posteromedial site (B). Cortical bone is removed until the groove is about 10 mm deep. After that the flap is folded into the groove leaving a groove of 3–6 cm length and 6–8 mm depth (C). The fibro-osseous bed of the groove is still preserved to support smooth gliding of the tendons.

groove at the posterior fibula. But in 11% the posterior surface of the fibula was flat and in 7% the surface was even convex. This osseous border of the posterolateral fibula is enhanced by a fibrous ridge to prevent displacement of the tendons. The whole complex is covered by the SPR (*Fig. 1*). Many authors believe in such an anatomical abnormality prerequisite to suffer peroneal tendon displacement [26, 83, 113, 162]. Partial or complete absence of the SPR have been described as well [26, 162].

During ankle sprains which tear of the calcaneofibular ligament, tearing of the SPR can be present as well. This acquired laxity is thought to be responsible for destabilization of tendon complex [26, 136, 139]. The Eckert and Davis grading of SPR injuries [26] seems useful to us (see *Fig. 1*).

Often congenital abnormalities and injuries are combined [26, 83, 113, 162]. Due to the fact that major symptoms like swelling and pain are often associated with ankle sprains, the peroneal injury is not diagnosed. The initiated treatment does address only the ankle sprain [67, 83, 119, 124, 129, 139]. Lateral ankle sprains are mainly treated with a casting or rigid footwear preventing pro-, supination and plantar flexion of the ankle. Full weight bearing is tolerated [2]. Due to the fact that the peroneals contract involuntarily during standing phase and passive dorsiflexion of the ankle, repetitive peroneal tendon dislocation occur despite cast protection of the ankle and prevent healing of the SPR [136]. Chronic tendon subluxation causing tendonitis/tendovaginitis and/or longitudinal split tendon lesions has been reported to be the typical patient presentation after nonoperative therapy of misdiagnosed peroneal complex injury. The ankle itself usually has been found stable. For that reason all traumatic lateral ankle instabilities should be evaluated for peroneal symptoms as well [67, 83, 119, 124, 129, 139].

Most common chronic subluxations of the peroneals occur in skiing [19, 25, 72, 75]. As the ski-boot fixes the foot in dorsal extension and prevents pro-/supination, the peroneals are under constant tension. All axial power of compression is conducted into dorsiflexion of the ankle and provokes subluxation of the peroneal

tendons. If a prerequisite instability of congenital or posttraumatic origin is present, subluxation occurs. Typically the PBT dislocates from its position as it runs usually deep medial to the PLT. Either the PBT dislocates within the fibro-osseous channel causing tendonitis or it luxates over the sharp posterolateral rim of the fibula where it is easily harmed in a longitudinal fashion. All of the well documented cases in literature found the split tendon lesion localized next to the fibro-osseous rim and found the PBT more often involved than the PLT [7, 69, 126, 127, 136].

During horse riding the foot is constantly held in pronation and dorsiflexion. This mild habitual stress to the SPR may be responsible for its laxity. Chronic tenosynovitis as reaction to habitual dislocation of the peroneals has been reported in jockeys [54, 96, 103, 139].

But peroneal pathologies are reported in a variety of additional activities such as soccer, football, basketball etc. [23, 28, 63, 118, 120, 125, 145, 152, 159]. Therefore specific history of trauma is often more helpful to establish diagnosis than relation to a kind of sport.

Peroneal tendonitis if not related to instability may be due to abnormal reasons of tendon course constrictions [18, 129]. These conditions become usually symptomatic in running sports. At locations of abrupt changing of tendon direction as there is the peroneal sulcus, the peroneal trochlea and the lateral edge of the cuboid bone, it flattens and thickens out. At the margin of the cuboid bone the PLT forms in up to a quarter of individuals a sesamoid bone, the os perineum, to withstand the shearing forces to the tendon. These narrow fibro-osseous channels bear the risk of stenosing tenosynovitis especially at fulcrum sites. Only a small thickening of ligaments or tendon will start a vicious circle of compression and reactive tendon edema and synovial fluid production [146]. Congenital prominence of the peroneal trochlea, a descending PBT belly at the sulcus, additional PBT muscle tendons and a peroneus quartus are reasons for development of tendovaginitis or predispositions for development after minor trauma [42, 115, 121, 145, 156].

Complete ruptures of the peroneals are very rare. Only seven cases of sport related PLT ruptures are described in literature [23, 28, 63, 118, 120, 152, 159]. All of these suffered an ankle distortion and felt a snapping at the lateral malleolus during soccer, running and walking sport. In two of these cases the PBT was ruptured as well [152, 159].

Clinical presentation and evaluation

Typically patients present with constant pain at their lateral malleolus after treatment for ankle sprain. Sometimes this pain is associated with subjective feeling of instability and/or with a history of a sudden repetitive snapping at the lateral malleolus. Snapping and feeling of instability occur often during running on uneven grounds. On physical examination localized posterolateral ankle pain and/or swelling are presented without ankle instability. Even complete ruptures produce only minor loss of motion. Active eversion with the ankle held in dorsiflexion should be checked. To evaluate muscle strength and provoke dislocation or snapping mild resistance against eversion should be applied. Eversion is typically weak and impaired in complete or partial disruption of one tendon. But severe tendonitis either due to chronic subluxation or narrowing of fibro-osseous channel can produce the same symptoms. In addition instability may be subtle or within the fibro-osseous channel and can not be visualized during examination [26, 119].

In all cases loaded radiographs of the ankle and foot should be taken to rule out fractures. Fractures of the fibula rim and fractures or proximal dislocations of an os perineum at the ridge of the cuboid should raise the suspicion to peroneal tendon injury [78, 79]. Ultrasound can document complete tendon disruption or unrecognized tendon dislocation in a case of severe ankle swelling [153]. In addition tendonitis with excessive fluid collection within the tendon sheath can be shown. MRI is most useful to visualize the amount of split tendon lesions in axial pictures [69], but can diagnose ruptures and excessive fluid due to tenosynovitis as well [135].

Conservative and surgical treatment

Therapy of peroneal tendon instability:

If the athlete wants to finish his sport season, firm taping can diminish symptoms and the patient may try to continue his sport. But exact diagnosis has to be established and complete ruptures and ankle fractures have to be ruled out. Operation can be postponed into season break.

Most authors believe that conservative treatment for chronic peroneal tendon subluxation/luxation is doomed to failure due to its idiopathic anatomical predisposal [27, 87, 139]. In contrast most of these authors treat acute dislocation of peroneals initially with a short leg cast non-weight bearing for at least 4 weeks. Although conservative treatment was only sufficient in more than 50% of cases they reserved surgery for patients with persistent tendon subluxation.

Many kinds of surgical procedures have been described in literature. But mainly three objectives have been followed: Tendon rerouting procedures, SPR repair with or without augmentation procedures and osseous peroneal sulcus deepening procedures.

Rerouting of the peroneals:

Rerouting of the peroneals under the calcaneofibular ligament (CFL) have been achieved by elevating the CFL with its bony attachment from the calcaneus or fibula, by lateral malleolar osteotomy or by cutting the peroneal tendons. In contrast to disturbance of ligament or tendon integrity reports of cases treated with these procedures have presented good results without further dislocations and return to sport [73, 111, 113, 124].

SPR repair with or without augmentation:

SPR repair for acute and chronic subluxation have been presented by Eckert and Davis who graded SPR injuries (see above) [26].

They recommend suturing the retinaculum to the fibrous ridge (grade I) and the ridge and retinaculum to the lateral malleolus (grade II). Fractures of the fibula rim (grade III) were reduced open and fixed with Kirschner-wires. Three of their 73 patients after suture repair had further dislocations [26].

Augmentation of the SPR was achieved by using a tendon sling from the Achilles tendon [27, 54], slings of the PBT [138], peroneus quartus tendon [92], or periosteum flaps [155]. Postoperative treatment was a short leg cast non-weight bearing for 4–8 weeks. In their patients follow up all authors had mainly good results.

Peroneal sulcus deepening procedures:

Different bone block procedures have been introduced to deepen the peroneal groove. Kelly osteotomized half of the fibula in sagittal direction and mobilized the distal 2–3 cm of the fibula posteriorly 5–6 mm [57]. DuVries modified Kelly's procedure and used a smaller wedge shaped bone block to mobilize it posteriorly and leaves the tip of the fibula with its ligaments unchanged [8]. Subsequent studies using these bone block procedures in 33 patients had no further dislocation in follow up of two years and more. Some had a crepitus of tendons without symptoms, thought to be caused by the sharp inferior margin of the osteotomy [83, 84, 91].

Our preferred procedure was invented by Zoellner and Clancy [162]. With this method the fibro-osseous bed of the groove was still preserved to support smooth gliding of the tendons (see Fig. 2).

Reports in literature with three studies comprising 17 patients have shown good results and no further dislocation. The advantage of this procedure is a reduced time in short leg casting after surgery because it lacks osteosynthesis. In addition after surgery the construct is stable immediately. The tendons are pushing the osteo-periosteal flap into its bed ensuring healing and preventing dislocation [5, 67, 141].

Surgery for tendon dislocation within the sulcus:

We found three cases in literature where persistent pain due to PBT luxation within the peroneal sulcus causing a peroneal tendonitis was treated surgically [43, 85]. In the first case stability was achieved successfully by suturing a retinaculum sling over the PBT and under the PLT [85]. In the second case such sling procedure failed and success was salvaged by tenodesis of the PBT to the PLT [43]. The third case was treated with a rerouting maneuver under the CFL [43]. All went back to their old level of activity after surgery and symptoms resolved.

Therapy of peroneal tendon ruptures:

Even though success with conservative treatment of total PLT ruptures is mentioned in literature [106, 116] most authors agree that complete rupture is best treated with surgery [1, 18, 28, 108, 118, 154]. Success has been uniformly reported in end-to-end suture of the tendons [1], in a suture of a fracture of the os perineum [109], debridement of a fracture of the os peroneum and tendon repair [78, 79, 108], tenodesis of the PLT to the intact PBT [18, 28] and suture of the ruptured stump to the cuboid bone [118]. Non weight-bearing short leg cast for about 6 weeks followed surgery.

Longitudinal split lesions of the peroneal tendons occur mainly in the PBT and are treated depending on the cross-sectional amount of defect after debridement. Grading of split tendon lesions of the PBT was invented by Krause and Brodsky [69]. Central debridement and buried suturing of the surrounding tendon fibers is performed when 50% or more is remaining (grade I). If more than 50% (grade II) have to be debrided, tenodesis with suturing the proximal and distal ends to the PLT is advised [69]. All results of performed case studies were invariable good. All stated that the described procedure has to be combined with a stabilization procedure if dislocation was a concern [13, 18, 69, 78, 126, 146, 149].

Therapy of peroneal tendonitis:

Fore simple tendonitis nonoperative treatment with anti-inflammatory medication and a decrease of activity is most often suc-

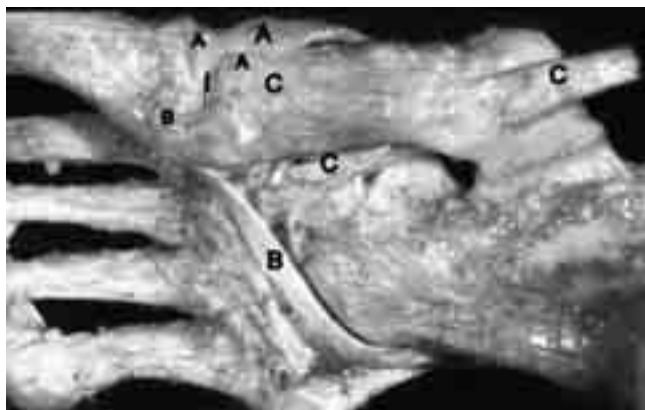


Figure 3: Normal anatomy of tendon insertion at the plantar pedis: (I) marks the site of the first metatarso-cuneiform joint. The tibial tendon inserts at the first metatarso-cuneiform joint (A). The long peroneal tendon inserts as well at the first metatarso-cuneiform joint (B). The posterior tibial tendon inserts at the first and second metatarso-cuneiform joint (C).

cessful [116, 129]. After decrease of symptoms combination with a stretching and proprioceptive training is recommended. Depending on the severity of symptoms immobilization up to 4 weeks in a short leg cast without weight bearing might be indicated. If symptoms persist surgery is performed [146, 149]. Tight tendon sheathes are released and left open, prominent peroneal trochleas are remodeled [116, 146], synovial tissue and abnormal muscles such as peroneus quartus, or a descending muscle belly or even rarely additional tendons of the PBT are excised [42, 121, 156]. These cases of decompression mentioned in literature had good results and patients gained activity after rehabilitation.

Sport related injuries of the anterior tibial tendon (ATT)

Pathophysiology and related sports

Sport related injuries to the anterior tibial tendon are rare. More often tendon injuries are related to systemic inflammatory diseases and partial or total ruptures due to degeneration [56, 88, 90, 94, 98, 105]. The tibialis anterior tendon seems to be at special risk in snow sports, namely ice hockey and skiing [14, 49, 56, 133, 140]. There are three case reports in literature of closed complete tendon rupture in skiing. Two of these were acute distraction due to fall and plantar flexion [14, 140], one is due to overuse during skating technique in cross-country skiing in an untrained individual [56]. Rigid footwear, such as ski boots, has shown to produce chronic inflammation of the anterior tibial tendon due to irritation of the ventral aspect of the tibia [12, 53].

The literature reported about seven ice hockey players in which the anterior tibial tendon was lacerated just over the top of the skate boot [49, 133]. The skate-blade had cut the tendon by direct laceration. Due to its exposed location at the ventral edge of the tibia, the tendon seems to be at special risk for acute exogenous trauma. In each case the boot tongue was worn downwards and not upwards to protect the ventral tibia.

Clinical presentation and evaluation

Complete discontinuity of the tendon is easily diagnosed by the inability to walk on heels. The lack of dorsal extension combined with a palpable mass at the ventral aspect of the tibia, mostly just around the extensor retinaculum, establishes the diagnosis. Underneath the extensor retinaculum the anterior tibial tendon changes its direction and is disposed to friction overuse. In addition vascularization of the tendon is minimal at this pulley site and gives an explanation why ruptures are usually located next to the extensor retinaculum [37, 110]. The fact that pain and hematoma is seldom

associated, often loss of function is the only symptom of complete closed tendon disruption. Diagnosis may be delayed due to extensive neurological examinations [88, 90, 98]. To rule out peroneal nerve palsy electromyography may help [56].

Depending on time elapsed between rupture and patient presentation, the proximal tendon end may adhere to surrounded tissue, mimicking decreased function due to muscle weakness not tendon rupture. In such uncertain cases with preserved but weak or painful function ultrasound [10] and better MRI [140] can verify total and partial rupture or tenosynovitis with peritendinous fluid collection [61, 62]. However, sport related injuries of the anterior tibial tendon seem to be predominantly acute function loss combined with acute trauma. The chronic progressive illness as known from patients with inflammatory diseases, with first painful function, then weakness and finally uneventful rupture is not due to sport but may be activity related [56, 88, 90, 94, 98, 105].

In cases of skin lacerations at the ventral apex of the tibia, it is mandatory to check function of the tibialis anterior tendon and other extensors [133]. In doubt the surgeon should explore the wound. Even small skin laceration can despise total discontinuity of tibial tendon as reported in two of the seven ice hockey players mentioned above [133].

Conservative and surgical treatment

Open tendon lacerations are best treated with nonabsorbable sutures. We preferred a modified Kessler [58] or Bunnell [15] criss-cross suture with a strong No. 2 core suture followed by fine adaptation with a 3-0 running suture. Surgery is followed by immobilization in a removable soft cast between 2 and 6 weeks postoperative depending on the patients compliance and strength of tendon repair. Normally after 2 weeks postoperative we go over to a kind of «Kleinert-Therapy» [65]: The soft cast is removed and an «Albrecht-Quengel-Orthotic» is applied for the rest of the 6 postoperative weeks. This orthotic allows active plantar flexion to neutral and extends passively dorsal with a quill-feather. This provides early tendon movement without force and decreases adhesions to the tendon repair. After this period increasing active mobilization training is started with proprioceptive elements. Full sportive activity is usually reached after 3 months.

The closed ruptures irrespective to sport are best treated surgically as well [31, 38, 56, 90, 94]. Although there are nonoperative regimes which have reported success by casting for 6 weeks [17, 98], there is no doubt that nonoperative treatment leads to decreased dorsiflexion of the ankle. Falls related to a resulting drop-foot can cause major trauma, not only in elderly people. Destabilization of the midfoot and progression to a pronated flatfoot with heel-cord contracture has been observed after nonoperative treatment [31, 38, 56, 81, 90, 94, 105]. Therefore we recommend surgery in all patients if there is no absolute contraindication.

In general reconstructive techniques to bridge defects of the tibial tendon can be achieved by autologous tendons such as the free semitendinosus and the transferred extensor hallucis longus- or peroneus longus tendon [31, 81, 93, 105, 151]. We have made good experiences with these mentioned techniques. But we prefer the peroneus longus tendon as it inserts next to the anterior tibial tendon at the plantar aspect of the first and second tarsometatarsal joints. In addition the existing pronated flatfoot is corrected by transferring the pronative force of the peroneus longus muscle into a supinative force of the anterior tibial tendon. Postoperative management follows as mentioned above (Fig. 3).

In a rare case of tendonitis-tendovaginitis of the anterior tibial tendon footwear should be questioned [53]. Often only changing the irritating footwear relieves the symptoms. In recalcitrant tendonitis immobilization in a short leg cast up to 6 weeks and local and/or systemic anti-inflammatory medications are advised and sufficient. Steroid injection for any kind of tendovaginitis is not recommended as it may lead to tendon rupture [10, 56, 151].

In the literature treatment for avulsion fractures of the anterior tibial tendon from the navicular bone is described [94, 129]. We do not believe in its existence due to the fact that the anterior tibial

tendon inserts plantar around the first and second tarsometatarsal joint not at the navicular bone (*Fig. 3*).

Sport related injuries of the extensor hallucis longus tendon (EHLT)

Pathophysiology and related sports

The extensor hallucis longus tendon (EHLT) is seldom injured during sports. Complete closed ruptures are only seven times mentioned in literature. Two tendons ruptured during soccer [70, 89], one in Taekwondo [107], one during walking in hiking boots [99] and one while leaning back in downhill skiing [132]. None of these athletes had a diagnosed chronic disease or took any medications. On one hand the mechanism of injury seems to be chronic overuse, on the other hand the tendon ruptures due to axial and plantarflexion trauma to the great toe.

The remaining two EHLT ruptures reported in literature were post hallux surgery [112, 161]. One patient has received corticoid injections to his painful rigid first metatarsophalangeal joint [112].

A far more common reason for EHLT injury is laceration due to falling sharp objects or high velocity injuries to the foot [9, 30, 38, 76, 133, 134, 157]. A higher incidence of forefoot extensor lacerations were found to be climate related. In warmer regions with bare foot walking the EHLT is easily harmed due to its superficial course at the dorsum of the foot [157]. But sport related we found only two cases [133]. In both cases the EHLT of an ice hockey player was cut by direct laceration of the blade of an ice hockey boot.

The entity of tendonitis is reported in association with marathon runners [12]. Repetitive ankle extension was thought to be responsible for unspecific tendonitis of the foot- and toe extensors [53].

Clinical presentation and evaluation

In closed ruptures pain was reported to be of short duration and often even does not lead to an interruption of the causative sport. The diagnosis of complete rupture usually can be established on exam. Inability to palpate the tendon at the dorsum of the foot during dorsiflexion coupled with weakness in dorsiflexion is diagnostic for disruption. To choose the right place for incision ultrasonography may help to find the tendon ends [153].

If a laceration at the dorsum of the foot is presented, loss of function combined with the location of skin wound should be suspicious for tendon injury [133].

The diagnosis of tendonitis can be made with painful extension against resistance and local pain elicited with palpating the tendon course. Continuity and excessive fluid can be visualized in ultrasonography or MRI if dorsiflexion is found weak [130, 153].

Conservative and surgical treatment

Despite the fact that nearly all closed or open disruptions have been sutured or reconstructed with good results [9, 30, 38, 55, 76, 128, 133, 134, 157] there are authors who believe in successful conservative treatment [30, 38, 157]. Floyd and associates reported about one out of 13 patients with EHLT lacerations who regained active dorsiflexion with nonoperative treatment [30]. Griffiths reported about six cases of EHLT lacerations in which all regained function but one without suturing [38]. He proposed that repair of tendon is not necessary due to its well known tendency of spontaneous self repair after tenotomy. Wicks and colleagues repaired eleven EHLT lacerations in children and graded two results fair because of painful scarring and stiffness of the MTP joints [157]. Thus they proposed conservative treatment since walking remains full mobility of the great toe without major functional disability.

All aforementioned sport related cases underwent surgery and went back to their previous level of activity. Open lacerations should encompass primary tendon suture within routine wound

exploration. Tendon repair in a Bunnell [15] or Kessler [58] type stitch with nonabsorbable 2-0 or 3-0 core-suture has been reported to be successful. Follow up regimes encompasses casting in dorsiflexion or neutral for 6 weeks. To reduce resistance to great toe dorsiflexion, the dorsum of the foot should remain free during casting. If the surgeon wants to limit postoperative scarring and toe stiffness dynamic splinting is advised [134, 55, 128].

Degenerative ruptures due to overuse or chronic inflammatory disease may present with a significant zone of defect [107, 161]. In such cases free semitendinosus tendon graft [107] or local tendon transfer of the second toe extensor [74], tendon of the extensor hallucis brevis or the peroneus tertius tendon [89] have been reported to produce good results. In addition a fascia lata allograft was successfully used to bridge a defect in a severed EHLT [161]. Follow up treatment as mentioned above.

Sport related injuries of the extensor digitorum longus tendon (EDLT)

Pathophysiology and related sports

Injuries to the extensor digitorum longus tendon encompasses dislocation due to rupture of the extensor retinaculum [3], tendonitis due to chronic compression of a tight boot [12] and laceration due to falling sharp objects [30, 76, 157]. Even though many institutions have to deal with tendon injuries to the forefoot every year the literature has no major reports. Anzel and colleagues found 1104 cases of tendon injuries treated at their hospital, 146 occurred at the lower leg and 15 of these involved the extensor tendons of the toes [4]. Due to the fact that injuries to these tendons have mostly no significant contribution to the level of patients activity treatment is difficult to evaluate. That leads to the circumstance that reporting results of treatment is not attractive.

We found only two reports of EDLT injuries in literature related to sport. Akathar and Levine reported about a healthy 57 years old man who turned his ankle into inversion during squash as he felt a snap and sharp pain at the anterior aspect of his ankle. During surgery rupture of the extensor retinaculum was revealed which lead to dislocation of the EDLT. No history of systemic diseases or medications could be found [3].

The other case was the aforementioned ice hockey player with a combined laceration of the EDLT with the anterior tibial- and extensor hallucis longus tendon [133].

Clinical presentation and evaluation

Lacerations of the EDLTs via the dorsum pedis often lack significant loss of function [76, 133, 157]. Compensation due to the extensor digitorum brevis may occur. In addition the tendinous interconnections connecting the four EDLTs often compensate laceration of a single one. Diagnosis often is first established during wound exploration [133].

Conservative and surgical treatment

Even though no adverse loss of function is lost with conservative treatment of an asymptomatic tendon laceration, all open wounds should be explored [133]. Exploration should encompass restoration of anatomy in terms of tendons should be sutured. A non-absorbable 2-0 or 3-0 core and running suture is sufficient. Early dynamic splinting or immobilization may follow surgery as patient compliance or strength of suture dictates.

Sport related injuries of the posterior tibial tendon (PTT)

Pathophysiology and related sports

Posterior tibial tendon dysfunction (PTTD) is the major cause of acquired flatfoot deformity in an adult [33]. Hallmarks of the



Figure 4: Clinical signs for posterior tibial tendon dysfunction: (A) The first metatarsal rise sign: With passive external rotation of the tibia the first metatarsal bone rises from the ground. (B) Double-heel rise test/tip-toe test without the physiological varisation of the hindfoot. (C) Too-many-toes sign showing the excessive forefoot abduction [44, 45, 51, 100].

progressive deformity are valgus and pronation of the hindfoot, abduction and supination of the forefoot and flattening of the longitudinal arch [44, 52]. Being initially correctable and flexible, these deformities eventually can become rigid and cause even some ankle joint mal-alignment and fibular impingement by the calcaneus [64]. The clinically accepted four-stage classification of PTTD was introduced by Johnson and modified by Myerson. The four stages are: Stage I: Tenosynovitis with minimal deformity, Stage II: PTT insufficiency with flexible flatfoot deformity, Stage III: PTT insufficiency with rigid flatfoot deformity, Stage IV: Stage III with valgus talar tilt in ankle mortise [51, 100].

Based on the underlying pathology posterior tibial tendon lesions can be classified in tenosynovitis/tendinosis, partial or total ruptures, and anterior sub/luxation. However, the most common entity is the tenosynovitis, or stage I of the PTTD. A correlation between this early stage of the PTT dysfunction and middle-aged females has been observed as well as in patients with hypertension, obesity, rheumatoid arthritis, and sero-negative inflammatory arthropathies [47]. In younger patients tenosynovitis is less common and has been reported in only 3% of active runners [77]. The rupture of the PTT is typically missed and often diagnosed unfortunately late. An average delay in diagnosis of 43 months (range, 1 month to 12 years) has been described [80]. The PTT rupture can be partial or complete, whereas the chronic degenerative rupture is more common than the acutely caused or traumatic type [80, 160]. After PTT rupture patients show clinical signs of progressive PTTD and a remarkable loss of muscular function, combined with atrophy, fatty degeneration, and muscular fibrosis [148]. Epidemiologically, posterior tibial tendon rupture occurs typically in middle-aged and low sports active people, most commonly women. The majority of patients do, however, have a history of previous tenosynovitis. Nevertheless these entities may be also encountered in young and athletic people. In this regard Woods and Leach reported on six athletes suffering from PTT rupture [160]. Unexpected pronation trauma followed by an abrupt and painful loss of force represents the typical mechanism of injury (twisting the foot, stepping in a hole) [148]. The majority of patients have, however, previous to the PTT rupture often a history of antecedent tenosynovitis. Isolated anterior PTT dislocation is the rarest PTT lesion and has been documented in the literature only in terms of case reports, this especially related to ballet and running sports [11, 59, 71]. The typical mechanism of injury in these reports was here the combination of extreme dorsiflexion and inversion of the hindfoot. In the majority of the cases the flexor retinaculum was torn and had to be sutured at time of surgery.

Clinical presentation and evaluation

Posterior tibial tendon disruptions lead to a progressive flatfoot deformity: pes planovalgus et abductus et supinatus, at the beginning flexible, later on rigid and fixed. Subjectively, patients with

PTTD complain in an early stage about pain, tenderness and swelling on the posteromedial aspect of the foot and ankle, in stage IV typically on the lateral aspect. Athletic individuals claim disability and prolonged time away from sports being unable to develop enough push-off power and foot control [29, 44]. Clinically patients exhibit an asymmetrical planovalgus deformity of the affected foot, with excessive hindfoot valgus, abduction of the forefoot, loss of height in the arch, and pronation of the foot. Further evidence of posterior tibial tendon dysfunction is present when weakness of supination is encountered on manual testing of the muscle (inversion strength of the hindfoot by plantar flexed ankle) [148]. A few reliable clinical PTTD tests have been described in the literature (see Fig. 4).

Weight-bearing antero-posterior and lateral radiological assessment of the foot and ankle should be obtained in order to reveal the hindfoot alignment and evaluate any possible arthritis or subluxation at the ankle, subtalar, Chopart or midfoot level [51]. Significant angular deformities can be measured (see Fig. 5).

Further imaging as computed tomography and magnet resonance imaging are rarely necessary. The CT may be of significant help to exclude a possible tarsal coalition. The MRI is not required to establish the diagnosis of a PTTD. But since several studies have shown and confirmed the high frequency of the co-lesion of the spring ligament in PTT rupture cases, MRI may play an important role for the preoperative evaluation of the medial ligaments and therefore the surgery decision making [6, 35].

Conservative and surgical treatment

In PTTD stage I nonoperative treatment is primarily indicated. The combination of immobilization for 2–3 weeks by a walking boot, stabilizing shoe or cast together with a non-steroidal anti-inflammatory drugs (NSAIDs) therapy represent the common standard [44, 158]. Local steroid infiltrations have shown counterproductive results and should therefore be avoided. The series by Trevino et al. reported partial and completely PTT ruptures after steroid injections for the treatment of PT tenosynovitis [146]. In cases of failed nonsurgical management of PT tenosynovitis or in cases with stenosis or signs of intra-tendon degeneration surgical treatment with open synovectomy, tendon release and debridement provides good to excellent results [44, 86]. As alternative surgery technique, newest studies report pain and symptoms relief at 1-year follow-up after two-portal endoscopy for the treatment of chronic tenosynovitis [150]. In stage II–III of PTTD surgical reconstruction of the degenerated, elongated, total or complete rupture tendon is in the majority of the cases inevitable. Several reconstructive techniques have been described: a) end-to-end suture of the tendon, b) reinsertion, shortening or advancement of the tendon on the navicular bone, c) side-to-side reconstruction by a supporting tendon transfer. The flexor digitorum longus (FDL) is most often used in tendon transfers because of its size, strength, and anatomical nearness [46, 51, 160]. But other tendon transfer

have been described in the literature in extenso as well: Flexor hallucis longus (FHL) transfer, tibialis anterior transfer (Cobb procedure), peroneus longus transfer, and others [24, 35]. Since PTTD is often combined with attenuation in the spring ligament complex or even in the deltoid ligament intraoperative evaluation and additional repair of the medial ligament structures should be performed [24, 46]. However, it has been widely recognized that reconstruction of the posterior tibial tendon and ligaments provides resolution of medial pain but does not provide correction of the deformity, which often progresses and becomes painful [33, 100]. In stage II fusions such as subtalar, talonavicular, double, or triple arthrodesis correct deformity more than sufficient, but it is at the cost of uncomfortable stiffness and risk of arthritis in neighboring joints [102]. For this reason, in stage II/III tendon reconstruction procedures should be combined with bony osteotomies in an attempt to give long-lasting correction of both pain and deformity. Both, lateral column lengthening osteotomy [46] and medial displacement calcaneal osteotomy [100] were shown to have a significant role in management by restoring normal biomechanics, allowing the reconstructed and transferred tendons to function successfully. Finally, in end stage III/IV, with a rigid and severe flatfoot deformity and/or secondary degenerative changes in the subtalar, talonavicular and Chopart joint, only selective arthrodesis (as subtalar, talonavicular, triple arthrodesis) have shown to provide the sufficiently stable and plantigrade correction of the hindfoot [44, 51, 100].

Sport related injuries of the flexor hallucis longus tendon (FHLT)

Pathophysiology and related sports

The flexor hallucis longus tendon is at risk in ballet dancing [22, 39–41, 122] and running sports [20, 36, 117]. We found only seven cases of closed disruption of the FHLT described in literature [20, 48, 50, 68, 114, 117, 143]. All ruptured under the plantar pedis during running sports like marathon, soccer or walking. If one looks carefully at the locations of rupture it is obvious that the tendon ruptured at its fulcrum sites as it runs around the sustentaculum tali and the inter-sesamoid groove. This leads to hypothesis of chronic overuse as mechanism of injury for total rupture.

Exogenous trauma with laceration of the tendon occurred most often in barefoot runners [30, 32]. Usually they step on a sharp object such as glass. As the tendon runs superficial to the plantar pedis especially at the metatarsophalangeal joint it bears most of the body weight during ground contact.

More often than complete rupture of the tendon is a chronic tenosynovitis of the FHL [39–41, 60, 104, 131]. Although reported as well in runners [20, 117, 142] and tennis players [36, 95, 144] the main sport to memorize with chronic FHL tenosynovitis is classical ballet dancing [22, 34, 39–41, 60, 123]. Reason for that is thought to be the main stabilization function of the FHLT during en-pointe dancing. In this situation the FHLT serves as the Achilles tendon of the ankle [39–41].

Clinical presentation and evaluation

Usually acute rupture is felt by the athlete with a sudden loss of great toe push off [20, 68]. This symptom is not mandatorily associated with pain. On clinical examination the patient will be able to bend the metatarsophalangeal joint but not the interphalangeal joint. A clear decrease of power in plantarflexion of the great toe can be felt compared to the uninjured site. If diagnosis is still uncertain ultrasound or MRI [16] can visualize a partial or complete rupture. Dynamic pedobarography is another option to visualize diminished great toe push off. However, diagnosis normally can be made by history and examination alone.

Tenosynovitis of the FHL presents usually with a sudden onset of pain during or after ballet dancing at the posterior medial aspect of the ankle [40, 41, 123]. Typically the locus of swelling and

tenderness lies behind the medial malleolus. The exact source of symptoms can be differentiated best from adjacent tendon injuries by sequential testing [41]. The «Hamilton maneuver» [41] tests the FHL and the FDL with the foot in en-pointe position and flexion of the hallux against mild resistance. Simultaneously posteromedial palpation at the ankle will differentiate the painful tendon excursion occasionally combined with friction rub. In contrast to

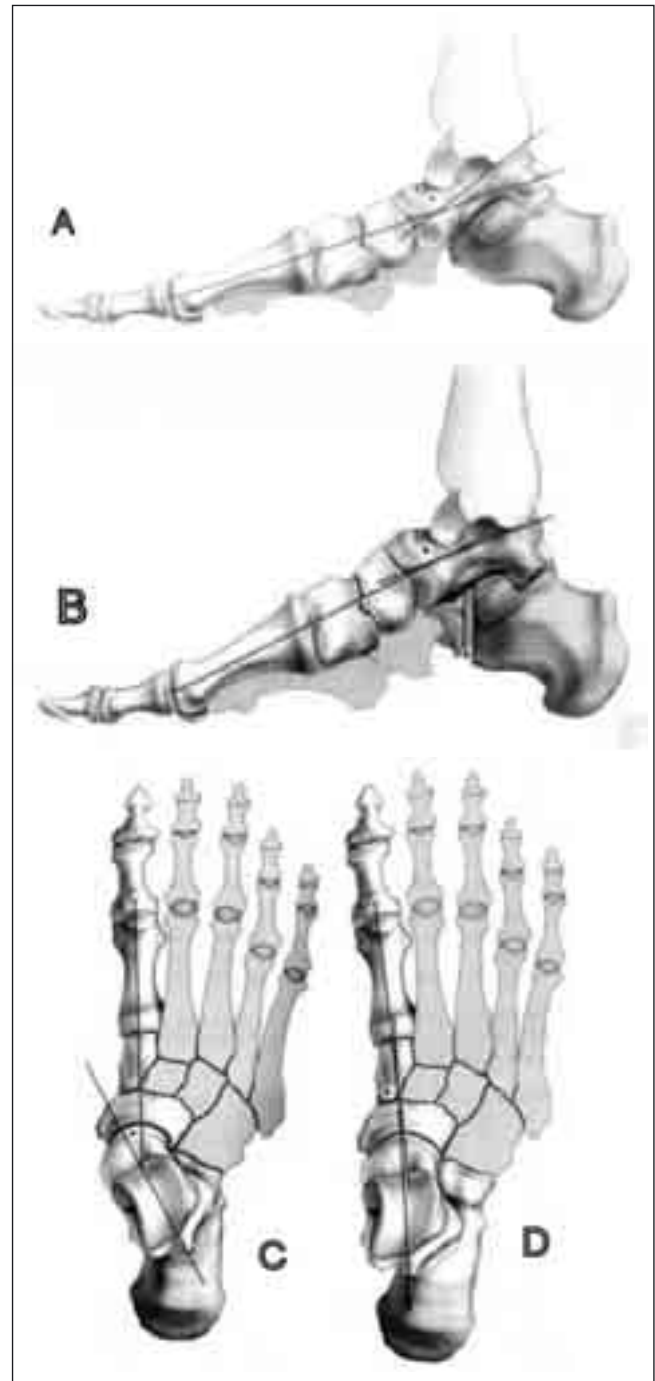


Figure 5: Flatfoot angles: On the lateral radiograph, the lateral talometatarsal angle (angle between talar head and axis of the neck and the first metatarsal axis), and on the antero-posterior radiograph, the dorsoplantar talometatarsal angle (angle between talar head and axis of the neck and the first metatarsal axis) may be measured. The lateral talometatarsal angle has been described as 0 degrees in normal subjects (B), under 15 degrees in mild flatfoot deformities, and over 15 degrees in severe flatfoot deformities (A). Normal a.p. view is shown in picture (D), where a severe deformity can be judged by abduction of the forefoot and uncovered talar head (C) [14, 80].

tendonitis of the posterior tibial tendon the pain is not associated with painful supination against mild resistance.

To distinguish FHL symptoms from posterolateral impingement, forceful passive plantar flexion should not produce posterolateral pain [21, 41, 82]. In addition standard X-rays should be obtained to rule out fracture of the lateral talar process as well as an impingement due to bone spurs or an os trigonum [40, 82]. The latter can be defined best by a lateral view in en-pointe position.

Achilles tendonitis can be differentiated by careful examination of the locus dolenti. With Achilles tendonitis the pain lies at the tuber calcanei and the tendon itself and not over the fibro-osseous tunnel of the FHL. MRI has no value in diagnosis of FHL tenosynovitis as the tendon sheath often communicates with the ankle joint space and therefore is associated with more or less fluid collection around the tendon which is misdiagnosed as reactive tenosynovitis [16]. But MRI can rule out osteochondritic lesions of the talus which may as well cause posteromedial ankle pain.

FHL tendonitis can be associated with locking and triggering of the tendon in total plantar flexion [39, 95, 101, 123, 137]. As the tendon dives under the lancinate ligament (retinaculum flexorum) it runs in a firm fibro-osseous tunnel. As known from the trigger finger, the tendon overuse mainly occurs at fulcrum sites. Chronic overuse leads to micro trauma and scarring with exuberant scar tissue formation. This mass is then captured proximal to the lancinate ligament during plantar flexion and can be felt in examination. This capture mechanism can be reproduced during examination by active plantar flexion of the great toe. If locked in, the patient is unable to extend the big toe. But passive extension of the interphalangeal joint produces a painless snap with subsequent free motion of the great toe. Patients normally can bend the toe easily in neutral position but are unable to bend in en-pointe position [39, 95, 101, 104, 123, 137].

Conservative and surgical treatment

Open lacerations of the FHL should be sutured the same way as flexor tendons of the hand [20, 30, 32, 50, 68, 114, 117, 143]. 2-0 core stitch and a running 4-0 nonabsorbable suture have been used with success. Surgery is followed by casting and weight bearing as tolerated for 6 weeks [32]. The FHL tendon is mostly harmed within its distal 12 cm as it runs superficial under the skin and plantar fascia [30, 32]. More proximally at midfoot level, next to the knot of Henry, it is covered by the overlying FDL tendon laterally and medially by the abductor hallucis muscle belly. Usually the proximal end of the lacerated tendon is easily found because it does not retract. The «vide supra» is a fibrous slip which tethers the FHL to the flexor digitorum longus tendons (FDLT) about 12 cm proximal to its insertion at the great toe and prevents proximal end retraction of more than 2 cm. The adverse effect of nonsurgical treatment with loss of push and observed gait imbalance led to the opinion that all ruptures of the FHL should be repaired. There was no report of successful nonoperative treatment for FHL rupture in literature. However, after suturing the interphalangeal joint mobility was often decreased. But the crucial function of the great toe during gait, which is push off and stabilization pressure up to neutral joint position, was restored [30, 32].

In cases of lost proximal tendon end during repair, wide exploration is not recommended [32]. The crucial function can be restored by suturing the proximal tendon end of the FHL to the underlying flexor hallucis brevis muscle.

Treatment of FHL tendonitis depends on the duration and the stage of disease. Acute tendonitis without triggering is treated with anti-inflammatory medication and tendon rest. Usually in combination with a short sport break or dancing without en-pointe training will cure symptoms within days to weeks [34, 39–41, 122]. In addition instruction to keep bodyweight directly over the foot during dancing, avoiding hard floors and using rigid well fitting shoes will reduce additional strain to the tendons and support toe stability. If symptoms do not resolve totally within 3 months or even increase, a short immobilization of the foot in a soft cast is recommended [40].

If triggering of the FHL is present surgery is indicated earlier [66, 123, 147]. After initial nonoperative treatment for up to 6 weeks persistent tendon triggering should be explored. Primary tendon sheath release is performed but should be combined with release of the lancinate ligament in tight cases [66]. Usually the source of triggering is a bumpy mass which contains degenerative scar tissue or calcified nodules. These enlargements should be opened and debrided. If there is a longitudinal tear with core fiber rupture, the scar tissue should be debrided and the tendon sutured with buried stitches. No closure of the tendon sheath or lancinate ligament is recommended to ensure free gliding of the tendon [66, 123, 128, 147]. Surgery is followed by mild range of motion training for 14 days until wound healing is completed. Subsequent unprotected gait and return to en-pointe dancing after 6–8 weeks is achieved [66, 128].

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