



# Hindfoot Tendinopathies

# 69

Pim A. D. van Dijk

## 69.1 Introduction

Tendinopathies are common hindfoot injuries that can result in high morbidity for the patient. While the exact incidence in the hindfoot remains unknown, around 30% of all musculoskeletal consultations in the general practice are associated with this disabling condition [1]. In the acute phase, hindfoot tendinopathies are often missed or mistaken for an ankle sprain [2]. On the other hand, overuse or chronic injuries are difficult to diagnose due to the slow onset of symptoms. As tendinopathies can have a strong impact on the patient's quality of life, timely and accurate diagnosis is important for effective management and to avoid socioeconomic burden [3].

In recent times, our understanding of tendon injuries has evolved significantly and today tendinopathy is considered a chronic, degenerative

pathology. The cause of tendinopathies is mostly multifactorial and can be an expression of systemic or general musculoskeletal problems. A combined thorough patient history and physical examination are the cornerstone for diagnosing the tendinopathy itself as well as to find possible contributing factors or underlying diseases. Additional diagnostic modalities such as ultrasound and magnetic resonance imaging can be used to confirm the clinical suspicion of tendinopathy.

This chapter provides an overview of the pathophysiology, patient history, risk factors, and physical examination of tendinopathy with special emphasis on the hindfoot tendons.

## 69.2 Nomenclature

Historically, many confusing terminologies have been used to define tendon injuries in the clinical setting. In 1998, the umbrella term “tendinopathy” was introduced to describe the combination of pain, swelling, and impaired function of the tendon in the clinical setting [4]. It was found that only after histologic examination of the damaged tissue, one can properly distinguish between subtypes of tendinopathy such as tendinitis, tenosynovitis, and tendinosis. In the context of physical examination of hindfoot tendinopathies, as in this chapter, the general term tendinopathy is therefore most appropriate to use.

---

P. A. D. van Dijk (✉)  
Department of Orthopaedic Surgery, Amsterdam  
University Hospital–Academic Medical Centre,  
University of Amsterdam,  
Amsterdam, The Netherlands

Academic Centre for Evidence Based Sports  
Medicine, Amsterdam, The Netherlands

Amsterdam Collaboration on Health and Safety  
in Sports, Amsterdam, The Netherlands  
e-mail: [p.a.d.vandijk@gmail.co](mailto:p.a.d.vandijk@gmail.co);  
[p.a.vandijk@amc.uva.nl](mailto:p.a.vandijk@amc.uva.nl)

## 69.3 Pathophysiology

Healthy tendons are highly organized structures to allow the transmission of high-energy forces between bones and muscles. Regulation of this strict organization is essential to maintain the tendon's integrity and mechanical properties and to prevent injuries [5, 6]. Normal response of tendon tissue to damage consists of inflammation, followed by proliferation or deposition of collagen matrix, and remodeling [7, 8]. In case of an inadequate inflammatory response, the tendon's structure becomes hypervascularized and disorganized by an increase of fibrin depositions, collagen breakdown, and a reduction of macrophages and neutrophils: the first step in the development of tendinopathy.

Both magnitude and distribution of the tendon's load play a major part in the development of tendinopathy [9, 10]. During repeated motion and loading, tendons become susceptible for tendinopathy because the tendon's repair mechanism has less time to heal possible microtraumas [9, 11]. Moreover, muscle activation and force redistribution within the tendon can result in nonuniform loading of the tendons, which in turn may cause microtraumas [9]. In case of ongoing mechanical forces in spite of these microtraumas, further tendon deterioration occurs which eventually leads to tendinopathy.

No consensus exists as to the exact etiology of tendinopathy. Several theories have been proposed, most of them being related to either the inadequate vascularization of the tendon or the tendon's inflammatory response to damage [12–17]. Ischemia might be induced by maximal loading of the tendon, followed by relaxation and subsequent generation of free radicals. These radicals potentially contribute to the development of degenerative changes in the tendon [17]. Moreover, local hypoxia may result in inadequate levels of ATP, which may also lead to tendon degeneration [12]. Others describe that tenocyte apoptosis is induced by high strain application [14]. In animal and human studies, tendon stress was related to the release of harmful inflamma-

tory mediators such as interleukin-6 and prostaglandin E<sub>2</sub>, which potentially induce histological degenerative changes [15, 16].

While tendons have some regenerative capacity, it is not likely that the tendon's tissue will return to the pre-injury level of mechanical properties and highly structured organization [18].

### 69.3.1 What Causes Pain in Tendinopathy?

While pain is the main reported symptom in tendinopathy, the cause remains an enigma. As per definition, pain serves to protect the tendon and surrounding tissues. Tendon pain is closely linked to extensive loading and activities, while it is barely experienced during rest or low-load activities. Moreover, "warming-up" of the tendons reduces pain [19, 20].

Many theories have been proposed in literature, as the molecular biology of tendons covers many potential contributors to the development of pain [21]. Some authors suggest that degeneration and disruption of the tendon tissue, including deposit of fibrin and mechanical breakdown of collagen, contribute to the development of pain as this disruption may stimulate nociceptors [22, 23].

Others hypothesize that the cause of tendon pain has to do with the tendon's complex network of neurons and neuronal mediators' innervation. The latter is also involved in the tendon's inflammatory response with levels increasing after injury [24–26]. This might explain a synchronized mechanism of tissue repair and stimulation of the nerves at the same time [25]. In 2003, Kahn et al. raised "the biochemical hypothesis" of pain in tendinopathy, relating to the release of biochemical agents in tendon inflammation. These agents might irritate nociceptors in the surrounding tissue or the tendon itself and in this way cause pain [27].

More recently, a theory was published suggesting that tendons itself cannot be the cause of pain due to their aneural and avascular nature. The fact that up to 32% of asymptomatic, unpainful Achilles tendons show histopathological

changes contributes to this theory [28, 29]. Instead, it is more likely that the pain is originated from outside the tendon. Damaged tendons generate cytokines, which induce neovascularization and neoinnervation from the paratenon or vincula into the tendon, as seen in chronic painful tendons. It is these neonerves that are responsible for the pain [30].

---

## 69.4 Patient History and Risk Factors

In general, up to 83% of all patients provide the diagnosis in their clinical history [31]. In patients with a hindfoot tendinopathy, there is little to suggest that this is any different. The cardinal complaint in patients with tendinopathy is dose-dependent pain during loading of the tendon, or in other words “too soon, too much, and/or too often” [32]. In early stages, pain is typically provoked after longer periods of loading while proper “warming-up” decreases pain complaints. In later stages of disease, pain can even be present in rest with worsening during activities. Seldomly, patients report an insidious onset of pain. Other complaints in tendinopathy include overuse, swelling, and functional impairment.

Tendinopathies are most common in the active population, as physical activities and/or occupation often determine the amount of force and stress on the tendons. Any tendon can be affected, depending on the type of sports or occupation performed. Runners and dancers, for example, show higher prevalence of flexor hallucis longus or Achilles tendinopathy. In fact, 52% of male elite runners are affected by Achilles tendinopathy at one point in their life [33].

Besides activity and overuse, several extrinsic and intrinsic factors are associated with the development of tendinopathy. Extrinsic factors include training errors such as disbalanced intensity, hill work, uneven grounds, fatigue, wrong technique, and excessive distances. Especially changes in load, technique, and/or equipment are associated with the onset of tendinopathy [32]. Also, environmental changes

and outdoor training in cold weather are considered risk factors [34].

Intrinsic factors include biomechanical abnormalities such as malalignment and difference in leg length, age, gender, and health status. Adults are more prone to tendinopathy in the lower extremity in comparison to adolescents [35]. In contrast to other anatomical regions of the body, in hindfoot tendinopathies, there is no difference in incidence between male and female patients [36].

Diseases such as diabetes, obesity, and autoimmune disorders may affect the biology and mechanical function of tendons, making the patient more prone to the development of tendinopathies. Moreover, tendinopathy may be the first clinical presentation of various metabolic disorders such as gout or hypercholesterolemia. Several pharmacological subgroups are associated with the development of tendinopathy. Among them are quinolone antibiotics, corticosteroids, statins, and aromatase inhibitors [37].

---

## 69.5 General Physical Examination

### 69.5.1 Inspection

Every orthopedic physical examination starts with a general inspection [38]. Since this is thoroughly described elsewhere in this book, it is not further discussed in this chapter. In hindfoot tendinopathies, special attention should be paid to the patient’s habitus, walking pattern, (mal) alignment, and possible leg-length difference.

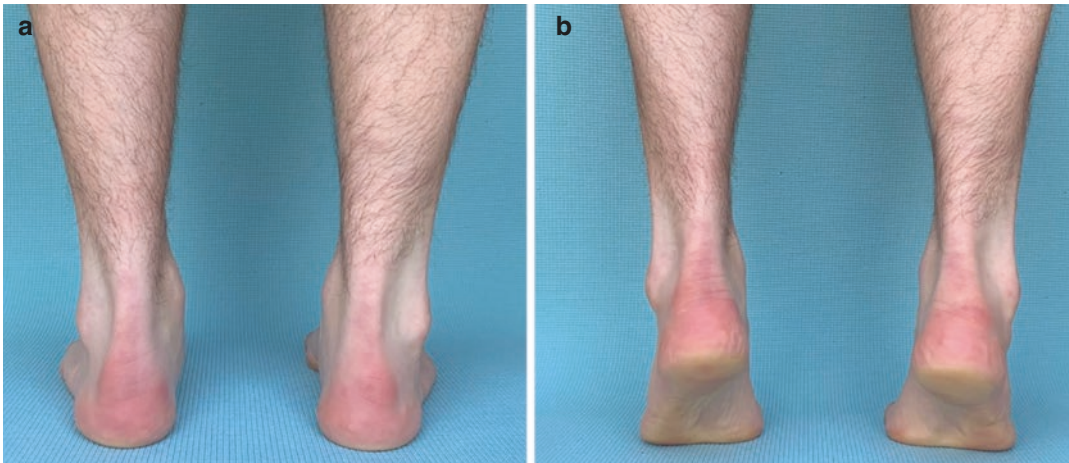
More specific for hindfoot tendinopathies, inspection should focus on the foot and ankle. Check the shoes for signs of abnormal or asymmetrical wear, which can indicate malalignment. With the patient on bare feet, check the alignment of the foot and ankle from behind and check the arch height (Fig. 69.1). A normal hindfoot has a valgus of approximately 5°. Depending on the type of malalignment, load of specific tendons is increased, which puts them at higher risk for the development of tendinopathy (see paragraph F). In case of a flatfoot, the Coleman block test can

be used to evaluate the flexibility of the malalignment. A block is placed under the lateral side of the foot in order to eliminate contribution of the first ray and pronation of the forefoot. In case of a flexible flatfoot, the alignment of the foot is corrected to neutral or valgus. A rigid hindfoot will not correct during the Coleman block test.

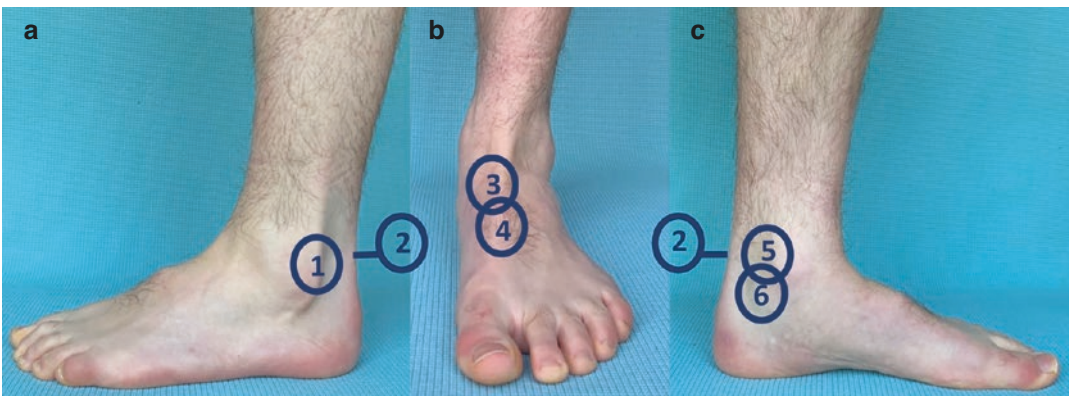
Check the lower extremity for scars, swelling, muscle atrophy, change of color, and hair growth. Swelling along the course of a specific tendon may indicate tendinopathy. Poor blood supply, an intrinsic risk factor for tendinopathy, may be indicated by decreased hair growth, hypertrophic nails, and a thinner skin.

### 69.5.2 Palpation

Palpation of the hindfoot is best performed with the patient sitting in a chair and the examiner's chair opposed. It should be done in a systemic manner and always compared to the other ankle. Due to the foot's superficial contours, the surface anatomy can be used as a guide for identification of possible pathology (Fig. 69.2). Check for areas of tenderness and bony or soft-tissue thickening: recognizable tenderness on palpation of the affected tendon is one of the most specific findings for tendinopathy during physical examination. Proper knowledge of the hindfoot's anatomy



**Fig. 69.1** Hindfoot evaluation in (a) neutral and (b) forefoot loaded position



**Fig. 69.2** Evaluation of tendons in the ankle via surface anatomy

**Table 69.1** Hindfoot tendon function

Hindfoot tendon	Function
<b>Flexors and plantiflexors</b>	
Posterior tibial tendon	Plantarflexion, inversion and adduction of the ankle
Peroneus brevis tendon	Plantarflexion, abduction, eversion of the ankle
Peroneus longus tendon	Plantarflexion of the hallux, abduction and eversion of the ankle
Flexor hallucis longus tendon	Flexion of the hallux (distal phalanx)
Flexor digitorum longus tendon	Flexion of digiti 2-5
Achilles tendon <i>Discussed in chapter 5 of the Ankle section</i>	Plantarflexion of the ankle
<b>Extensors and dorsiflexors</b>	
Anterior tibial tendon	Dorsiflexion, inversion and adduction of the ankle
Peroneus tertius*	Dorsiflexion and eversion of the ankle
Extensor hallucis longus	Extension of the hallux (distal phalanx)
Extensor digitorum longus	Extension of digiti 2-5

is therefore essential. Once areas of tenderness are found, define the limits of tenderness and possible changes by loading and motion of the ankle.

### 69.5.3 Range of Motion and Muscle Strength

Range of motion of the ankle should be performed systematically. It is important to test both active and passive range of motion and compare to the contralateral side. Please refer to Table 69.1 for an overview of the function of specific hindfoot tendons. Deficits in range of motion or reduction of muscle strength may be explained by injury of specific tendons. It should be kept in mind that the culprit may be in a different anatomic region. For example, lack of active hallux extension may indicate pathology of the extensor hallucis longus but can also be related to a nervus peroneus injury of L5 radiculopathy.

### 69.5.4 Pain Provocation Test

As tendon pain is loading dose-dependent, recognizable pain may be provoked during extensive loading or active motion against resistance. In

Table 69.1, and as further discussed in paragraph F, the specific function to test for each tendon is summarized.

## 69.6 Physical Examination of Hindfoot Tendinopathy

### 69.6.1 Posterior Tibial Tendon

The posterior tibial tendon acts as the primary invertor of the ankle and dynamic stabilizer of the medial arch and plays an important role in the medial ankle stability during walking. Dysfunction of the tendon is the most common cause of acquired flatfoot deformity. While changes in the tendon's structure become obvious in later stages of tendinopathy, the degenerative process begins far before clinical disease is apparent. Adequate clinical history and physical examination lead to earlier diagnosis, potentially preventing the patient from further deterioration and invasive treatments.

Posterior tibial tendinopathy is often seen after overuse or extensive loading in high-impact sports such as tennis, soccer, and basketball, as the tendon may suffer repetitive microtraumas due to extensive loading. Woman and patients older than 40 years are also at



**Fig. 69.3** Assessment of Posterior Tibial tendon function

greater risk. Patients typically demonstrate pain at the medial side of the ankle during activities such as running and walking. In later stages of disease, pain can also be experienced in front of the foot or even on the lateral side of the ankle.

During inspection, a pes planus or “too-many-toes sign” may be observed. A Coleman block test often reveals a flexible flatfoot in early stages of the disease, and a more rigid flatfoot in severe tendinopathy. Swelling may be presented over the course of the tendon, and pain is often provoked during palpation of the tendon (Fig. 69.2). Active plantar flexion and inversion against resistance may reveal pain and loss of muscle strength (Fig. 69.3). Single-leg heel raise is a useful clinical tool to differentiate the severity of tendinopathy: in an early stage, patients are able to stand tiptoes on the affected foot without pain. In later stages, standing on the affected foot and coming up on tiptoes will provoke pain or the rigid deformity may prevent the patient from completing the test at all [39].

### 69.6.2 Anterior Tibial Tendon

The anterior tibial tendon functions as a dorsiflexor and invertor of the ankle. Moreover, it stabilizes the ankle during walking as the foot hits the ground and pulls it clear of the ground during push-off. Tendinopathy of the anterior tibial tendon may result from overuse, with specific risk in activities such as running up and down hill. During uphill running, the tendon must lift the foot higher than normal. In downhill running, the tendon is lengthening and contracting at the same time, which puts even higher loads on the tendon. Moreover, the tendinopathy may be caused by tight shoelaces or strapping over the front of the ankle, causing higher compression on the tendon.

Patients typically present with pain complaints in front of the ankle that worsens with activity. Other symptoms include weakness during dorsiflexion or “slapping” of the foot on the ground when walking.

During physical examination, pain and swelling can be found over the course of the tendon, especially in front of the ankle (Fig. 69.3). Active dorsiflexion against resistance often exacerbates pain and may reveal loss of muscle strength (Fig. 69.4).

### 69.6.3 Peroneal Tendons

Together, the peroneal tendons act as the primary evertors and abductors of the foot. In this way, they play an important role in the active lateral ankle stability, the foot’s eversion strength, and stabilization of the lateral column of the foot during stance. Tendinopathy of the tendons is often described after a gross ankle sprain in the patient’s history or in case of chronic lateral ankle instability.

Typically, patients present with lateral ankle pain or pain along the course of the tendons that worsens with activity. Other symptoms include tenderness, giving way, and lateral ankle instability. On inspection, a cavovarus foot may be observed just as swelling over the course of the

peroneal tendons. Palpation of the tendons often generates recognizable tenderness, pain, and/or crepitus (Fig. 69.2). Passive inversion and plantar

flexion or active eversion and dorsiflexion against resistance typically exacerbate pain (Fig. 69.5). Moreover, muscle strength can be weaker when compared to the contralateral side.

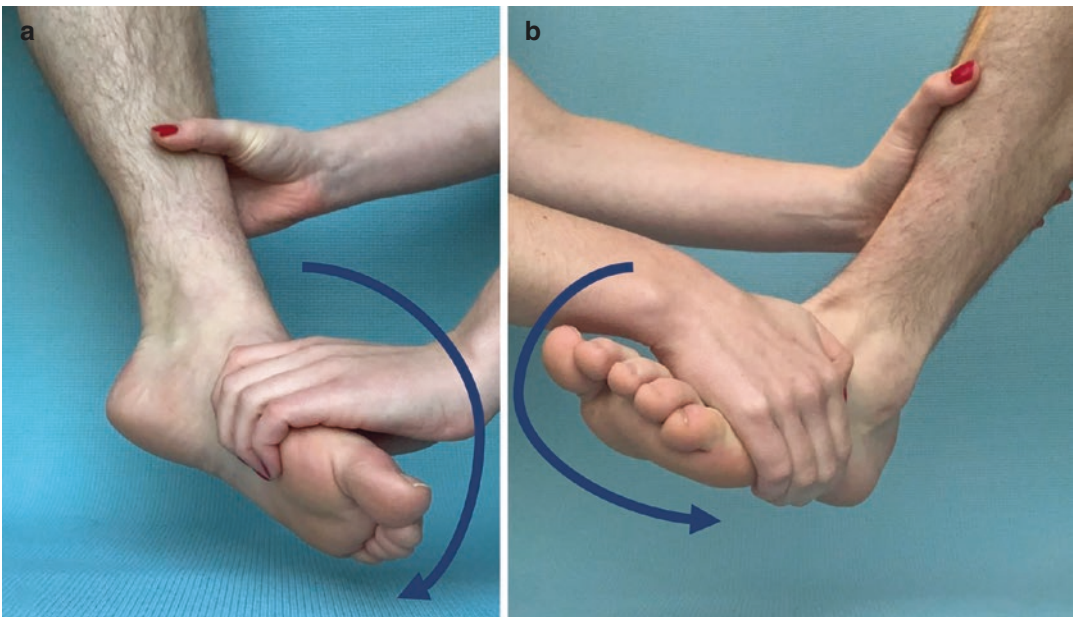


**Fig. 69.4** Evaluation of Anterior Tibial tendon

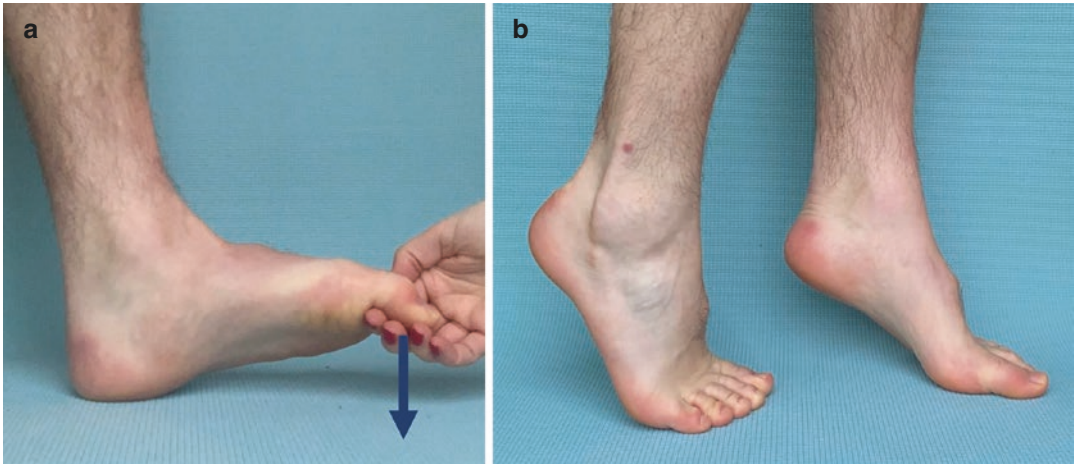
#### 69.6.4 Flexor Hallucis Longus

The flexor hallucis longus tendon is the primary flexor of the hallux by plantarflexing the talocrural, metatarsophalangeal, and interphalangeal joints of the hallux while also aiding in plantar flexion and inversion of the ankle. Tendinopathy of the flexor hallucis longus, also known as “dancer’s tendinopathy,” may result from high tendon loads in extreme plantar flexion of the ankle and flexion of the hallux. It is most often found in classical ballet dancers but is also related to athletes performing frequent push-off maneuvers such as gymnasts, skaters, long-distance runners, and swimmers.

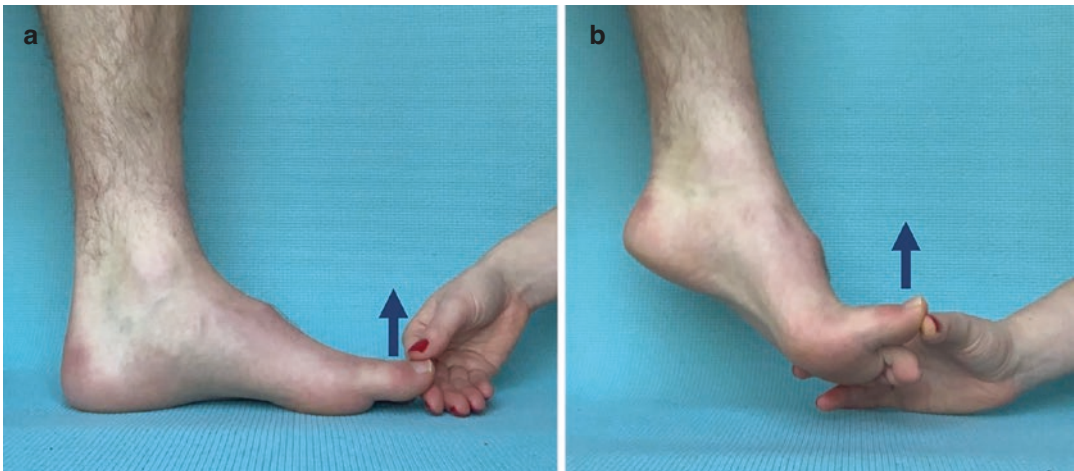
Pain typically starts around the posteromedial side of the ankle or on the medial side of the subtalar joint, below the tip of the medial malleolus. It may be provoked by plantar flexion such as push-off in running or relevé in ballet. Moreover, it is related with other pathologies such as an os



**Fig. 69.5** Evaluation of Peroneal tendon function



**Fig. 69.6** Evaluation of Flexor Hallucis Longus tendon function



**Fig. 69.7** Evaluation of Extensor Hallucis Longus tendon function

trigoneum, bony avulsions, ankle and subtalar arthrosis, and loose bodies [40–43].

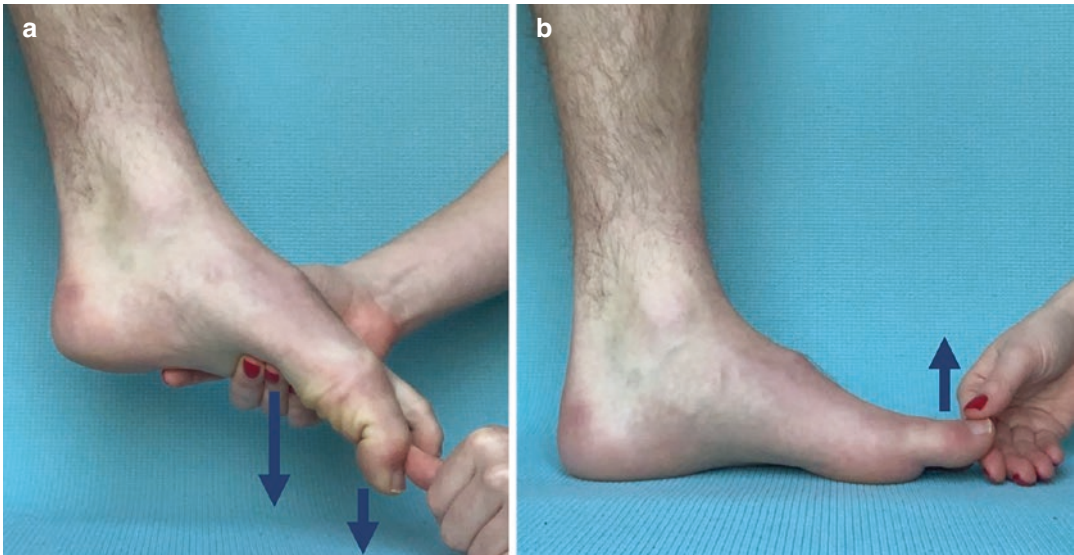
Tenderness on palpation of the flexor hallucis longus usually reveals flexor hallucis longus tendinopathy (Fig. 69.2). The tendon can be palpated behind the medial malleolus in its sliding canal by asking the patient to flex the toes repetitively with the ankle in a position of slight plantar flexion (10–20°). Flexion of the hallux against resistance or walking on tiptoes may provoke recognizable pain (Fig. 69.6). Moreover, passive extension of the hallux with the ankle in both neutral and plantarflexed positions can be

compared: in case of limited to no extension in neutral position that disappears in plantar flexion, the flexor hallucis longus was likely entrapped which might indicate tendinopathy (Fig. 69.7). Passive extension of the hallux with the ankle in plantar flexion can also provoke pain complaints (Fig. 69.7b).

### 69.6.5 Extensor Hallucis Longus

The extensor hallucis longus tendon acts as the primary extensor of the hallux and contributes to





**Fig. 69.8** Evaluation of Extensor Hallucis tendon strength and function

dorsiflexion of the ankle. Moreover, it dorsiflexes the ankle and inverts the foot. In this manner, it allows clearance off the ground during walking. Isolated tendinopathy of the extensor hallucis longus is uncommon [44]. It may be caused by eccentric overload with repeated friction of the tendon passing under the extensor retinaculum at the front of the ankle, also known as “ultramarathoner’s ankle” [45]. Other causative factors include excessive pronation, eccentric overload, muscle imbalance, and tight shoelaces or strapping over the front of the ankle [46–48].

Patients typically indicate pain over the course of the tendon, especially in front of the ankle (Fig. 69.2). Other symptoms include weakness during extension of the hallux, “slapping” of the foot on the ground when walking, and a characteristic ultramarathoner’s gait.

During physical examination, pain and swelling can be found over the course of the tendon, especially in front of the ankle (Fig. 69.2). Forced passive flexion of the hallux with the ankle in plantar flexion and active extensor of the hallux

against resistance often exacerbate pain (Fig. 69.8). The latter may also reveal loss of muscle strength.

## 69.7 Conclusion

Tendinopathies are common injuries in the hindfoot and can result in high morbidity for the patient. While significant deterioration of the tendon’s structure becomes obvious in later stages of tendinopathy, the degenerative process begins far before clinical disease is apparent. Adequate clinical history and physical examination are key to early diagnosis, potentially preventing the patient from further deterioration and invasive treatments.

Typically, tendinopathy occurs in the active population and results from overuse or “too soon, too much, and/or too often.” In case of symptoms such as loading dose-dependent pain, swelling, tenderness along the course of a specific tendon, and provocation of pain during active motion against resistance, always consider the possibility of a tendinopathy.

## 69.8 Pearls and Pitfalls

- Adequate clinical history and physical examination are key to early diagnosis of tendinopathy, potentially preventing the patient from further deterioration and invasive treatments.
- The cardinal complaint in patients with tendinopathy is dose-dependent pain during loading of the tendon, or in other words “too soon, too much, and/or too often.”
- Tenderness on palpation of the affected tendon is one of the most specific findings for tendinopathy during physical examination.
- Proper knowledge on the anatomy of the hindfoot is mandatory for diagnosing hindfoot tendinopathies.

## References

1. Kaux JF, Forthomme B, Goff CL, Crielaard JM, Croisier JL. Current opinions on tendinopathy. *J Sports Sci Med*. 2011;10(2):238–53.
2. Gorter K, de Poel S, de Melker R, Kuyvenhoven M. Variation in diagnosis and management of common foot problems by GPs. *Fam Pract*. 2001;18(6):569–73.
3. Yelin E, Weinstein S, King T. An update on the burden of musculoskeletal diseases in the U.S. *Semin Arthritis Rheum*. 2019;49(1):1–2.
4. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. *Arthroscopy*. 1998;14(8):840–3.
5. Bi Y, Ehrichiou D, Kilts TM, Inkson CA, Embree MC, Sonoyama W, et al. Identification of tendon stem/progenitor cells and the role of the extracellular matrix in their niche. *Nat Med*. 2007;13(10):1219–27.
6. Lin TW, Cardenas L, Soslowsky LJ. Biomechanics of tendon injury and repair. *J Biomech*. 2004;37(6):865–77.
7. Fu SC, Rolf C, Cheuk YC, Lui PP, Chan KM. Deciphering the pathogenesis of tendinopathy: a three-stages process. *Sports Med Arthrosc Rehabil Ther Technol*. 2010;2:30.
8. Andarawis-Puri N, Flatow EL, Soslowsky LJ. Tendon basic science: development, repair, regeneration, and healing. *J Orthop Res*. 2015;33(6):780–4.
9. Nakama LH, King KB, Abrahamsson S, Rempel DM. Effect of repetition rate on the formation of microtears in tendon in an in vivo cyclical loading model. *J Orthop Res*. 2007;25(9):1176–84.
10. Nakama LH, King KB, Abrahamsson S, Rempel DM. Evidence of tendon microtears due to cyclical loading in an in vivo tendinopathy model. *J Orthop Res*. 2005;23(5):1199–205.
11. Arndt AN, Komi PV, Bruggemann GP, Lukkariniemi J. Individual muscle contributions to the in vivo Achilles tendon force. *Clin Biomech (Bristol, Avon)*. 1998;13(7):532–41.
12. Birch HL, Rutter GA, Goodship AE. Oxidative energy metabolism in equine tendon cells. *Res Vet Sci*. 1997;62(2):93–7.
13. Goodship AE, Birch HL, Wilson AM. The pathobiology and repair of tendon and ligament injury. *Vet Clin North Am Equine Pract*. 1994;10(2):323–49.
14. Arnoczky SP, Tian T, Lavagnino M, Gardner K, Schuler P, Morse P. Activation of stress-activated protein kinases (SAPK) in tendon cells following cyclic strain: the effects of strain frequency, strain magnitude, and cytosolic calcium. *J Orthop Res*. 2002;20(5):947–52.
15. Skutek M, van Griensven M, Zeichen J, Brauer N, Bosch U. Cyclic mechanical stretching enhances secretion of interleukin 6 in human tendon fibroblasts. *Knee Surg Sports Traumatol Arthrosc*. 2001;9(5):322–6.
16. Stone D, Green C, Rao U, Aizawa H, Yamaji T, Niyibizi C, et al. Cytokine-induced tendinitis: a preliminary study in rabbits. *J Orthop Res*. 1999;17(2):168–77.
17. Wang MX, Wei A, Yuan J, Clippe A, Bernard A, Knoops B, et al. Antioxidant enzyme peroxiredoxin 5 is upregulated in degenerative human tendon. *Biochem Biophys Res Commun*. 2001;284(3):667–73.
18. Frank C, McDonald D, Shrive N. Collagen fibril diameters in the rabbit medial collateral ligament scar: a longer term assessment. *Connect Tissue Res*. 1997;36(3):261–9.
19. Bagge J, Gaida JE, Danielson P, Alfredson H, Forsgren S. Physical activity level in Achilles tendinosis is associated with blood levels of pain-related factors: a pilot study. *Scand J Med Sci Sports*. 2011;21(6):e430–8.
20. Ferretti A. Epidemiology of jumper's knee. *Sports Med*. 1986;3(4):289–95.
21. Rio E, Moseley L, Purdam C, Samiric T, Kidgell D, Pearce AJ, et al. The pain of tendinopathy: physiological or pathophysiological? *Sports Med*. 2014;44(1):9–23.
22. Snedeker JG, Foolen J. Tendon injury and repair—a perspective on the basic mechanisms of tendon disease and future clinical therapy. *Acta Biomater*. 2017;63:18–36.
23. Gotoh M, Hamada K, Yamakawa H, Inoue A, Fukuda H. Increased substance P in subacromial bursa and shoulder pain in rotator cuff diseases. *J Orthop Res*. 1998;16(5):618.
24. Danielson P, Alfredson H, Forsgren S. Distribution of general (PGP 9.5) and sensory (substance P/CGRP) innervations in the human patellar tendon. *Knee Surg Sports Traumatol Arthrosc*. 2006;14(2):125–32.
25. Ackermann PW, Ahmed M, Kreicbergs A. Early nerve regeneration after Achilles tendon rupture—a prerequisite for healing? A study in the rat. *J Orthop Res*. 2002;20(4):849.

26. Alfredson H, Thorsen K, Lorentzon R. In situ microdialysis in tendon tissue: high levels of glutamate, but not prostaglandin E2 in chronic Achilles tendon pain. *Knee Surg Sports Traumatol Arthrosc.* 1999;7(6):378–81.
27. Khan KM, Cook JL, Maffulli N, Kannus P. Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin. *Br J Sports Med.* 2000;34(2):81–3.
28. Haims AH, Schweitzer ME, Patel RS, Hecht P, Wapner KL. MR imaging of the Achilles tendon: overlap of findings in symptomatic and asymptomatic individuals. *Skelet Radiol.* 2000;29(11):640–5.
29. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L, et al. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. *Br J Sports Med.* 2003;37(2):149–53.
30. van Sterkenburg MN, van Dijk CN. Mid-portion Achilles tendinopathy: why painful? An evidence-based philosophy. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(8):1367–75.
31. Hampton JR, Harrison MJ, Mitchell JR, Prichard JS, Seymour C. Relative contributions of history-taking, physical examination, and laboratory investigation to diagnosis and management of medical outpatients. *Br Med J.* 1975;2(5969):486–9.
32. Renström PA, Woo SL. Tendinopathy: a major medical problem in sport. In: Woo SL, Renström PA, Arnoczky S, editors. *Tendinopathy in athletes.* London: Wiley-Blackwell; 2008.
33. Kujala UM, Sarna S, Kaprio J. Cumulative incidence of Achilles tendon rupture and tendinopathy in male former elite athletes. *Clin J Sport Med.* 2005;15(3):133–5.
34. Milgrom C, Finestone A, Zin D, Mandel D, Novack V. Cold weather training: a risk factor for Achilles paratendinitis among recruits. *Foot Ankle Int.* 2003;24(5):398–401.
35. Albers IS, Zwerver J, Diercks RL, Dekker JH, Van den Akker-Scheek I. Incidence and prevalence of lower extremity tendinopathy in a Dutch general practice population: a cross sectional study. *BMC Musculoskelet Disord.* 2016;17:16.
36. Morton S, Williams S, Valle X, Diaz-Cueli D, Malliaras P, Morrissey D. Patellar tendinopathy and potential risk factors: an international database of cases and controls. *Clin J Sport Med.* 2017;27(5):468–74.
37. Knobloch K. Drug-induced tendon disorders. *Adv Exp Med Biol.* 2016;920:229–38.
38. van Dijk PA, van Dijk CN. Orthopedisch onderzoek. In: Wiersinga WJ, Levi M, Schimmer B, Savelkoel J, van der Zande JMJ, editors. *Handboek voor de co-assistent.* Houten: Springer; 2021.
39. Ellis SJ, Deyer T, Williams BR, Yu JC, Lehto S, Maderazo A, et al. Assessment of lateral hindfoot pain in acquired flatfoot deformity using weight-bearing multiplanar imaging. *Foot Ankle Int.* 2010;31(5):361–71.
40. Hamilton WG. Tendinitis about the ankle joint in classical ballet dancers. *Am J Sports Med.* 1977;5(2):84–8.
41. Sammarco GJ, Cooper PS. Flexor hallucis longus tendon injury in dancers and nondancers. *Foot Ankle Int.* 1998;19(6):356–62.
42. Michelson J, Dunn L. Tenosynovitis of the flexor hallucis longus: a clinical study of the spectrum of presentation and treatment. *Foot Ankle Int.* 2005;26(4):291–303.
43. Solomon R, Brown T, Gerbino PG, Micheli LJ. The young dancer. *Clin Sports Med.* 2000;19(4):717–39.
44. Perlman MD, Leveille D. Extensor digitorum longus stenosing tenosynovitis. A case report. *J Am Podiatr Med Assoc.* 1988;78(4):198–9.
45. Fallon KE. Musculoskeletal injuries in the ultramarathon: the 1990 Westfield Sydney to Melbourne run. *Br J Sports Med.* 1996;30(4):319–23.
46. Bishop GW, Fallon KE. Musculoskeletal injuries in a six-day track race: ultramarathoner's ankle. *Clin J Sport Med.* 1999;9(4):216–20.
47. So CH, Siu TO, Chan KM, Chin MK, Li CT. Isokinetic profile of dorsiflexors and plantar flexors of the ankle—a comparative study of elite versus untrained subjects. *Br J Sports Med.* 1994;28(1):25–30.
48. Stanish WD, Curwin S, Rubinovich M. Tendinitis: the analysis and treatment for running. *Clin Sports Med.* 1985;4(4):593–609.