Achilles tendinopathy: understanding the key concepts to improve clinical management
Achilles tendinopathy: understanding the key concepts to improve clinical management

Charlotte Ganderton¹, Jill Cook², Sean Docking², Ebonie Rio², Mathijs van Ark²,³, Jamie Gaida²,⁴

¹. La Trobe University, College of Science, Health and Engineering, Melbourne, Australia
². Monash University, Department of Physiotherapy, Melbourne, Australia
³. University of Groningen, University Medical Center Groningen, Center for Sports Medicine, Groningen, The Netherlands
⁴. Canberra University, Discipline of Physiotherapy, Canberra, Australia

Abstract

Introduction: Achilles tendinopathy is commonly encountered in clinical practice yet can be quite difficult to successfully treat. Relative overload is the precursor to most presentations, while systemic conditions can decrease the amount of load that triggers overload. While there is evidence for the use of eccentric exercise, it is not recommended in isolation for most presentations of Achilles tendinopathy as it fails to address strength and kinetic chain deficits, which can leave the individual vulnerable to recurrence. Insertional tendinopathy requires a tailored management that avoids dorsiflexion, as this position compresses the tendon onto the calcaneus.

Purpose: This masterclass summarises the tendinopathy continuum and articulates the authors' clinical reasoning and hands-on experience managing Achilles tendinopathy. We outline graded loading concepts while emphasising that relying on recipes is likely to fail. We also provide a perspective on the role of central pain processing and peripheral input from nociceptive fibres in the context of tendinopathy.

Implications: Rehabilitation should be tailored to address identified impairments (muscle bulk asymmetries, kinetic chain dysfunction, tolerance of energy storage and release in the Achilles tendon), and progressively work toward movements and activities relevant for the individual’s sport or daily activities. Within the three-stage rehabilitation sequence, stage 1 aims to reduce pain and increase calf muscle bulk; stage 2 focuses on improving power within the whole kinetic chain, and movement control during jumping and landing; and stage 3 begins to retrain sport specific load, and carefully introduces movements that require energy storage and release within the tendon.

Introduction

Treating achilles tendinopathy requires great clinical skills applied by thoughtful practitioners. Although often thought of as a simple diagnosis with straightforward treatment, it can be a complex and challenging condition to manage. The pathology, the differential diagnoses of mid and insertional Achilles tendinopathy and the role of eccentrics need to be considered for every individual you see with this condition.

Tendons are specialised connective tissue structures that transfer forces produced by contractile cells of muscles to bone. The Achilles tendon has an additional specialised function to provide shock absorption and energy storage (e.g. while hopping) to facilitate efficient movement. The Achilles mid-portion is between 2cm and 6cm from the calcaneus; the Achilles insertion less than 2cm from the calcaneus.

Tendon pathology continuum model

Tendon is comprised of cells (tenocytes) embedded in an extracellular matrix of collagen, elastin and ground substance (proteoglycans, glycoproteins and water) (Figure 1). Tendon cells are responsive to load, and will adapt the extracellular matrix to withstand the loading environment. Disruption to this process through relative overload alters the proteins produced by the cell, which can result in tendon pathology that ultimately decreases the capacity of the tendon to tolerate loading.

Figure 1: Normal tendon histology (H&E stain, 100x)
The transition from normal tendon to pathology has been described in the tendon pathology continuum model by Cook and Purdam (Figure 2). It describes three overlapping stages within a continuum – reactive tendinopathy, tendon dysrepair and degenerative tendinopathy. These stages have increasing degrees of pathology and decreasing capacity of the pathological area to recover. Progression is influenced by change in tendon load and intrinsic factors, including genetics, adiposity, cholesterol, insulin resistance, diabetes and menopause.

Reactive tendon pathology: A short term adaptive, non-inflammatory response of the cell (activation and proliferation) in response to acute overload. The extracellular matrix is altered with increased proteoglycan content but with little change in the collagen. The proteoglycans produced (predominantly aggrecan & versican) bind more water and cause tendon thickening that is commonly seen in response to an acute tensile or compressive overload. This increase in cross-sectional area reduces tendon stress by decreasing force per unit area. This process occurs in response to i) acute overload, ii) return to loading after a rest, or iii) following a direct blow to the tendon and is reversible if appropriately managed.

Tendon dysrepair: An attempt of the tendon to regain extracellular matrix structure through ongoing cell activation, resulting in greater disruption of the extracellular matrix and possible vascular and neuronal ingrowth. These tendons are difficult to distinguish clinically but are more common in a young person with a chronically overloaded tendon.

Degenerative tendon pathology: The tendon often remains hypercellular but there is little capacity for reversal of pathology due to significant (collagen) fibrillar disorganisation. Areas of cell death due to apoptosis, trauma or tenocyte exhaustion may be apparent on biopsy. Islands of degenerative tendon are interspersed between other stages of pathology and normal tendon (Figure 3). Typically, these tendons are seen in active middle age people, and clinically they may have focal nodular areas in the tendon.

Other pathology: Combinations of pathology can occur, the most common being a reactive on degenerative pathology, where there is reactive pathology in the remaining normal part of the tendon, while the degenerative part remains unchanged (Figure 4). These different stages of pathology all require different rehabilitation, as there is no ‘one size fits all’ exercise program.

Assessment for tendinopathy

The subjective assessment should include a thorough history of loading patterns, 24-hour response to loading (i.e. pain and stiffness) and whether known tendinopathy risk factors are present (diabetes, rheumatological conditions, fluoroquinolone antibiotics). The clinician needs to be aware that a player who has recently returned to sport after time off with an injury may be susceptible to tendinopathy, especially if they rapidly return to full load. Physical examination of Achilles tendinopathy should involve assessment of calf muscle bulk and strength, kinetic chain function and the capacity of the tendon to store and release elastic energy. In addition to a complete biomechanical analysis, the patient’s pain level during graded functional tasks (i.e. double heel rise followed by single-leg heel raise and/or repeat single-leg hops) should be quantified using a pain scale (numerical rating scale). Dysfunction should be assessed using a validated outcome measures such as the Victorian Institute of Sport Assessment – Achilles (VISA-A) questionnaire. It is important to note that
tendons may be painful to palpation (especially in athletes) in people who do not have tendinopathy, and that palpation pain does not help predict prognosis. Pain localisation depends on whether the presentation is mid-substance — where people pinch with two fingers either side of the mid tendon, or insertional — where people point with one finger near the Achilles insertion. These pain presentations do not refer and vague pain is likely to be associated with another diagnosis. Potential differential diagnoses include seronegative arthropathies, irritation or neuroma, or trigonum syndrome, and peroneal tendon subluxation or dislocation. The presence of an invaginated plantaris tendon or a plantaris tendon that is firmly attached to the Achilles tendon via a retinacular-like structure may be detected in cases of recalcitrant medial-sided mid-portion tendinopathy. When retrocalcaneal bursitis is diagnosed, usually via imaging, it best to consider the entire enthesis organ. That is, the bursa and tendon should not be considered in isolation. Finally, it should be recalled that pathology can exist on imaging without the tendon being the source of symptoms, and therefore a clinical diagnosis incorporating the above pain localisation patterns and the presence of load-related pain should form the basis for client management.

Eccentric training in Achilles tendinopathy

Eccentric training is a common intervention when rehabilitating Achilles tendinopathy, however, systematic reviews have identified low methodological quality among studies on this intervention. Additionally, there is substantial variability in the protocol used for eccentric training, for example i) 3x15 straight, 3x15 knee bent, twice daily ii) gradual increase up to 3x15 over 1-2 weeks, iii) 1x15 straight, 1x15 knee bent, iv) once daily, v) thrice daily, vi) addition of 10-second hold. There are unique characteristics of eccentric exercise that may have important implications for tendinopathy. These include modulation the neurological stretch response, perturbations of tendon force, increased shear forces between the tendon and paratendon structures, pain modulation, or adaption of mechanotransduction signalling in passive tendon structures. Despite these data, it is unclear whether it is important to isolate the eccentric component of the exercise rather than performing a concentric-eccentric exercise.

The number of repetitions, sets and frequency of calf exercises in the Alfredson program (3 sets of 15 repetitions of heel drops off a step with the knee straight and knee bent, at slow speed, twice daily) do not correlate with guidelines for strength training to achieve hypertrophy and contractile strength in the triceps surae muscle. Thus, a functional strengthening and endurance program that includes loading into the stretch shortening cycle (SSC) should also be used in conjunction with an eccentric program when rehabilitating an Achilles tendinopathy. The next section provides further detail of how eccentrics may fit within a graded loading program.

Exercise therapy for Achilles tendinopathy

Once Achilles tendinopathy is diagnosed, a rehabilitation program should be commenced with the primary focus of decreasing or abolishing pain during tendon loading activities and improving function. The program should be designed around an individual’s pre-injury function and sporting level with the ultimate goal of treatment being full return to activity. Essential elements include: regular increases in weight/resistance to progressively load muscle tendon unit (low repetition, high load) followed by increased speed of exercises to load the tendon. A three stage progressive loading rehabilitation program has been described that involves addressing functional impairments, from muscle-tendon strength, to power (including kinetic chain and lumbo-pelvic control), to sport-specific exercise. Not only does the calf and Achilles complex need full scrutiny and rehabilitation, but the strength and function of the kinetic chain must be treated as well. The program should be adapted for different presentations and for different stages of pathology. Rehabilitation should be continued until the tendon regains full capacity to store and release energy without immediate or latent pain, and then be followed by a maintenance program if returning to competitive sport. The energy storage activities used at the end of the rehabilitation program should match those required in the individual’s sport or in a patient-centred goal. Ongoing monitoring with clinical tests and standardised outcome measures should be undertaken throughout the rehabilitation process.

Exercise is the best intervention for tendinopathy, and is the only stimulus with the capacity to positively affect tendon matrix. While exercise is the cornerstone of rehabilitation for all stages of pathology, its application should vary according to presentation. Reactive tendinopathy requires relative rest from high tendon load activities (energy storage and release activities such as running) and prescription of isometric exercise for pain reduction. In contrast, dysrepair and degenerative pathology require progressive loading to moderate cell response and address

---

**Table 1: Rehabilitation exercises to restore functional impairments in rehabilitation stages. Adapted from 'Rehabilitation of Achilles and patellar tendinopathies', A Kountouris and J Cook, 21(2), 295-316, 2007 in Best Practice & Research Clinical Rheumatology.**

<table>
<thead>
<tr>
<th>Rehabilitation Stage</th>
<th>Functional impairment</th>
<th>Exercise Aim</th>
<th>Example Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Muscle-tendon function</td>
<td>Decrease pain, improve Achilles – calf strength</td>
<td>Heavy sustained isometric contractions (preferably single leg), progressing on to heel raise exercise on a step or flat floor (note that insertional presentations should not be given exercises below neutral off a step due to compression)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Power and lower limb kinetic chain function</td>
<td>Improve capacity and control during faster movements. Promote smooth movements at the hip, knee and ankle during impact and load</td>
<td>Skipping – commence with two legs and stop between each skip; progress to continuous; progress to single leg and then add speed</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Sport-specific exercise with emphasis on energy storage and release</td>
<td>Gradually increase sports-specific load to the tendon and monitor reaction to higher impact loading</td>
<td>Sprinting Jumping/landing Acceleration activities Change of direction activities</td>
</tr>
</tbody>
</table>
Pain relief can be achieved with a substantial heel raise (preferably added to the outside of the shoe, in-shoe raises are often poorly tolerated) for everyday activity keeping the tendon out of compression, i.e. progressive loading in more plantar flexion.

The key diagnostic feature separating mid-portion and insertional Achilles tendinopathy is the area of pain, i.e. progressive loading in more plantar flexion. Applying the concept clinically, insertional Achilles tendinopathy presents with pain occurring in dorsiflexion based activities but less so in plantarflexion. Thus, stretching the Achilles over a step or completing the original Alfredson program may provoke pain as will running up hills with a triphasic inflammatory process. Nociception from vascular structures, tenocytes, biochemical changes, cell activation, ion channel and matrix changes may contribute to the development of a painful tendon.

Some features of tendon pain are physiological whereas others are pathophysiological, and both peripheral and central input contribute to the clinical features of tendinopathy. Localised pain in response to tendon loading appears to have a physiological mechanism driven by primary nociceptive factors. Many tendinopathies, especially in the lower limb, have a relatively simple pain presentation without typical chronic pain features such as spreading, despite a long duration of pain.

Diagnostic and therapeutic difference between treating mid-portion and insertional Achilles tendinopathy

Normal tendon attaches to bone through the enthesis organ. This complex attachment allows compression of the tendon against the upper aspect of the calcaneus to reduce load on the insertion and provide a mechanical advantage to the muscle-tendon unit.

This area of compression proximal to the tendon insertion is where pathology most commonly occurs and compression is maximal in dorsiflexion. Applying the concept clinically, insertional Achilles tendinopathy presents with pain occurring in dorsiflexion based activities but less so in plantarflexion. Thus, stretching the Achilles over a step or completing the original Alfredson program may provoke pain as will running up hills or on soft surfaces such as the beach (the heel digs into the sand which increases dorsiflexion) (Table 1 & Figure 6). In addition to aggravating activities, the key diagnostic feature separating mid-portion Achilles from insertional Achilles is the area of pain, remembering that tendon pain is always well localised and the area indicated as their main site of pain is a good diagnostic sign.

Insertional Achilles tendinopathy should be treated by keeping the tendon out of compression, i.e. progressive loading in more plantar flexion. Pain relief can be achieved with a substantial heel raise (preferably added to the outside of the shoe, in-shoe raises are often poorly tolerated) for everyday activity. As pain reduces and tendon capacity improves, compressive loads can be gradually re-introduced if required.

Figure 5: Tendon attachment to bone and the enthesis organ. Schematic representation demonstrating the complex nature of the tendon attachment to the bone and the fibrocartilage where the tendon and bone are adjacent. Reproduced from Cook JL. Tendon. In: Kolt G, Snyder-Mackler L, editors. Physical Therapies in Sport and Exercise. London: Churchill Livingstone; 2007

Figure 6: 1. A sagittal section of an Achilles tendon insertion, the figure on the left demonstrates a tendon inserting inferior to a prominent superior tuberosity on the calcaneus, the figure on the right shows a tendon that inserts on the posterior surface of the calcaneus that lacks a prominent superior tuberosity. Compression will occur at the superior aspect of the calcaneus in the tendon in the left figure, but not in the tendon in the right figure. Reproduced with permission from A. Rufai, J. R. Ralphs, M. Benjamin, Journal of Orthopaedic Research, 'Structure and histopathology of the insertional region of the human Achilles tendon', 13, 4, 585-593, 2005, Wiley Online Library DOI: 10.1002/jor.1100130414
There may be important differences between upper and lower limb tendinopathies in terms of the contribution from peripheral tissue or central drivers. While the role of central sensitisation has been highlighted for rotator cuff and lateral elbow tendinopathy, there is preliminary evidence that central sensitisation does not occur in Achilles tendinopathy. The importance of upper limb function for self-care and occupational activities, might contribute to these differences in central sensitisation between upper and lower limb tendinopathies. As all nociceptive input is evaluated in terms of threat, the increased threat associated with upper-limb tendinopathy (e.g. negative thoughts and feelings arising due to fear of pain with daily activities, fear of tendon rupture, fear of unemployment) likely amplify the pain experience. We should keep these differences in mind and be careful of applying research findings from the rotator cuff to Achilles tendinopathy.

Fundamentally, a thorough assessment of all contributions to tendon pain should be undertaken when diagnosing and treating tendon injury. In light of the current literature, clinicians should consider the impact that both peripheral and central mechanisms have on tendon injury. Perhaps the most effective means of addressing both contributors is a graded loading program that reduces fear of loading, addresses deficits and incorporates education within the rehabilitation process.

What should physiotherapy for patients with Achilles tendinopathy look like in 2015 and beyond?

Physiotherapy for tendinopathy should move to a thoughtful and planned loading intervention that is tailored to each person’s presentation. Reliance on recipe programs is ineffective and not suitable for clinical practice. Referral for considered interventions should be based on a failure to progress with an adequate conservative program, rather than a knee jerk response to tendon pain. Tendon pain appears to be tissue protecting and interventions that completely remove neural input might be dangerous. Adjunct treatments such as electrotherapy and massage have limited evidence for their effectiveness, however may be used as needed but should never form the basis of a tendon treatment. Furthermore, progression to surgery or invasive procedures should not be based on failure to improve from passive therapies. Patience of, and education for, the person with tendinopathy is essential and should be central to a physiotherapy program.

References


