3 Achilles tendinosis, non-insertional

3.1 Introduction

Achilles tendinosis refers to pain associated with injury (typically overuse injury) of the Achilles tendon, which connects the calcaneus to the calf muscle. The term tendinopathy includes tendinosis (a chronic problem involving micro-tears of the tendon), tendinitis (inflammation of the tendon), and paratendinitis (inflammation of the thin sheath surrounding the tendon). The term tendinopathy, however, is used colloquially to refer most commonly to tendinosis. This article deals primarily with non-insertional achilles tendinosis that occurs 2-6 cm proximal to the tendon insertion on the calcaneus.

3.2 Anatomy

The Achilles tendon attaches the posterior calf muscles – the gastrocnemius and soleus – to the calcaneus. Its action is to actively plantar flex the ankle, and to resist dorsiflexion. There is a bursa interposed between the anterior surface of the tendon and the surface of the calcaneus. The Achilles tendon is thick at its insertion, reflective of the high loads it can withstand, up to 10 times body weight.
Biomechanics

The pathophysiology of tendinosis is not fully understood, but likely relates to poor vascular supply and microtears occurring with overuse and excessive loading. Most of the blood supply to the tendon arises from the richly vascular paratendon sheath. The “watershed” area of the tendon (i.e., the region of diminished blood supply) is 2-6 cm above the insertion point of the Achilles tendon on the calcaneus. This area may be less resilient to repetitive microtrauma, rendering it more susceptible to degeneration and rupture.

Additionally, loading on the Achilles tendon can reach up to 12.5 times body weight during running. Given that tendons require approximately 100 days to synthesize the main structural proteins, frequent microtrauma during chronic and excessive loading may not allow sufficient time for tendon repair. Inadequate repair leads to decreased collagen production and tenocyte death, which renders the tendon more vulnerable to injury. Damaged tendons become calcified, thickened, inelastic, and fibrotic, predisposing them to rupture when sudden shear stress is applied.\textsuperscript{1,2}

Pathogenesis

The exact etiology of Achilles tendinosis is unclear, but the main pathologic stimulus is an overuse injury of multifactorial origin involving several intrinsic factors (foot misalignment, poor flexibility, joint laxity) and extrinsic factors (activity level, type of activity, training technique, footwear, training environment). In general, Achilles tendinosis most commonly develops when athletes abruptly increase their training activity.

As discussed above, microtears heal slowly due to limited vascular supply to the tendon and slow protein regeneration. Hypoxia and ischemia-reperfusion injury, as well as exercise-induced hyperthermia, play a role in the pathogenesis. Recurrent microtrauma occurring with continued training can lead to tendon degeneration. Additionally, tendon that has been stretched repeatedly to >4% of its original length loses elasticity, which supports the idea of overuse in the pathogenesis of the disease.\textsuperscript{1-3}

The difference between tendinosis and tendinitis is a histopathologic distinction:
**Tendinosis** involves non-inflammatory collagen degeneration (thin, frayed, disoriented fibers) within the tendon, patchy hypercellular areas crowded with tenocytes, and scattered vascular ingrowth. Additionally the collagen degeneration is typically characterized as mucoid (increased glycosaminoglycan matrix to collagen ratio) or lipoid (lipid accumulation that disrupts collagen fiber structure). The histopathologic changes are likely secondary to chronic overuse injury.  

**Tendinitis**, on the other hand, is characterized by inflammation associated with acute hemorrhage, granulation tissue, and fibroblast/myofibroblast proliferation – ie, attempts to repair of the tendon after vascular disruption. While also a plausible response to tendon injury, studies have shown that this is rarely the cause of chronic Achilles tendon pain and when present, is associated with partial rupture of the tendon.  

**Paratendonitis** (also known as paratenonitis) is acute edema and hyperemia of the outer layer of the tendon with inflammatory infiltration occurring where the tendon rubs over a bony prominence. Although it can lead to thickening of the paratendon and adhesion to the underlying tendon, it does not necessarily progress to degenerative tendinosis and, thus, will not be considered further in this article. For distal Achilles tendinosis, it is possible that bone has contributed to pathogenesis, including tendinosis. One should not conclude that tendon rubbing over bony prominence causes degeneration.  

Stop-and-go sports, cold weather training, foot misalignment, and poor running mechanics have been associated with Achilles tendinopathy. Although Achilles tendinopathy can occur in non-athletes, the incidence of tendon overuse injuries is highest in those who partake in strenuous activity such as running and jumping. Kvist studied the epidemiology of Achilles tendon disorders in competitive and recreational athletes. He found that 66% of 698 patients had Achilles tendinopathy; running was the main sports activity in 53% of patients who presented with an Achilles tendon disorder. Other observational studies have shown that competitive athletes have a 24% lifetime incidence of Achilles tendinopathy, with an even higher incidence — 40-50% — in competitive runners. Achilles tendinopathy accounts for 6-17% of all running injuries among recreational runners.  

Additional risk factors related to microvascular changes that are implicated in the pathogenesis of the disease include diabetes, obesity, hypertension (for women), and use of oral steroids. Fluoroquinolone use has been identified as a risk factor for tendonopathy and tendon rupture; however, the mechanism of this association is not clear. Additionally, one allele of the alpha 1 type V collagen (COL5A1) gene has been associated with increased incidence of chronic Achilles tendinopathy.  

Finally, the prevalence of Achilles tendon diseases is higher with older age and male gender. The peak age for Achilles rupture is 30-40, when degenerative changes and continued or occasional high stress from sports combine. Achilles rupture is also 4-5 times more common in men compared with women.  

### 3.5 Clinical Presentation  

The acute phase of Achilles tendinopathy generally presents as swelling, edema, crepitus, and tenderness approximately 2-6 cm proximal to the tendon insertion. The clinical history may provide important clues such as recent increase in training regimen, history of excessive supination, increased speed or hill training, or improper or worn-out footwear. In chronic cases, exercise-induced pain is the primary complaint. The pain is typically described as burning and is worse at the beginning and end of a training session, with less discomfort in between. Tender, nodular swelling are additional indicators of chronic tendinosis. Of note, however, tendinosis may be subclinical/asymptomatic until it presents as a rupture.
3.6 Differential Diagnosis

The differential diagnosis of posterior heel or calf pain is extensive.\(^2,\)\(^10\)

Associated with overuse, microtear, or excess loading:

- Achilles tendinosis
- Achilles tendon rupture
- Ankle sprain, the most common misdiagnosis of an Achilles tendon rupture. Ankle sprain typically results from landing as opposed to pushing off with the foot, as in an Achilles tendon rupture.
- Stress fracture (calcaneal, tibial), especially in athletes involved in sports requiring running and jumping. Pain typically begins with onset of activity and does not subside.
- Retrocalcaneal bursitis

Conditions to consider include:

- Plantar fasciitis, the most common cause of heel pain in adults, especially first thing in the morning or upon standing after prolonged sitting
- Tarsal tunnel syndrome, heel pain associated with paraesthesias
- Posterior impingement syndrome, impingement between the tibia and talus; most painful in plantarflexion
- Sever’s disease (calcaneal apophysitis), typically in young overweight boys

Additional causes of prolonged heel pain not associated with overuse or microtears:

- Heel pad atrophy, especially in older, obese patients
- Hematogenous osteomyelitis
- Osteoarthritis, septic arthritis, rheumatoid arthritis, sero-negative arthropathies Reiter’s syndrome, rheumatic fever
- Bone cyst
- Bone tumor

3.7 Physical Examination

On physical exam, inspection of the region of the Achilles tendon for bruising, swelling, or foot misalignment can help identify fracture, sprain, or tendon rupture. Inspection of the patient’s footwear and assessment of the patient’s gait and running mechanics may help identify potential causes of tendinosis.

Palpation of the tendon for tenderness, thickening, or defect may help localize the pain above the insertion point (likely tendinosis) versus at the insertion point (more likely calcaneal bursitis, strain at the tendon insertion, or insertional tendonitis, often with calcification of the tendon). Palpation has a sensitivity of 73% and specificity of 89% in detecting a partial tendon tear. The presence of crepitus on palpation during dorsiflexion and plantarflexion also suggests tendinopathy, while a decrease in strength of the muscle suggests other diagnoses.\(^11\)
If tendon rupture is a concern, the Thompson test (calf-squeeze test) is more reliable than the patient’s ability to plantar flex or walk. This test is 96% sensitive and 93% specific. The Matles test (assessing foot position when the patient lies prone with knees flexed to 90 degrees – the foot should appear plantar-flexed) is less sensitive and specific than the Thompson test.\(^\text{11}\)

### 3.8 Imaging

Achilles tendinopathy is a clinical diagnosis and imaging is not recommended to diagnose the condition, except to rule out other conditions such as fracture or tendon rupture.

X-ray can help to rule out fracture or to potentially show calcification that would be consistent with tendinopathy, calcaneal bursitis, or insertional pathology.\(^2\) Ultrasound can assess tendon function and appearance by detecting tendon thickening, hypoechogenicity (relating to the increase in glycosaminoglycans within the matrix), disordered fibers (altered collagen structure), tissue gaps (microtears), and fluid.\(^12\)

Similarly, tendon rupture can be diagnosed solely on clinical exam, but MRI can be used to confirm abnormalities when rupture is suspected. Tendinopathy may present as increased T2-weighted signal on MRI (increased glycosaminoglycans or fluid within the microtears) or an increase in the tendon diameter (tendon thickening secondary to degenerative changes). Additionally, MRI may show alternate causes of the patient’s symptoms such as calcaneal bursitis.\(^13\)

A study of patients with symptomatic Achilles tendinopathy showed that ultrasound identified abnormalities in 65% of the patients, while MRI detected abnormalities in 56%, highlighting the role of history and physical exam in the diagnosis of the disease.\(^14\)

### 3.9 Conservative Treatment

Treatment of acute Achilles tendinopathy includes rest, ice when symptomatic, a short 7-10 day course of NSAIDS, and supporting the tendon with a heel lift or ACE bandage. There is little evidence behind the treatment of acute tendinopathy. NSAIDs have not been shown to promote faster return to activity, but have been useful for short-term pain management. Glucocorticoid injections have been used to improve pain in the short-term, but studies have not shown functional or symptomatic improvement after 12 weeks; there have also been reports of tendon rupture after glucocorticoid injection. Once healing has begun, physical therapy, deep-friction tissue release (massage), and ultrasound have been shown to reduce symptoms over the course of several weeks.\(^12\)

Treatment of chronic Achilles tendinosis also includes eccentric exercises (application of load during muscle lengthening). Somewhat paradoxically, these exercises have been shown to decrease pain and shorten the time to return to sports. Long-term ultrasound of patients who underwent eccentric rehabilitation has demonstrated normalization of the tendon. Presumably by building up a functionally stronger tendon, the patient can decrease the effective load on the injured part of the tendon, giving it a better chance at recovery. It is not known whether these exercises serve a preventative role prior to injury.\(^15,16\)
Finally, there are several less well studied options for treatment of chronic tendonopathy, including heel lifts, orthotics, braces, low-level laser therapy, platelet-rich plasma injection, shock wave therapy, and topical nitrates. Night splints and glucocorticoid injections are not recommended given studies showing a lack of efficacy. Given the supportive evidence behind eccentric exercises compared to these other treatment modalities (especially for recreational athletes and those with mid-portion tendinosis), it seems logical that these options may be used as adjuvant treatment to eccentric rehabilitation in the initial phase of treatment.²

Prevention of Achilles tendon injury is aimed at reducing the risk for runners and other athletes and includes the following:²

- Warm-up before training.
- Avoid cold weather training or wear appropriate running attire if cold weather training is unavoidable.
- Avoid hard or slippery running surfaces.
- Increase training in gradual increments (distance/frequency/duration), increasing by no more than 10% per week.
- Replace running shoes before the sole breaks down or cushioning is lost.
- Ensure proper running mechanics
- Address imbalances in muscle strength or flexibility.

Proper warm-up, avoiding slippery and hard running surfaces, and increasing training intensity gradually have been shown to decrease Achilles injury in various studies. All of these options are clearly cost-effective measures that can be feasibly implemented in a training regimen. Orthotics, stretching, and weight loss to reduce strain in obese patients have not been shown to reduce the incidence of tendon injury in clinical studies.

3.10 Operative Treatment

For refractory cases of chronic tendonopathy or for high-level athletes who do not show as much benefit from eccentric rehabilitation, there are other options.

- Sclerosing injections have been used in high-level athletes to target the painful vessels and nerves on the ventral side of the damaged Achilles tendons.
- Similarly, a mini-surgical scraping procedure targets the blood vessels and nerves on the ventral tendon.
- Longitudinal tenotomies can be done, either open or percutaneous and with or without ultrasound guidance. However, there is inadequate evidence to recommend these procedures.
- Excisional surgery is an option for defined intra-tendon lesions. Surgery for tendinosis involves excising the degenerated portions of the tendon and any osteophytes, or in some cases, using another tendon such as the plantaris or flexor hallucis longus to add strength to the Achilles tendon.¹⁷

3.11 Outcomes

Treated Achilles tendinopathy has a favorable prognosis; however, complications occur when athletes resist the idea of diminished physical activity or when tendinopathy is asymptomatic until it presents in the late stage as an Achilles tendon rupture.
An 8-year follow-up study of 107 patients with less than 6 months of untreated Achilles tendinopathy showed that 84% had full recovery of their activity level and 94% were asymptomatic or had only mild pain with strenuous exercise. Despite this, on ultrasound and clinical examination, researchers noted a clear difference between the affected and unaffected ankles. Additionally, 29% of patients required surgery for their Achilles tendon within the 8 year follow-up period.\textsuperscript{18}

A study of long-term prognosis of eccentric calf-muscle training for chronic Achilles tendinosis showed promising results of decreased pain and improved performance that were greater at 4 years' follow-up than immediately after treatment despite no further active treatment. Eccentric training for Achilles tendinopathy has not been shown to have any complications for tendinosis; however, risk of tendon rupture is an important possibility for partially torn tendons. Ruling out a partially torn tendon is imperative before beginning the rehabilitation program.\textsuperscript{15}

Despite promising initial short-term data, sclerosing injections do not appear to have a favorable long-term prognosis according to a retrospective study of patients who underwent treatment — 53% of patients who had received the injections sought other conservative or surgical treatments after the injection therapy because of continued symptoms. Women were 3.8 times more likely to have unsatisfactory outcomes with the injection treatment than men.\textsuperscript{19}

Arthroscopic surgery to debride the ventral neovascularized area carries the risk of infection, wound complications, and Achilles rupture. These risks are lower for arthroscopically assisted procedures compared to open procedures. Studies have shown complication rates of about 7-11% following open surgery, with complications most commonly including wound infections, hematomas, suture abscesses, fibrotic reactions, and sural nerve irritations.\textsuperscript{20}

As mentioned above, glucocorticoid injections have been associated with Achilles tendon rupture in case reports. While this is not high-quality evidence, steroids are not recommended given the potential risk of this complication.\textsuperscript{21}

### 3.12 References

3. Maffulli N, Kader D. Tendinopathy of the tendo Achilles. JBJS. 2002 Jan;84(1).