The Achilles tendon

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Abstract

The Achilles tendon is subject to two main pathologies: rupture and tendinopathy. The anatomy and biomechanical properties of both the Achilles and the plantaris tendon have a role to play in these. The histology in tendinopathy is primarily non-inflammatory. The available treatment options are eccentric stretching exercises, injection therapies, extracorporeal shock wave treatment, endoscopic, percutaneous or open surgery.

The Achilles tendon is the most commonly ruptured tendon and the incidence of rupture is increasing. The injury can be treated with functional rehabilitation or surgery, which may be open, endoscopic or percutaneous.

Keywords Achilles tendinopathy; Achilles tendon; Achilles tendon rupture

Anatomy and histology

Heading anatomy

The Achilles tendon (TA) is not found in the great apes and its presence is the hallmark of bipedal man.

It originates from three muscle bellies. The medial and lateral heads of gastrocnemius, which originate above the knee at the medial and lateral condyles of the femur, cross the knee separately and fuse into a single muscle belly in the superficial posterior compartment of the leg. The gastrocnemius usually contains mostly fast twitch type muscle fibres. The medial head is broader and longer than the lateral head.[1] The third muscle belly, soleus, lies in the deep posterior compartment and originates from the tibia. It is the main ankle plantar flexor and makes the largest contribution to the Achilles tendon. It consists primarily of slow twitch type muscle fibres.[1]

The plantaris is a separate musculo-tendinous unit, found between the gastrocnemius and soleus, and usually inserts at a point anteromedial to the Achilles insertion. It is a vestigial muscle and is absent in 8% of individuals.[1]

The Achilles calcaneal insertion is not a single point. In addition the fascicles rotate as they move distally, so that the medial head of gastrocnemius fibres attach on the inferior facet of the calcaneal tuberosity, the lateral fibres of the gastrocnemius on the lateral part of the middle facet and the fibres from the soleus on the medial part of the middle facet.[2] The retrocalcaneal bursa lies anterior to the tendon, over the superior facet of the calcaneal tuberosity. (See Figure 1). There is some continuity, formed by the fibres coming from the medial head of the gastrocnemius, between the insertion of the TA and the plantar fascia, which reduces with age.[2]

The tendon has no true synovial sheath, but is surrounded by a paratenon, which is vascular. Blood supply comes from the musculo-tendinous junction, the osteotendinous junction and the surrounding soft tissues. It is thought the midportion of the Achilles is the least well vascularized.[1] A thin layer of fluid separates the paratenon and epitenon and facilitates gliding.

Histology and mechanical properties

Like other tendons, the TA is an anisotropic material composed of a hierarchical structure of fascicles, fibres and fibrils. The fibrils are crimped when unloaded and straighten under load. This crimping accounts for the nonlinear "toe" region observed in the stress–strain curve seen when loading the tendon (see Figure 2). The tendon is composed primarily of type I collagen (90%) and elastin (2%).[3] During activities the TA can bear loads in excess of 3500 N.[3]

The plantaris tendon is stiffer and stronger than the TA and has been implicated in the pathogenesis of non-insertional Achilles tendinopathy. Tethering between the plantaris and the medial aspect of the TA may initiate the tendinopathic process.[4]

Tendinopathy

Achilles tendinopathy can be subdivided into disorders of the Achilles insertion, including insertional tendinopathy, Haglund's deformity, and retrocalcaneal bursitis, and disorders of the midportion of the Achilles, including non-insertional Achilles tendinopathy and paratendinopathies.

Non-insertional Achilles tendinopathy

Pathology: the pathology is thought to be primarily one of degeneration and failure to repair microdamage. In the early phase there may be an acute inflammatory response, in particular within the paratenon, but the histology in the chronic phase

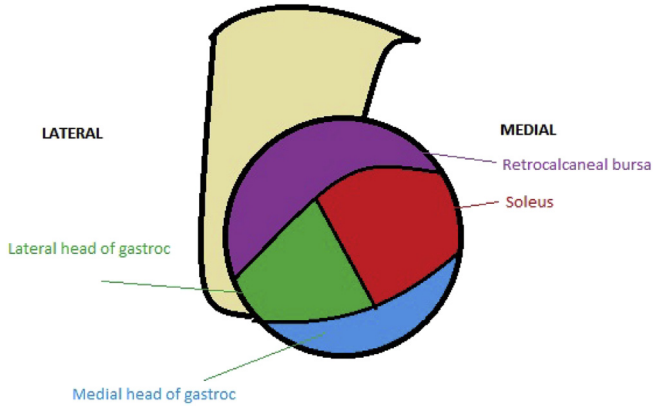

Figure 1 Diagram of the TA footprint on the posterior tuberosity of the calcaneum.

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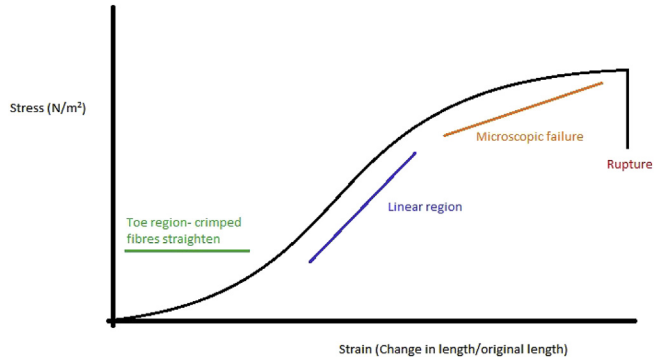

Figure 2 Stress–strain curve.

is marked by the absence of inflammatory cells. There is degeneration of the collagen, both mucoid and lipoid, with disorientation and thinning of the fibres. An angioblastic response is frequently seen, with scattered and randomly oriented blood vessel ingrowth.[5] Neo-innervation accompanies the neovascularization. There is an increase in interfibrillar glycosaminoglycans. There are many factors involved in this process, notably matrix metallo-proteinases.[6] As mentioned above, the differential movement medially between plantaris and the TA may contribute to the initiation on the pathology.

Conservative treatment:

- Non Steroidal Anti Inflammatories (NSAIDs) and corticosteroids

NSAIDs have been found to be useful in relieving pain in particular in the acute setting, but the process is primarily degenerative rather than inflammatory, there is a concern the underlying pathology may in fact be worsened.[6] Steroid injections, again, have been reported as giving short-term relief, but there is a significant concern around complications, notably tendon rupture, and as such are not generally recommended.[5,7]

- Stretching regimes

There is good evidence to support eccentric stretching exercises,[6] both in reducing symptoms and in normalizing the appearance of the tendon on ultrasound, with a reduction in the degree of neovascularization seen. Of note, eccentric exercises have been shown to be superior to concentric exercises at 12 weeks, but the classically described heel drop regime is time consuming and intensive.[8] After five years follow up, just under 40% of patients were entirely pain free, although some ongoing improvement in pain can be seen after the first three months.[9] The mechanism of action is not clearly known, and the peak tendon forces and length have not been found to be different in eccentric *versus* concentric exercises.[10] The addition of a night splint has not been found to of benefit.[11]

- Extracorporeal Shock Wave Therapy (ESWT)

Low energy ESWT has been used to treat a wide range of tendinopathies and has been found to be effective in both insertional and non-insertional tendinopathy, especially when combined with an eccentric stretching programme.[12] ESWT produces micro cavitations within the tissues. As the cavitations collapse, energy is released, and this stimulates a biological response. This has both a direct effect on the nerve endings, with reductions in the amount of local substance P found in the tissues, and an effect directly on the tendon itself, with changes to local mediators such interleukins, metallo-proteinases and insulin like growth factor 1.[1]

- Platelet Rich Plasma (PRP)

PRP has been used in the treatment of several tendinopathies. Chronically tendinopathic TA shows a distinct lack of inflammatory cells and the rationale is that activated platelets will release cytokines to promote a healing response.[6,7] PRP has been used, in particular in combination with eccentric stretching exercises, however a recent Cochrane metanalysis failed to find conclusive evidence for effectiveness either in Achilles or other tendinopathies.[13]

- Glyceryl Trinitrate (GTN) patches

There is conflicting evidence with regards the effectiveness of GTN patches, with some studies suggesting significant and sustained reduction in pain while others found no benefit. It is believed to work by stimulating collagen production by fibroblasts,[14] but nitric oxide, which is the active drug released by GTN patches, is also thought to be involved in the tendinopathic process, so this remains controversial.[6]

- High volume injections and prolotherapy

As neovascularization is often found in the tendinopathic TA, and can be demonstrated on ultrasound studies, high volume (40–50 mls) normal saline injections have been used with the therapeutic aim of stripping the neovascularization and accompanying nerves from the tendon. Studies are confounded by the fact that some injections contained steroid,[15] and some the addition of aprotinin, which has since been withdrawn due to safety concerns regarding it's use in cardiac surgery.[16] Finally hyperosmolar dextrose, which is injected into the abnormal tendon areas, aims to stimulate an acute inflammatory response. Some early benefits have been found but long-term results were similar to eccentric stretching alone.[17]

Surgical treatments: on the whole, non-operative treatment is the norm for non-insertional tendinopathy, but where conservative measures have failed, normally after a minimum of 3–6 months, surgical management can be considered. The surgical goals are debridement of degenerate areas within the tendon substances and division of any fibrotic adhesions to the paratenon, while reconstructing the tendon where needed. Open and percutaneous procedures are described.

- Open debridement

The patient is positioned prone or lateral decubitus and the TA exposed via a posterior or postero-medial approach with thick skin flaps. Adhesions between the paratenon and tendon are released and any areas of degenerate tendon debrided. If debridement of more than 50% of the tendon or excision of a segment of tendon is required, augmentation or reconstructive techniques must be considered, such as those used in chronic TA ruptures (see below). This can either be harvested through the existing incision or through a separate plantar medial incision to obtain maximal length. Post-operative regime usually includes a short period in a splint followed by a rehabilitation protocol in a protective boot.

Complications of surgery are reported in a large series to be in the region of 10%, with over 50% of those relating to wound healing difficulties.[18] At seven months follow up, a prospective