Achilles tendinopathy: pathology and management strategies

As patient expectation is rising, surgery is being considered more frequently for Achilles tendinopathy. Traditional open interventions have a significant morbidity, rising interest in less invasive techniques. This review discusses some of the current concepts and controversies in the management and pathology of tendinopathy of the Achilles tendon.

Achilles tendinopathy is becoming an increasing problem in the industrialized nations, partly because individuals are participating in more sporting activities and partly because they are doing this later in life. Conservative measures have traditionally been effective, but more and more patients require invasive interventions. This, together with recent advances in the understanding of its pathophysiology, has resulted in a lower threshold for surgery for this condition.

Anatomy

The Achilles tendon is the largest tendon in the body and is formed by the three heads of the triceps suri muscle: the gastrocnemius forms the two superficial heads and inserts into the dorsal aspect of the tendon, it acts to plantar flex the ankle as well as flexing the knee and is used in fast movements (the 'speed' muscle). The soleus, in contrast, is attached to the deep surface, does not affect knee movements and is a powerful plantarflexor at the ankle (the 'power' muscle).

Different parts of the tendon have different vascular supplies. Zantop et al (2003) studied the vascular density throughout the tendon using antibodies to a component of the basement membrane. They concluded that the proximal part has the highest capillary density which was supplied via a recurrent branch of the posterior tibial artery. The distal part of the tendon was supplied by the rete arteriosum calcaneare, via the fibular and posterior tibial arteries. The least vascular area was in the mid-portion where the predominant vessels arise from the ventral paratenon.

The tendon is attached to the postero-inferior aspect of the calcaneus rather than the proximal part of calcaneal tuberosity. There are two bursae near the tendon insertion site: one bursa rests in between the tendon and the proximal part of the tuberosity known as the retrocalcaneal bursa. This was a constant finding in 40 specimens examined by Kachlik and colleagues (2008) who defined the retrocalcaneal bursa as a 1–2 cm long synovial fold beginning on the upper wall of the bursa and distally interposed between the ventral surface of the Achilles tendon and the posterior surface of the calcaneal tuberosity. A second bursa is located superficial to the tendon and below the skin.

Pathology and terminology

Clain and Baxter (1992) classified Achilles tendinopathy into two subtypes of insertional and non-insertional tendinopathy.

Insertional tendinopathy is at the site of tendon insertion into the calcaneus. It may be secondary to mechanical irritation by a prominent calcaneal tuberosity (Haglund's deformity). Patients may present with a swelling at the insertion ('pump bump') which may cause problems with shoe wearing. There may be inflammation of the retrocalcaneal bursa (retrocalcaneal bursitis). X-rays may demonstrate calcaneal spurs or calcific tendinosis at the insertion.

Non-insertional tendinopathy occurs proximal to the tendon insertion and may be confined to the paratenon (paratendinitis) or within the tendon (tendinosis) or a combination of both. Patients present with pain at the mid-substance that may be associated with a fusiform swelling. The prognosis for surgical treatment is better if tendon degeneration has not occurred. Puddu et al (1976) proposed three stages of peritendinitis, peritendinitis with associated tendinosis or tendinosis alone.

Although in early peritendinitis an increase in inflammatory cells is observed in the paratenon, Astrom and Rausing (1995) noted a distinct lack of inflammatory cells within the degenerate tendon associated with collagen disarray and a poor healing response. It is because of this lack of inflammation that the term tendinitis is not a suitable description for this condition and its use should be avoided (Khan et al, 2002).

The degenerate tendon also demonstrates neovascularization; a process of infiltration of new vessels and nerves into the area of degeneration (Alfredson and Cook, 2007). As well as the Achilles tendon this phenomenon has been demonstrated in various sites around the body including tendinopathy of the patella, supraspinatus, flexi carpi ulnaris and even the tibialis posterior

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tendons (Knobloch, 2008). There is currently a belief that neovascularization and the accumulation of pain-producing neuropeptides is the cause of discomfort in Achilles tendon disease (Alfredson and Cook, 2007; Knobloch, 2008). A number of new treatments are now aimed at disturbing this phenomenon.

Clinical evaluation

Non-insertional tendinopathy

Diagnosis is usually clinical by demonstrating pain at the mid-substance of the tendon, either by direct palpation or by provoking pain with tendon-loading activities such as a single or repeated heel raise. Pain may be associated with a fusiform swelling and crepitus. A calf squeeze test must be performed to rule out a rupture.

Imaging can be performed if the diagnosis is in doubt. Ultrasound demonstrates areas of hypoechoicity while magnetic resonance imaging may show a high signal area over the degenerate tendon on T2 sequences.

Colour Doppler ultrasonography can also be used to assess the area of tendinopathy and is particularly useful in demonstrating areas of neovascularization.

Insertional tendinopathy

Diagnosis is again clinical but patients may require further assessment with radiographs. There is pain and tenderness at the tendon insertion site. There may be a swelling associated with the subcutaneous Achilles bursa (pump bump), and the condition may be bilateral. It is important to obtain radiographs to rule out the presence of a Haglund's deformity or enthesopathy. The prominence of the calcaneal tuberosity is assessed on lateral radiographs using the parallel lines as described by Heneghan and Pavlov (1982) (*Figure 1*) – any prominence of the calcaneal tuberosity beyond the superior line is regarded as a deformity. Insertional tendinopathy may be associated with seronegative arthropathies and the patient should be assessed for systemic features of these conditions. An autoimmune profile and HLA-B27 testing may be appropriate in selected cases.

Figure 1. Lateral view of the calcaneus in a patient with retrocalcaneal bursitis. Parallel pitch lines (solid-white) demonstrate prominence of the tuberosity beyond the superior line (dashed-yellow).

Magnetic resonance imaging may demonstrate a retrocalcaneal bursitis with bone oedema in the prominent tuberosity. The tendon itself may also demonstrate areas of high signal and thickening (*Figure 2*).

Non-surgical treatment

This should be the mainstay of management and can be successful in the majority of cases. There are various non-surgical techniques described, most claiming good success rates in treating this condition.

The mainstay of physical therapy for tendinopathy is eccentric training. This is a lengthening muscle contraction. The sarcomere crossbridges are at their maximal overlap at the beginning of the contraction, therefore the eccentric contraction generates more tension than both concentric and isometric contractions. Alfredson et al (1998) stressed the importance of eccentric training and suggested a model for training which involves no concentric exercise at all and requires the patients to complete the training programme despite pain in the tendon. If pain is not provoked, the load is increased until pain is felt; this is a unique feature of this training regimen and has very good published long-term results.

Corticosteroid injection is another common but controversial aspect of management of Achilles tendinopathy. Many authors believe their use is not justified and evidence for their use lacking (Maffulli and Kader, 2002; Paavola et al, 2002). Animal studies have also shown a deterioration of the tendon's mechanical properties with intratendinous injections (Hugate et al, 2004). There have even been case reports of tendon rupture following steroid injections. However, others have shown improvements in symptoms with peritendinous corticosteroid injections or advocate their use in refractory cases (Fredberg et al, 2004; Alfredson and Cook, 2007).

Other treatments that have been successfully tried include injection of sclerosants (Alfredson and Ohberg, 2005), application of topical glycerol trinitrate (Paolini and Murrell, 2007), deep friction massage, use of a heel lift orthotic, therapeutic ultrasound and even extracorporeal shockwave therapy (Rasmussen et al, 2008) Alfredson and Cook (2007) suggested an algorithm for treating non-insertional Achilles tendinopathy with some recommended timeframes before surgery is considered (*Figure 3*).

Surgical management

Non-insertional tendinopathy

Conservative methods fail to resolve symptoms in around 30% of patients (Paavola et al, 2000a) and surgery may become necessary. When surgery is considered,

open surgical debridement of the Achilles tendon with or without paratenon stripping has traditionally been the gold standard for treating non-insertional tendinopathy with success rates as high as 80% reported in the literature (Leach et al, 1992).

Tallon and colleagues (2001) critically reviewed the available literature and divided the reported surgical techniques into four groups: open tenotomy with removal of abnormal tissue but without stripping of the paratenon; open tenotomy with stripping of the paratenon as well as removal of any abnormal tissue; open longitudinal tenotomies, with or without paratenon stripping; and percutaneous longitudinal tenotomy. They challenged the good results reported by the authors and concluded that the study methods influenced the results.

In fact open surgery of Achilles tendon is associated with relatively high levels of morbidity. In a series of 432 patients who had undergone surgery for chronic Achilles tendon overuse injury there was an 11% complication rate (Paavola et al, 2000b). Complications may include wound breakdown, infection, edge necrosis, hypertrophic scarring and sural nerve injury. *Figure 4* shows hypertrophic scarring in a patient who had undergone an open decompression for non-insertional tendinopathy – this was associated with considerable discomfort when wearing shoes. These high rates have been consistently seen in most studies involving open decompression which has led to increased interest in minimally invasive techniques.

Testa et al (2002) performed an ultrasound-guided percutaneous longitudinal tenotomy and in their published series 75% had an excellent or good outcome. McShane et al (2007) performed a related procedure with the use of a 20 G spinal needle under ultrasound guidance directed to the abnormal area with multiple passes at the tendon nodule. The results are said to be promising but are as yet unpublished. In the authors' institution percutaneous blunt paratenon stripping is performed with good or excellent results in 75% of patients (Naidu et al, 2009).

There has been a report of a case of successful treatment of non-insertional Achilles tendinopathy using gastrocnemius recession alone. At 24 months the patient was said to be asymptomatic with resolution of pathological changes of the tendon on magnetic resonance imaging (Gentchos et al, 2008). The major advantage is the avoidance of any incisions around the tendon at the ankle.

Insertional tendinopathy

Insertional tendinopathy, especially those associated with a prominent calcaneal tuberosity, can be even more refractory to conservative measures. Traditional open decompression using a longitudinal incision involves resection of the prominent tuberosity, complete debridement of the bursa, excision of thickened paratenon, and removal of calcific deposits within the tendon. In one series 90% of patients undergoing an open decompres-

Figure 2. Sagittal magnetic resonance imaging of the ankle demonstrating (a) high signal areas in the tendon insertion and retrocalcaneal bursa on T2 sequence and (b) tendon thickening on T1.

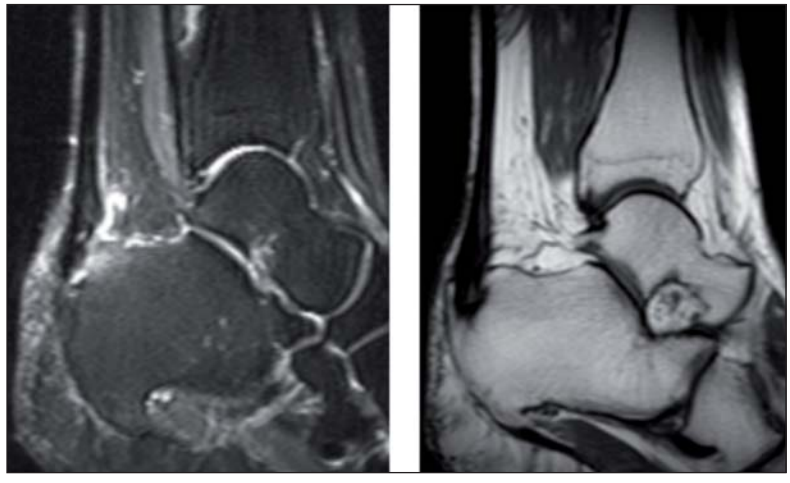
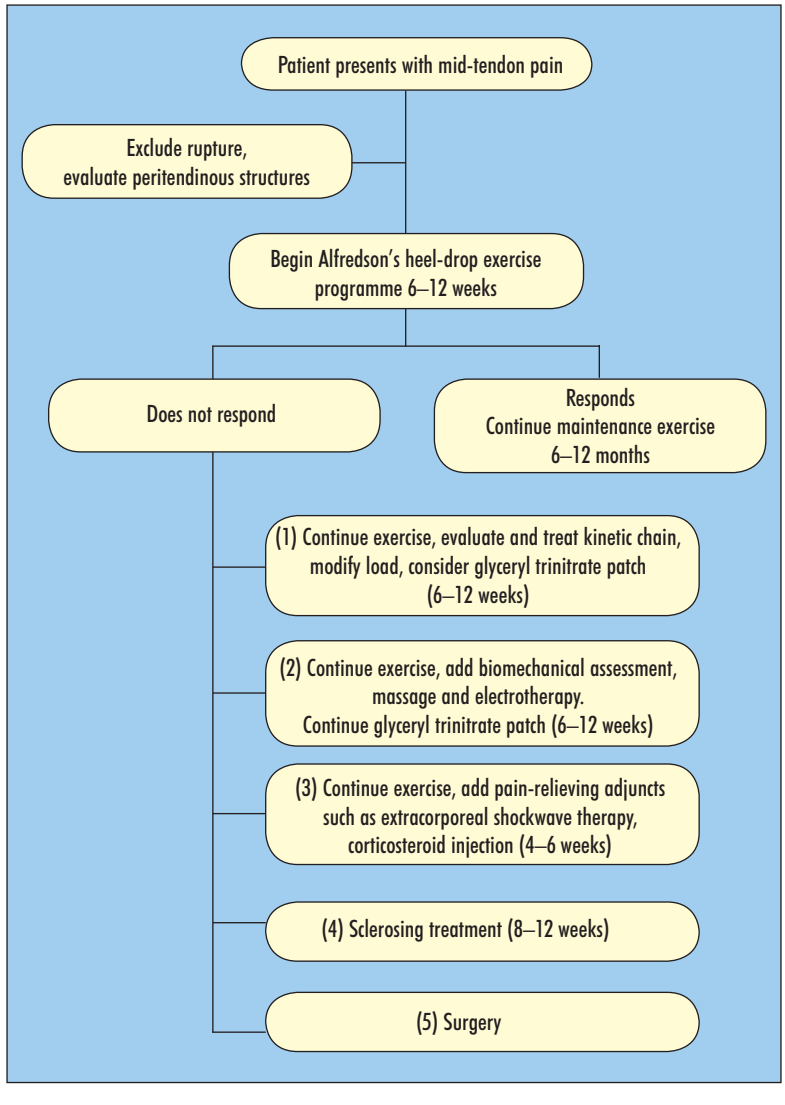


Figure 3. Suggested algorithm for management of non-insertional tendinopathy. From Alfredson and Cook (2007).



sion reported complete or significant pain relief (Yodlowski et al, 2002). Some authors advocate complete detachment of the tendon insertion with reattachment using suture anchors and a proximal tendon lengthening using a V-Y lengthening of the proximal aponeurosis (Wagner et al, 2006). Other reconstruction options include the use of autograft, allograft or augmentation using flexor hallucis longus or plantaris tendons.

A variety of incisions have been described for open decompression procedures. These aim to reduce soft tissue and wound healing complications that are relatively troublesome in this area. Possible incisions include the more traditional longitudinal incisions that can be on the medial or lateral side of the tendon, a longitudinal central tendon splitting approach (McGarvey et al, 2002), or a transverse semi-circular (Cincinnati) incision to access the insertion of the tendon (Carmont and Maffulli, 2007). The latter achieves excellent cosmetic results (*Figure 5*).

The endoscopic technique has gained popularity (Ortmann and McBryde, 2007). In this technique medial and lateral portals are made over the posterosuperior portion of the calcaneus to gain access to the retrocalcaneal space, a small endoscopic shaver can be used to excise the bursa and create a space for visualization of the tendon and underlying bone. A larger motorized burr is then used to remove as much of the prominent Haglund's deformity as required. The latter can be performed under simultaneous image intensifier control to ensure accurate excision of the prominent tuberosity (*Figure 6*). The

Figure 4. Symptomatic hypertrophic scarring complicating open surgical decompression of non-insertional tendinopathy.

Figure 5. Decompression and debridement of a Haglund-associated tendinopathy through a Cincinnati incision. a. The prominent tuberosity is removed with an osteotome after detachment and debridement of the affected tendon and bursa. b. The tendon is then reattached to the exposed calcaneus. c. The skin incision has an excellent cosmetic outcome as seen just after closure and (d) after maturity.

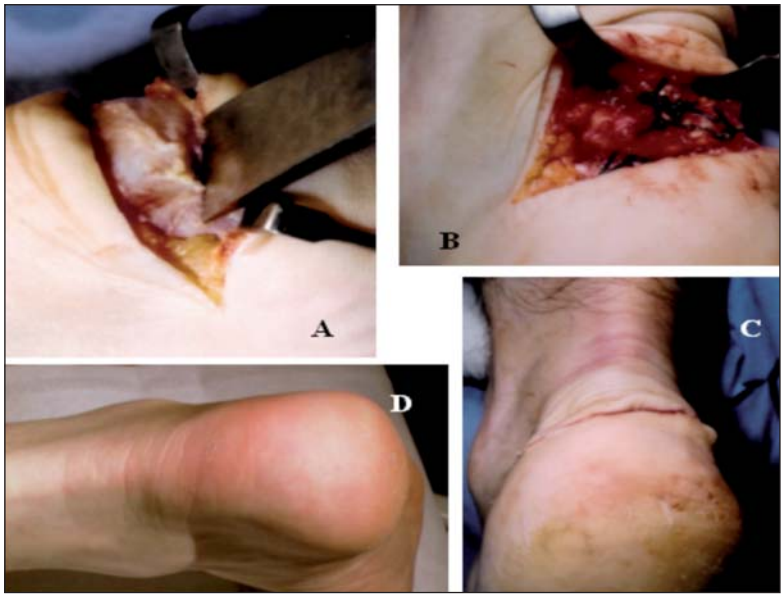
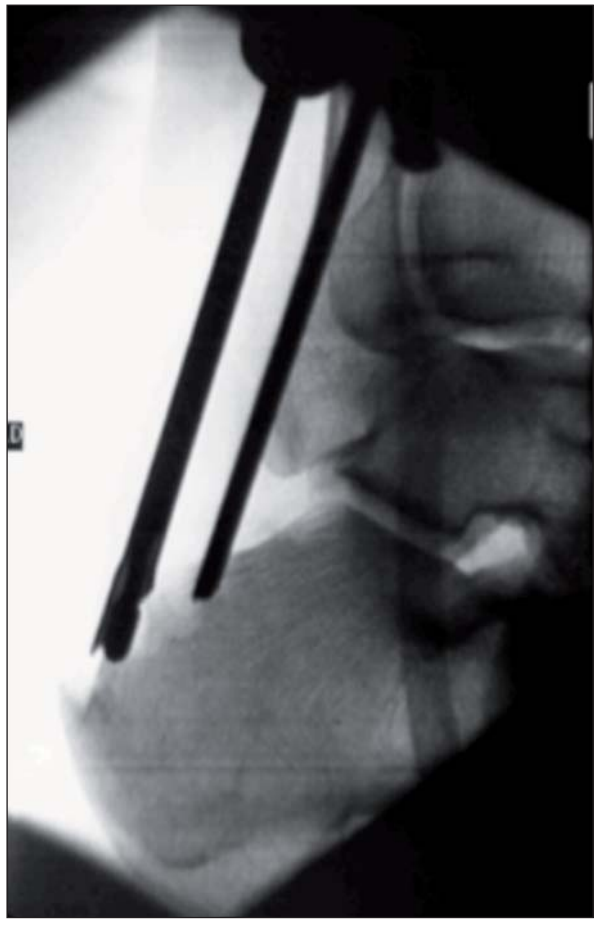


Figure 6. Intraoperative fluoroscopy showing simultaneous use of endoscopy and fluoroscopy to aid excision of a Haglund deformity.



endoscopic technique is associated with a lower incidence of wound problems and faster rehabilitation, but adequate debridement of any intratendinous pathology may not be possible.

Conclusions

Tendinopathy of the Achilles tendon is being encountered with increasing frequency. Although non-surgical management is the mainstay of treatment in up to 80% of sufferers, surgery does become necessary in a significant proportion of cases. Traditional open techniques have enjoyed high success rates but are associated with a relatively high incidence of wound problems. This has resulted in a swing towards minimally invasive methods in the treatment of both insertional and non-insertional types with comparable rates of success. **BJHM**

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KEY POINTS

- Neovascularization and pain-producing neuropeptides have been implicated as the cause of symptoms in Achilles tendinopathy.
- A variety of non-surgical treatment methods are available some with good long-term results.
- A significant proportion of patients will require surgery.
- Less invasive surgical options may have a lower morbidity with similar efficacy to open approaches.