■ INSTRUCTIONAL REVIEW: FOOT AND ANKLE

Achilles tendinopathy

A REVIEW OF THE CURRENT CONCEPTS OF TREATMENT

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The two main categories of Achilles tendon disorder are broadly classified by anatomical location to include non-insertional and insertional conditions.

Non-insertional Achilles tendinopathy is often managed conservatively, and many rehabilitation protocols have been adapted and modified, with excellent clinical results. Emerging and popular alternative therapies, including a variety of injections and extracorporeal shockwave therapy, are often combined with rehabilitation protocols. Surgical approaches have developed, with minimally invasive procedures proving popular.

The management of insertional Achilles tendinopathy is improved by recognising coexisting pathologies around the insertion. Conservative rehabilitation protocols as used in non-insertional disorders are thought to prove less successful, but such methods are being modified, with improving results. Treatment such as shockwave therapy is also proving successful. Surgical approaches specific to the diagnosis are constantly evolving, and good results have been achieved.

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The two main categories of tendo Achilles disorder are classified according to anatomical location, to broadly include insertional and non-insertional conditions.

Non-insertional Achilles tendinopathy

This condition may affect 9% of recreational runners and causes up to 5% of professional athletes to end their careers.[1] The underlying cause is multifactorial and as a result optimal treatment is unclear.

Degeneration of the body of the tendo Achilles has been found in 34% of tendons at autopsy[2] and in 32% of ultrasound-screened asymptomatic individuals.[3] In the acute phase there may be an inflammatory cellular reaction in the peritendineum, with circulatory impairment and oedema.[4] The peritendineum may become filled with fibrinous exudate, perceived as crepitus, and may form adhesions, causing the more chronic condition of paratendinopathy.[4] Injury within the tendon itself will also lead to an initial inflammatory response but an imbalance between matrix degeneration and synthesis may lead to tendinopathic changes. There are many factors implicated in this process, in particular the activity of matrix metalloproteinases (MMPs); specifically, decreased expression of MMP3 is associated with Achilles tendinopathy.[5]

Non-operative management. The mainstay of treatment for non-insertional Achilles tendinopathy is conservative, with initial rest, modification of training regimes, specific exercises and correction of underlying lower limb malalignment with orthoses.[6,7] Most patients will be able to return to previous activities, and in an eight-year follow-up study[8] only 29% failed to respond adequately to non-operative management.

Non steroidal anti-inflammatory drugs (NSAIDs) have been shown to have a modest effect on symptoms,[9] but this was not supported in a randomised study including a placebo arm.[10] The scientific basis of NSAIDs use in chronic tendinopathy is questionable in the histological absence of inflammatory cells in the tendinopathic tissue.[2,11-13] Any short-term benefit is likely to be due to their analgesic effect.[14] Some studies have highlighted the possible detrimental effects of NSAIDs: celecoxib inhibits tendon cell migration and proliferation,[15] and NSAIDs increase leukotriene B, which may contribute to the development of Achilles tendinopathy.[16]

Corticosteroid injections are reported to reduce pain and swelling and improve the ultrasound appearance of the tendon[13,17] but their vasoconstrictive action via prostacyclin, adrenoceptors and inhibition of nitric oxide synthase might be responsible.[18] Corticosteroid injections

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may have some early benefit, but adverse effects were reported in up to 82% of corticosteroid trials[19]; these include tendon rupture,[20-23] and decreased tendon strength is reported in animal studies.[24-27] Any possible benefit of corticosteroid injection appear to be outweighed by potential risks.[28]

Multiple studies and systematic reviews [29-31] have found eccentric exercises to be beneficial in the early treatment of non-insertional Achilles tendinopathy, but the mechanism by which these work is poorly understood. It has been shown that they lead to normalisation of tendon structure, observed on ultrasound, with an apparent reduction in neovascularisation.[32,33] The mechanical loading profile of eccentric exercises has been shown to produce increased stretching of the tendon compared with concentric exercises.[34] Alfredson et al[6] in a randomised study, reported that 82% of patients using eccentric exercises returned to normal activities at 12 weeks, compared with 36% who used concentric exercises, with sustained improvement at 12 months.[35] The 12-week regime is taken as the reference standard for treating non-insertional tendinopathy but six-week programmes have been described, also with reasonable results.[36-38]

Shockwave therapy. Conflicting results have been reported for extracorporeal shockwave therapy (ESWT), usually of low energy, but a recent randomised controlled trial (RCT) demonstrated significant improvement when this was combined with eccentric exercises compared with eccentric exercises alone,[39] and a further RCT reported improved scores after ESWT, particularly in women.[40] A non-randomised case-controlled study of patients failing to improve after at least three non-operative methods for a minimum of six months found improvement at 12 months in the ESWT group.[41] Saxena et al[42] recently reported on 74 tendons that underwent one session of ESWT weekly for three weeks and were followed for one year: 78%of tendons improved, but there was no control group for comparison.

How ESWT works is a matter for speculation, but it is known to cause selective dysfunction of sensory unmyelinated nerve fibres, and changes in the dorsal root ganglia have also been reported.[43] Cavitation may also play a role in interstitial and extracellular disruption leading to a healing response.[44] Changes in transforming growth factor-beta1 (TGF-β1) and insulin-like growth factor-1 (IGF-1) expression and decrease in some interleukins and MMPs have been demonstrated in rats and human cultured tenocytes.[45-48]

A randomised double-blind placebo-controlled study[49] demonstrated improvements in symptoms using glyceryl trinitrate (GTN) patches, and a follow-up study[50] showed continuing benefit for the GTN *versus* the control group three years after treatment (88% *vs* 67% symptom free). A subsequent randomised study[51] demonstrated no additional benefit of GTN patches over standard non-operative management at six months, and histological examination failed to demonstrate any difference in the formation of new blood vessels, collagen synthesis or stimulated fibroblasts between the two groups. In view of the fact that increased nitric oxide levels have been implicated in the development of degenerative conditions, including tendinopathy,[52-55] it has been suggested that in addition to having no benefit on the symptoms of non-insertional Achilles tendinopathy, GTN may in fact be detrimental to the underlying pathological process.[56]

Platelet-rich plasma (PRP) has become widely used in various areas of orthopaedics, with some studies demonstrating improved tendon healing using PRP compared with controls[57-60] but significant improvement in symptoms has not been found when using PRP to treat Achilles tendinopathy.[57,61] A randomised double-blind placebo-controlled study[62] evaluating eccentric exercises and PRP or saline injection showed no difference in improvement in pain and activity at six months, and a recent meta-analysis[63] concluded that although there may be benefit in using PRP to increase the healing strength in tendo Achilles repair following acute rupture, there was no evidence of any benefit in using PRP in the treatment of Achilles tendinopathy.

Small studies[64,65] with limited follow-up have demonstrated reduced pain and improved function following high-volume injections of 10 ml 0.5% bupivacaine and 40 ml normal saline into the paratenon. Chan et al[64] reported symptom improvement in 30 patients retrospectively reviewed, in the absence of a control group, at a mean of 30 weeks, and subsequently in 11 athletes with a minimum of eight months' follow-up.[65] However, 25 mg hydrocortisone was included in the injection, which may have affected their results, as this has been shown to provide early symptomatic improvement but is associated with a higher rate of later complications.[19]

Intratendinous hyperosmolar dextrose (prolotherapy) is thought to produce a local inflammatory response and increase in tendon strength, but evidence to support its use is lacking.[66] A pilot study by Maxwell et al[67] demonstrated a reduction in tendo Achilles pain both at rest and with exercise, and in a small randomised study Yelland et al[68] demonstrated improvements in outcome scores by combining prolotherapy with eccentric exercises.

Subcutaneous low-dose heparin aims to reduce adhesion formation, but evidence of its effect is conflicting and some reports suggest it can cause degenerative tendinopathy in rats.[9,69] Aprotinin is a potent MMP inhibitor, and some studies have demonstrated its success in treating Achilles tendinopathy.[70,71] However, in 2008 it was withdrawn because of severe complications with its use during heart surgery.[72] Early reports using polidocanol (a sclerosing therapy used to obliterate the tendon neovascularisation) injected under Doppler ultrasound guidance into the abnormal vessels on the ventral aspect of the Achilles tendon demonstrated significant improvements in pain and function scores.[73-78] However, these were small studies with limited follow-up using a drug that lacked approval by the United States Food and Drug Administration, and authors from other institutions have not reproduced these results.[79] Along similar lines, electrocautery has been investigated, with one study reporting good results in a series of 11 patients followed for six months.[27]

Further non-surgical options. The use of kinesiotape (elastic therapeutic skin tape) as a technique in the treatment of Achilles tendinopathy and a Cochrane database systematic review of deep frictional massage for tendinopathies failed to demonstrate significant benefit.[80,81] Therapeutic ultrasound has been shown to reduce the swelling in the acute inflammatory phase of soft-tissue disorders,[43] and may enhance tendon healing,[82-86] but a systematic review and meta-analysis have failed to demonstrate any benefit of therapeutic ultrasound over placebo for tendinopathy.[87,88] Dorsiflexion night splints are certainly a favoured treatment option for plantar fasciitis, with evidence to support their use,[89] but recent studies have failed to show benefit from their use to treat non-insertional Achilles tendinopathy, whether combined with eccentric exercises or not.[90,91]

Surgical treatment. Conventional surgical treatment has consisted of open release of adhesions with or without resection of the paratenon.[11,92,93] Macroscopic areas of tendinopathy are excised through a central longitudinal tenotomy, and multiple further tenotomies may be used on the surrounding tissue to initiate vascular ingrowth and a healing response.[11,94]

Even after extensive debridement there is normally enough tendon to achieve side-to-side closure of the principal tenotomy site. However, if > 50% of the tendon has been debrided, then augmentation is recommended. Small defects may be covered with a turn-down flap or using the plantaris tendon, but larger defects may require tendon transfer using, for example, peroneus brevis, flexor digitorum longus or flexor hallucis longus.[93,95,96] Success rates for open surgery vary widely,[97] but are generally reported as being between 75% and 100%.[92-95]

Complications are not uncommon, and in a large series of 432 consecutive patients Paavola et al[96] reported wound necrosis in 3%, superficial infection in 2.5% and sural nerve injury in 1%, with further complications including haematoma, seroma and thrombosis, leading to an overall complication rate of 11% and re-operation in 3%. Minimally invasive techniques may reduce the risks associated with open surgery while maintaining or improving the success rate. Multiple stab wounds with a scalpel may be performed under local anaesthetic, creating longitudinal fissures in the tendinopathic area previously identified with ultrasound. Maffulli et al[97] reported good to excellent results in 37 of 48 patients with this technique at a minimum of 22 months' follow-up.

Stripping of the paratenon is thought to remove the neovascularisation and denervate the diseased area of the tendon. A four-incision technique has been developed using a suture passed between them to strip the dorsal and ventral surfaces of the tendon with or without longitudinal tenotomies but no clinical data have yet been published on their results.[98] This technique has been conducted endoscopically with some reported improvement in small numbers of patients.[99-101]

Recent interest has focused on the role of the plantaris tendon, which is known to be stiffer and stronger than the tendo Achillis, and tethering of the plantaris to the medial aspect of tendo Achillis may initiate an inflammatory response and produce a localised tendinopathy.[102,103] Stripping the plantaris in such patients has resulted in good outcomes with endoscopic techniques, with significant reductions in pain and improved function reported with the latter in 11 patients at two years.[104,105] Gastrocnemius lengthening has been reported as beneficial: Dothan et al[106] treated 14 patients and found that 79% were able to return to their previous sporting activities at two years' follow-up with repeat MRI scans showing significant improvements in tendon quality at one year.

Insertional Achilles tendinopathy

Disorders of the Achilles insertion account for around 20% to 25% of tendo Achillis disorders.[107] Predisposing factors are increasing age, inflammatory arthropathies, corticosteroid use, diabetes, hypertension, obesity, gout, hyperostotic conditions, lipidaemias and quinolone antibiotics.[4,108,109] Other factors include genetic susceptibility,[110-112] and extrinsic factors such as increased repetitive loading[1,113,114] or inadequate footwear can contribute, with uneven wear causing excessive subtalar joint movement or poor shock absorption; uneven or sloping surfaces also play a role.[4] Intrinsic hindfoot and lower limb malalignment and altered biomechanics of the subtalar joint in particular can result in micro-tears and tendinopathic changes.[115-118]

A posterosuperior calcaneal prominence, originally described by Haglund,[119] has been associated with tendon attrition, pain and swelling.[120] However, insertional spurs are probably an adaptive process of formation rather than being due to tendon micro-tears or inflammatory changes. The increased surface area secondary to ossification of the fibrocartilaginous insertion is probably protective during increased loading, but is still not particularly helpful in understanding their role in insertional tendinopathy.[121]

The anterior aspect of the insertion is commonly affected more than the posterior aspect in tendinopathy. As the posterior aspect undergoes a higher strain on dorsiflexion, it has been thought that stress shielding and potential under-use phenomenon have a role to play in the aetiology of insertional tendinopathy.[122] However, the fact that fibrocartilaginous endochondral ossification of the insertion is more likely to occur on the anterior stress-shielded side[123] means that the precise role of loading is complex. It may be that the assumption that the material properties are similar in the normal tendon structure means the stress is preferentially taken up in the 'normal' rather than the 'tendinopathic' side. Hence the anterior side is stress shielded.[124]

The retrocalcaneal bursa is lined with sesamoid and periosteal fibrocartilage. During dorsiflexion of the ankle these layers are apposed and the tendon is compressed against the calcaneal prominence. Changes such as synovial fold hypertrophy,[125] calcification of the sesamoid fibrocartilage and cellular degeneration with bursal debris have been demonstrated.[123]

Non-operative, non-invasive therapy. Immobilisation is frequently used in the acute setting to control exacerbating factors, but prolonged immobilisation should be avoided. A rational treatment plan following any immobilisation should involve a gradual integration of reduced load-bearing activities and a monitored physical therapy or stretching regime.[126]

Patients who suffer from insertional disorders often have heel pain on loading. This can be due to ankle dorsiflexion causing retrocalcaneal bursa compression and impingement of the anterior fibres of the tendon.[127] A graduated shoe raise or heel lift can alleviate pressure on the insertion by plantarflexing the heel. This may potentially accelerate healing of a degenerate tendon insertion.[123] Hindfoot malalignment associated with insertional disorders can be corrected by insoles, if thought to be a provocative factor.[118] Correction of eversion and pronation can improve symptoms.[7]

The 12-week eccentric exercise programme described by Alfredson et al[39] is used with considerable success in non-insertional disorders, but two recent systematic reviews[128,129] have shown that a successful outcome following eccentric loading exercise is less likely in insertional disorders, with success rates of 28% and 32% reported.[130-133] This regime may cause the retrocalcaneal bursa to be compressed against the tendinopathic fibres of the anterior aspect of the Achilles tendon.[132] In order to determine whether the effect of ankle dorsiflexion was detrimental in the full-motion eccentric programme, the activities were modified by Jonsson et al,[132] eliminating the ankle dorsiflexion movement by using floor-level exercises only. They found improved outcomes in 67% of cases, compared with 32% for the original activities.

Regular stretching may increase the working length of the muscle–tendon unit and theoretically increase ankle dorsiflexion, although in reality perhaps by only 1°.[134] A recent study used an initial immobilisation for six to eight weeks and a subsequent stretching regime for patients with retrocalcaneal pain. After a mean treatment duration of 163 days (151 to 1012), 88% were satisfied with the results. Diabetes, previous steroid injections, posterosuperior prominences and smoking were associated with poorer results. It is difficult to ascertain whether it was the immobilisation or the stretching that contributed most to the perceived success of the regime.[126]

The increasing evidence in support of ESWT for the treatment of insertional tendinopathy, and the development of smaller, cheaper machines, is leading to an increased use of this method. ESWT can stimulate a tissue response at variable depths under the targeting device.[44] Many applications for the treatment of tendo Achillis disorders use a low-energy therapy (< 0.2 mJ/mm^2) over multiple sessions.[131,135] Another important parameter is the number of impulses emitted per treatment: often up to 2000 impulses in low-energy treatment and 3000 to 4000 in high-energy treatments.[136]

In a double-blind RCT, Rasmussen et al[40] reported improvements in the American Orthopaedic Foot & Ankle Society (AOFAS) score[137] of 70 to 88 in the intervention arm and 74 to 81 in the sham arm ($p = 0.05$). However, that study included both insertional and non-insertional disorders. Furia[138] reported a good or excellent result in 82.9% of ESWT patients compared with 39.4% of conventionally treated patients. Although this was a significant difference, with strict inclusion criteria of insertional tendinopathy only and a standardised ESWT procedure in a large cohort ($n = 68$), the control arm treatment was hugely varied and not clearly stratified. Another RCT compared eccentric exercises with ESWT and found improved results in the shockwave group ($p < 0.002$).[131] A recent systematic review suggested promising results at a minimum of three months' follow-up, but did not stratify the results according to insertional and non-insertional disorders.[139]

Non-operative invasive management. Corticosteroids have been used in isolated cases of retrocalcaneal bursitis,[140,141] but the volume of evidence is lacking. The historic risk of tendon rupture[142,143] probably explains their relatively infrequent use. Prolotherapy essentially irritates the tendon to stimulate a healing response through the release of pro-inflammatory mediators.[144] Hyperosmolar dextrose solution is commonly used, often coupled with an anaesthetic. A study by Ryan, Wong and Taunton[145] is widely quoted, which reported 22 patients treated with a median of five injections (1 to 13) of 1 ml lidocaine plus 1 ml 50% dextrose given once every three to eight weeks. The pain scores were reduced significantly at follow-up over 28 months but satisfaction scales were not used. Only one study using a sclerosing agent on insertional disease is reported, where 11 patients received repeated injections (up to five) and showed a reduction in pain after eight months, with a 73% satisfaction rate.[146] Monto[147] treated 30 patients (eight insertional tendinopathy, 22 non-insertional) with a single injection of 4 ml PRP, with a resultant mean AOFAS score increase from 34 to 88 at 24 months, although two of the eight insertional tendinopathies were classed as treatment failures owing to patient dissatisfaction and eventual surgery. Radiofrequency coblation (a controlled non-heat-driven process to break molecular bonds within tissues and dissolution) was used in 47 cases, 20 of whom had separate stab incisions made over the insertion and a micro-probe inserted into each hole. There was a 6.4% tendon rupture rate and a 15% re-operation rate but no functional outcome data were presented.[148]

Surgical therapy. Calcaneoplasty can be performed endoscopically with retrocalcaneal bursal debridement. This procedure affords small scars, minimal morbidity and a rapid return to activity.[149] Appropriate patient selection requires accurate clinical and radiological assessment and a diagnosis of retrocalcaneal bursitis[150] after exhaustive conservative therapies. The surgical technique has been reported with a variety of modifications and portal placements.[151-154] Surgery can result in good/excellent outcomes in > 75% to 95% of cases, but infrequent complications such as early rupture of the tendon have been encountered.[149,152,155] The increasing popularity of endoscopic treatments has meant that osteotomies such as that described by Zadek[156] and Keck and Kelly,[157] requiring longer immobilisation and rehabilitation, are less frequently seen.

Open surgery is usually reserved for recalcitrant disease in which conservative options have proved unsuccessful. The surgical plan must include debridement of the degenerate insertion, decompression of bursal tissue, resection of the bony prominence, reattachment of the insertion as required, and/or augmentation of the tendo Achilles with a tendon transfer/graft. Caution should be exercised in patients who are smokers, diabetic or who have peripheral vascular disease.[158,159] Numerous incisions are described, including longitudinal tendon splitting,[160-162] medially based,[93,163] Cincinatti/transverse[164] and laterally based.[165,166] There appears to be no significant advantage of one over another in published series.

Biomechanical and clinical data suggest that 50% of the tendon attachment can be safely debrided with minimal risk of re-rupture.[162,167] Reattachment can be carried out using bone anchors/screws or trans-osseous sutures. Recent mechanical data suggest that single- or double-row repairs result in similar peak loads to failure[168] but in complete detachments the footprint of the insertion can probably be better restored if double-row techniques are used. As long as proper reattachment is performed the tendon should maintain normal plantarflexion.[169] Despite debriding up to 70% of the insertion, Nunley et al[161] did not routinely augment and still achieved 96% satisfaction with good function at seven years. Reattachment has been described using a variety of methods.[160,162,170-173]

The most frequently used method of reconstruction method is augmentation using flexor hallucis longus (FHL), as it is in action during the same phase of gait, is in close proximity, has good vascularity with its low-lying muscle belly, and is the second strongest plantarflexor.[174] Both single- and double-incision techniques are described. The potential pitfall of the single-incision technique is the shorter length of tendon retrieved.[175,176] The double-incision technique carries added morbidity with dissection around the neurovascular bundle in the midfoot. More minimally invasive FHL harvesting techniques have been reported.[177]

Peroneus brevis transfer has been described,[178] although a major concern is increased ankle instability and the development of foot inversion. Flexor digitorum longus (FDL) transfer is used, but is significantly weaker than the FHL (by approximately 50%)[174] and its new course could cross the tibial nerve. Alternative autografts include patellar bone/quadriceps or patellar tendon, but this can cause morbidity of the knee joint[179,180] or hamstrings.[181] V/Y advancement of the gastrosoleus aponeurosis can bridge insertional defects > 2 cm. Wagner et al[163] compared advancements to simple debridements and reported no functional differences but those requiring advancements had more extensive disease.

A recent systematic review describes a rate of minor complications of 20% and a 3.1% rate of major complications.[129] Wound infection rates vary from 0% to 13% but most series report on a mix of insertional and non-insertional reconstruction.[161,164,171,179,182,183] The largest study[96] reviewed 432 patients and found that 4.7% of the insertional group had wound complications, including significant local necrosis.

In two systematic reviews, Wiegerinck et al[128] quoted 89% overall satisfaction, and Kearney and Costa[129] simply mentioned that the studies reviewed report good/excellent outcomes in most. Overall, most studies report satisfaction rates from 82% to 97% and significant improvements in function scores with reasonable follow-up (over four years in some).[161,164,170,171,79,182-184] There is no evidence to suggest that one particular method, i.e. FHL graft or detachment/reattachment *versus* debridement,[163] is superior from published data, but few directly comparative studies exist.

In conclusion, the majority of patients with non-insertional tendinopathy will respond to non-surgical management. Rest may be useful in the acute phase, and a structured course of eccentric exercises in more chronic cases. Paratenon injections and shockwave therapy may have a role. In the 20% to 30% of patients who do not respond surgery may be necessary, where minimally invasive techniques may reduce the risks of complications. Satisfactory outcomes following surgery may be expected in about 85% of patients.

In diagnosing insertional disorders clear distinctions exist between the differing pathologies described, and as a result differing therapies can be instituted. Early use of floor-level eccentric exercises is promising, and shockwave therapy is low risk and has evidence to support its use, but is probably less effective than in non-insertional disease. Endoscopic surgical excision of calcaneal prominences and/or bursae may be beneficial, but the procedure can be technically challenging. Open surgical debridement and reattachment with or without augmentation should be considered after all other treatment modalities have failed, with > 80% of patients likely to gain significant benefit.

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