

## **RESEARCH REPORT**

### **Achilles tendonitis**

#### **Background**

This literature review was carried out in response to a query in clinical practice. A patient with chronic Achilles tendon pain was referred to be considered for treatment with steroid injection. The patient was a 54 year old male nurse who had a one year history of painful thickened achilles tendon. He had been treated in physiotherapy with active exercises, ultrasound, transverse friction massage and heel raise. He continued to be symptomatic to the point where he was having difficulty at work.

An eccentric training program which has been shown to be effective in the treatment of chronic Achilles tendon pain was suggested rather than an injection. In view of his age and duration of symptoms, the risk of rupture associated with peritendinous injection could potentially result in a worse situation. The literature review was carried out to determine whether this clinical reasoning was evidence based.

#### **Aims of this literature review**

- Identify evidence of the effectiveness of corticosteroid injection in the treatment of chronic Achilles tendon pain
- Identify evidence of any adverse effects
- Identify evidence of effectiveness of alternative treatment strategies
- Aid future decision making

#### **Introduction**

The achilles tendon is a common site for injury and rupture (Pufe et.al 2001). Symptoms include posterior heel pain with stiffness before, during and after exercise. The tendon is sore and thickened impairing gait (Koenig et.al 2004, Alfredson 2002). Chronic symptoms are usually of gradual onset (Ohberg et.al 2004).

Very little is known about the pain mechanisms theories include overuse and degeneration (Alfredson et al 2002, Tasto et al 2003).Tendon, bursa and bone, alone or in combination may be the source of pain (Ohberg and Alfredson 2003). Malalignment of the rear foot leading to functional overpronation has been proposed as a possible cause (Koenig et al 2004).

Overuse especially among athletes has been suggested as a possible cause and not just in elite athletes. Symptoms are common among recreational athletes in the 35–50 age group but can be caused by less strenuous activities

or may develop without any obvious cause (Alfredson et.al 2002, Ohberg et.al 2004).

Painful Achilles tendons or spontaneous rupture can also be associated with spondyloarthropathies (Jebaraj and Rao 2006).

Tendon disorders can be demonstrated on ultrasound or MRI (Alfredson 2002). Structural changes on ultrasonography or magnetic resonance imaging are referred to as tendinosis. The cause and pathogenesis are not known, and it can be a difficult condition to treat (Ohberg et.al 2004).

As a result of increasing knowledge regarding the pathology of painful tendons the terms tendinosis or tendinopathy are considered to be more appropriate than the term tendonitis (Alfredson et al 2002). However it is still debated whether Achilles tendon pain is an inflammatory or a degenerative condition. This is reflected in the terminology where Achilles tendonitis and Achilles tendinosis describe the same condition and reflect different opinions upon aetiology (Koenig et.al 2004).

### **Pathology**

There are no signs of chemical inflammation in chronically painful Achilles tendons. Pathology is linked to degradation of collagen and hypercellularity (Alfredson et al 2002). Normal tendons are characterized by a well-organized collagenous fibrillar network sparsely interspersed with fibroblastic cells and vascular structures. Tendons experiencing tendinosis contain no inflammatory cells but exhibit changes in the collagen fiber ultrastructure (Tasto et al 2003 Alfredson 2002, Richards et.al 2005) with irregular fibre arrangement and a high concentration of glycosaminoglycans (Alfredson 2002). Local hypoxia, repetitive microtrauma or impaired wound healing may also contribute to tendonopathy. (Richards et.al 2005)

Ohberg et.al (2004) showed that tendons with chronic tendinosis had significantly higher concentrations of an excitatory neurotransmitter but not prostaglandins when compared with normal tendons, emphasising the lack of chemical inflammation involved in the chronic stage of this condition.

Lack of inflammatory tissue in surgical specimens has been cited as evidence of degenerative aetiology. However Koenig et al (2004) point out that surgical specimens are often obtained late in the disease therefore not excluding an inflammatory component in the earlier stages.

The vascularisation of tendons is relatively sparse compared to muscles. There are relatively few cells available for oxidative metabolism resulting in a low circulatory and metabolic response to loading (Alfredson et al 2002). Microtrauma or degeneration in the Achilles tendon may precede its rupture (Pufe et.al 2001, Richards et al 2005). In the majority of cases rupture is apparently spontaneous with absence of suitable precipitating trauma. Both chronic pain and rupture most frequently occur 3-6 cm above the calcaneal insertion, an area that has been shown to be hypovascular in normal tendons (Pufe et.al 2001, Alfredson 2002). It has been hypothesized that the lack of

vascularity compromises the nutrition required by tendon cells (Pufe et al 2001), making it more difficult for those cells to synthesize the extracellular matrix necessary for repair and remodelling of fatigue-damaged tendon (Tasto et al 2003).

However research has also shown that under resting conditions the blood flow is evenly distributed in the normal Achilles tendon and that peritendinous blood flow to the zone of the tendon with the highest incidence of injuries and painful conditions, is unaltered by age during exercise. Factors other than peritendinous blood flow may therefore be involved in the increased incidence of injuries and painful conditions in the mid-portion of the Achilles tendon among middle-aged individuals (Alfredson et al 2002)

Vascular proliferation has been demonstrated histopathologically at the site of spontaneous Achilles tendon ruptures suggesting some preceding degeneration or microtrauma. Subsequent vascular proliferation contributes to the repair and remodelling process but may mechanically weaken the tendon (Pufe et al 2001).

Alfredson et al (2002) suggest neovascularisation might be involved in the pain mechanisms associated with tendinosis. They studied painful chronic Achilles tendinosis, using ultrasonography and colour Doppler techniques. Neovascularisation in direct relation to the ventral side of the widened tendinotic tendon was found. No neovascularisation was seen in pain free controls. It is not known whether neovascularisation is a response to a primary injury or a metabolic disorder.

Ohberg and Alfredson (2003 and 2004) used ultrasound, colour Doppler imaging, immunohistochemical analysis of tendon biopsies and results of sclerosant injections to indicate an area of neovascularisation could be a mechanism in chronic Achilles tendon pain and suggest structural tendon changes associated with high levels of glutamate and lactate may trigger neovascularisation.

Alfredson et al (2002) found Achilles tendons with tendinosis had mean concentrations of lactate significantly higher than the concentrations of lactate in the normal tendons and suggest the higher lactate concentrations could act as a nociceptive pain stimulus in the tendon. Lactate concentrations in painful tendons at rest were of similar levels to those found in other studies post exercise. They conclude anaerobic conditions might be expressed as higher lactate levels.

Hypoxia induced by higher lactate levels may lead to neovascularisation and chronic painful tendinosis. Whether ischaemia precedes the start of tendinosis, or whether the tendinotic changes in the tendon give rise to ischaemia and the possible association with pain is not known. However lactate stimulates chemosensitive group afferents and is likely to be a nociceptive pain stimulus (Ohberg and Alfredson 2004).

## **Imaging**

According to Shalabi et al (2004) magnetic resonance imaging (MRI) and ultrasound (US) are the methods of choice when evaluating achilles tendon lesions, facilitating choice of treatment and follow up of morphology during the healing process. MRI allows discrimination of normal from pathological structures, observation of the internal substance of the tendon and sequential imaging of the healing process. It is superior to other imaging techniques in the detection and evaluation of intratendinous changes which are described in terms of tendon widening and altered tendon tissue appearance. Imaging can detect tendon matrix adaptation and can be used in addition to clinical outcome measures to evaluate the morphological effects of different treatments. Enhanced MRI is thought to be more sensitive than ultrasound in chronic Achilles pain identifying areas of enhancement in areas of the Achilles tendon that appear normal on ultrasound (Richards et.al 2005)

Ohberg et.al (2004) reported patients with painful thickening at the 2–6 cm level in the achilles tendon had localised widening, focal hypoechoic areas, and irregular structure corresponding to the painful area when assessed with grey scale US. Thickened retrocalcaneal bursa, calcifications, bone spurs and loose fragments have also been identified using US and colour Doppler in patients with chronic Achilles tendon insertional pain ( Ohberg and Alfredson 2003)

Koenig et.al (2004) recommend colour Doppler over grey scale US in diagnosis, location and follow-up of Achilles tendonitis. Findings on grey-scale US remain present even after symptoms have resolved and it is not possible to distinguish between active tendonitis and changes caused by a previous attack.

Colour Doppler is a method to study flows in blood vessels. It demonstrates only high level flows therefore normal TA blood flow is not shown (Ohberg and Alfredson 2004) but the areas of hyperaemia within a tendon referred to as neovascularisation can be identified. Colour Doppler findings are quantifiable and fluctuate with degree of pain. It is able to demonstrate abnormal activity inside the tendon, define its location and is able to grade the activity.

Intratendinous hyperaemia identified on colour Doppler by Koenig et.al (2004) was also found to respond to intratendinous glucocorticoid injection which may indicate an inflammatory component.

## **Treatment**

Numerous treatments for tendinosis have been described and include rest, activity modification, NSAIDs, corticosteroid injections, physical therapy, surgical debridement, ESWT (extracorporeal shock wave therapy), eccentric muscle training, local peritendinous glucocorticoid injections bracing and heel inserts (Tasto et.al 2003, Koenig et.al 2004). Common treatment objectives are to limit tissue injury and stimulate a healing response (Tasto et al 2003).

Torn rotator cuff tendons exhibit a decrease in growth factors that act as angiogenesis markers (Tasto et al 2003). The authors therefore suggest any

treatment modality that stimulates local blood supply and addresses the deficit of angiogenesis be beneficial in the treatment of tendinosis. Surgery is reserved for cases where these treatment strategies have failed (Koenig et.al 2004).

It is debated whether Achilles tendon pain has an inflammatory component, however glucocorticoid injection around the tendon is a common therapeutic procedure and the general recommendations are that injections should be administered peritendinously, while intratendinous injections are advised against for fear of rupture (Koenig et.al 2004).

Koenig et al (2004) used ultrasound Doppler to identify areas of vascular activity which were mainly within the tendon in patients with achilles tendon pain. Glucocorticoid was injected directly into the visual changes, i.e., intratendinously using US guidance. Dosage was 1 mL of 40 mg mL<sup>-1</sup> methylprednisolone acetate (Depo-Medrol) mixed with 0.5 mL lidocaine 1%. The patient was instructed to rest the leg for one day and to refrain from any strenuous activity for 2 weeks. All patients experienced a flare-up within the first 24 h after injection after which symptoms declined. All patients were symptom-free after 2 months.

On colour Doppler all patients initially had intratendinous hyperaemia. No peritendinous hyperaemia was found. Intratendinous hyperaemia disappeared in all but one patient during follow-up. Based on the results of this preliminary study Koenig et.al (2004) suggest when there is indication for glucocorticoid injection in achilles tendon pain the injection should be given into the area with hyperaemia (intratendinously) rather than at some distance to it (peritendinously). No ruptures were encountered but only 6 patients took part in this preliminary study.

Koenig et.al (2004) conclude that the dramatic effect on the hyperaemia inside the tendon seen after a single glucocorticoid injection supports an inflammatory background to these symptoms. They also claim intratendinous hyperaemia is identical to the inflammatory hyperaemia seen in the synovium in arthritis and tenosynovitis which also responds to glucocorticoid injections.

Glucocorticoid is also, a vasoconstrictor and this effect may explain the change in vascularity and the accompanying fall in pain if pain is coupled to hypervascularity. Therefore the anti-inflammatory effect may not necessarily be the mechanism by which improvement was gained with intratendinous glucocorticoid injection (Koenig et al 2004).

In a pilot study Ohberg and Alfredson (2003) used sclerosing injections under ultrasound guidance to areas of neovascularisation in painful achilles tendons destroying the vessels and accompanying nerves. They were able to show clinical improvement but no changes in tendon thickening or structure with short term follow up. This preliminary work suggests that neovessels and accompanying nerves may be the source of chronic Achilles tendon pain. The authors also speculate that sclerosant therapy may destroy parts of the normal circulation to the tendon and for this reason recommend sclerosant injections be performed locally outside the tendon and in the lowest possible

concentration. No adverse effects were reported from the 150 injections carried out. Further randomised study is necessary to investigate this treatment approach.

Treatment by heavy load eccentric calf muscle training has shown good clinical results, with pain abolished during a 12 week training period and a return to previous activity level (Ohberg et.al 2004, Ohberg and Alfredson 2004). However the mechanism by which these effects are achieved is not known.

In long term follow up of patients with chronic Achilles tendon pain undertaking eccentric calf muscle training Ohberg et.al (2004) found that tendon width had decreased significantly. In addition before treatment all patients had hypoechoic areas and an irregular tendon structure demonstrated on ultrasound. At follow up there were no hypoechoic areas and the tendon was of normal, regular structure. The authors suggest eccentric training regimen may therefore induce a response that normalises the concentrations of glycosaminoglycans and possibly also enables normalisation of the fibre arrangement, resulting in decreased tendon thickness.

Using Colour Doppler Ohberg and Alfredson (2004) were able to demonstrate areas of neovascularisation disappear following eccentric training and that passive dorsiflexion of the ankle stops the flow in neovascular vessels. They theorise repetition of this exercise damages neovessels and may explain an initial increase in pain experienced by patients participating in eccentric exercises for the treatment of chronic Achilles tendon pain. Structural abnormalities that remained at follow up also seemed to be associated with residual pain in the tendon (Ohberg et.al 2004)

### Conclusions

There is no evidence in the literature reviewed that chemical inflammation is a mechanism in chronically painful Achilles tendons however an inflammatory component cannot be excluded in acute pain.

Vascular proliferation has been demonstrated histopathologically at the site of spontaneous Achilles tendon ruptures suggesting some preceding degeneration or microtrauma

The fact that intratendinous hyperaemia identified on colour Doppler was found to respond to intratendinous glucocorticoid injection may indicate an inflammatory component. However glucocorticoid is also a vasoconstrictor and it has been suggested that the change in vascularity and reduction in pain may be due to an effect on the hypervascular areas of painful tendons.

Hyperaemia identified on colour Doppler is intratendinous, peritendinous hyperaemia has not been identified suggesting the injection should be given into the area with hyperaemia (intratendinously) rather than at some distance to it (peritendinously). However there are no large scale studies investigating this approach.

This literature review did not identify any evidence regarding an increased risk of rupture following achilles peritendinous steroid injection. However it does raise questions as to how effective peritendinous injection may be, given the pathophysiological processes that have been identified in chronically painful Achilles tendons.

Common treatment objectives are to limit tissue injury, stimulate local blood supply and a healing response. SOM practitioners do have other treatment modalities such as transverse friction massage at their disposal for the treatment of this condition. However there is also a lack of literature to support the use of TFM in the treatment of chronic Achilles tendon pain.

Treatment by heavy load eccentric calf muscle training has shown good clinical results with a reduction in tendon width. In addition a reduction in hypoechoic areas and a more regular tendon structure have been demonstrated on ultrasound following eccentric training. It would be possible for the effects of TFM on the Achilles tendon to be assessed in a similar way using ultrasound.

### **Relevance to SOM practice**

Investigation of the effects of TFM on the chronically painful Achilles tendon using ultrasound would be a valuable project for an SOM member to undertake.

Caution is advised when considering the use of injection therapy for treatment of chronic Achilles tendon pain, despite there being little evidence of rupture post peritendinous injection.

There is some evidence to suggest that intratendinous rather than peritendinous injections may have a more direct effect on pathophysiological changes but no evidence regarding any adverse effects of this approach. It will be necessary to follow up this literature review in order to determine the safest and most effective treatment for patients with chronic Achilles tendon pain.

### **References**

Alfredson H, Bjur D, Thorsen K, Lorentzon R, Sandstrom P 'High intratendinous lactate levels in painful chronic Achilles tendinosis. An investigation using microdialysis technique.' Journal of orthopaedic research 2002 sep 20 (5) p934-8 PMID12382956

Jebaraj I and Rao A 'Achilles tendon enthesopathy in ochronosis' journal of postgraduate medicine 2006 jan-mar 52 (1) p 47-8 PMID 16534167

Koenig M, Torp-Pederson S, Ovistgaard E, Terslev L, Bliddal H 'Preliminary results of Doppler guided intratendinous glucocorticoid injection for Achilles tendonitis in five patients' Scandinavian journal of medicine and science in sports Apr 2004 14 (2) p100-6

Ohberg L, Alfredson H 'Effects on neovascularisation behind the good results with eccentric training in chronic mid portion Achilles tendinosis?' Knee surgery sports traumatology arthroscopy Sep 2004 12 (5) p465-70

Ohberg L, Alfredson H 'Sclerosing therapy in chronic Achilles tendon insertional pain-results of a pilot study' Knee surgery sports traumatology arthroscopy Sep 2003 11 (5) p 339-43

Ohberg L, Lorentzon R, Alfredson H 'Eccentric training in patients with chronic Achilles tendonosis: normalised tendon structure and decreased thickness at follow up' British journal of sports medicine Feb 2004 38 (1) p8-11

Pufe T, Peterson W, Tillmann B, Mentlein R 'The angiogenic peptide vascular endothelial growth factor is expressed in foetal and ruptured tendons' Virchows Archive 2001 Oct 439 (4) p579-85 PMID11710646

Richards P, Win T, Jones P 'the distribution of microvascular response in Achilles tendonopathy assessed by colour and power Doppler' Skeletal radiology 2005 jun 34 (6) p336-42 PMID 15785932

Shalabi A, Movin T, Kristofferson-Wiberg M, Aspelin P, Svennsson L 'Reliability in the assessment of tendon volume and intratendinous signal of the Achilles tendon on MRI: a methodological description' Knee surgery sports traumatology arthroscopy 2005 sep 13 (6) p 492-8 PMID 16170584

Tasto J, Cummings J, Medlock V, Harwood F, Hardesty R, Amiel D ' The tendon treatment centre: new horizons in the treatment of tendinosis' Arthroscopy 2003 Dec 19 Suppl 1 p213-23 PMID 14673441

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