

LITERATURE REVIEW - ACHILLES TENDON

Background

This literature review resulted from a query in clinical practice. A 54 year old male nurse with a one year history of painful thickened Achilles tendon was being considered for treatment with peritendinous steroid injection. 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 it was causing difficulty at work.

Given his age and duration of symptoms injection was advised against due to the likelihood of a degenerate Achilles tendon that would be prone to rupture, potentially resulting in him being in a worse situation. An eccentric training program which has been shown to be effective in the treatment of chronic Achilles tendon pain was therefore recommended rather than an injection.

This literature review was carried out to assess the evidence base for this rationale.

Aims

- 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
- Should the same decision be made again?

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 (US) or magnetic resonance imaging (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 micro trauma 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). Micro trauma 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 micro trauma. Subsequent vascular proliferation contributes to the repair and remodelling process but may mechanically weaken the tendon (Pufe et al 2001)

Alfredson et.al (2002) suggests 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 ischemia precedes the start of tendinosis, or whether the tendinotic changes in the tendon give rise to ischemia 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) MRI and 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 TA 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 retrocalaneal 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 tendon pain. 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 achilles 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 indicating a possible inflammatory component.

Treatment

Treatments for tendinosis can include rest, activity modification, NSAIDs, local steroid injections, physical therapy, surgical debridement, ESWT (extracorporeal shock wave therapy), eccentric muscle training, 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). Surgery is reserved for cases where these treatment strategies have failed (Koenig et.al 2004).

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.

It is debated whether Achilles tendon pain has an inflammatory component, however steroid 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. Injection was carried out directly into the visual changes, i.e., intratendinously using US guidance. Dosage was 1 ml of 40 mg ml⁻¹ methylprednisolone acetate (Depo-Medrone) 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 this study.

Koenig et.al (2004) conclude the dramatic effect on the hyperaemia inside the tendon seen after a single steroid injection is evidence of an inflammatory mechanism. They also claim intratendinous hyperaemia is identical to the inflammatory hyperaemia seen in the synovium in arthritis and tenosynovitis which also responds to steroid 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 steroid 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)

Surgical treatment for tendons that fail to respond to conservative treatment can involve several procedures designed to irritate the tendon and initiate a chemically mediated healing response, ranging from simple percutaneous tenotomy to open removal of tendon pathology (Alfredson and Cook 2007).

Open Z-plasty to lengthen the Achilles tendon is described by Costa and Donall et.al (2006) and they point out that any benefits from surgery must be weighed against a relatively high complication rate for procedures in this region.

Complete detachment of the Achilles tendon, reattachment with suture anchors and proximal lengthening is a method of treatment for severe chronic insertional Achilles tendinosis used by Wagner et.al (2006) and debridement of the tendon insertion without detachment was used for less severe involvement.

Adequate attention to rehabilitation is likely to improve outcomes for those who are surgically treated (Alfredson and Cook 2007).

Summary

The literature reviewed suggests that chronically painful Achilles tendons with degeneration and micro trauma are at risk of rupture. Transverse friction massage is a treatment aimed at limiting tissue injury and stimulating a healing response by encouraging local blood supply and this is consistent with treatment aims recommended in the literature reviewed. In addition heavy load eccentric calf muscle training has shown good clinical results as well as reducing abnormal tendon pathology.

Neovascularisation appears to be an important pathological mechanism and there is no evidence for an inflammatory mechanism in chronic Achilles tendon pain. There was evidence that intratendinous treatment with injection has an effect on the neovascularisation without increasing the risk of rupture and is therefore more likely to be effective than peritendinous injection. This must however be considered with caution due to the small numbers of patients treated in this way. There was no literature giving an assessment of any increased risk of rupture associated with peritendinous injection.

Conclusion

There is a lack of clarity from the literature reviewed regarding the most effective approach and any increased risks associated with injection. Therefore the use of an eccentric training program for this patient, which has been shown to be both safe and effective appears to be an appropriate evidence based approach.

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