Understanding and managing Achilles tendinopathy

Achilles tendinopathy is common among athletes participating in racquet sports, track and field, volleyball and soccer. Greater participation in recreational and competitive sporting activities has lead to an increased incidence of Achilles tendinopathy in recent years. However, Achilles tendinopathy does not exclusively affect athletes. This article reviews the aetiology and management of Achilles tendinopathy.

The Achilles tendon is a confluence of the gastrocnemius and soleus muscles. The soleus muscle lies deep to the gastrocnemius muscle, arising from the posterior surface of the upper tibia. The tendon inserts on the posterior surface of the calcaneus distal to the posterior-superior calcaneal tuberosity. The Achilles tendon is not encased in a true synovial sheath, but is surrounded by paratenon composed of a single layer of cells. This tissue is richly vascularized, and is responsible for a significant portion of the blood supply to the tendon (Carr and Norris, 1989). This supply comes through a series of transverse vincula, which function as passageways for blood vessels to reach the tendon. The Achilles tendon also receives blood from vessels originating at the musculotendinous and osteotendinous junctions.

At about 12–15 cm proximal to its insertion rotation of the tendon begins, becoming more marked in the distal most 5–6 cm of the tendon. The tendon spirals approximately 90°, with the medial fibres rotating posteriorly and the posterior fibres rotating laterally. Angiographic injection techniques have demonstrated a zone of hypovascularity 2–7 cm proximal to the tendon insertion (Carr and Norris, 1989).

Healthy tendons are brilliant white, with a fibroelastic texture. Within the extracellular matrix network, tenoblasts and tenocytes constitute about 90–95% of the cellular elements of tendons and lie between the collagen fibres along the long axis of the tendon (Kirkendall and Garrett, 1997).

Biomechanics

Tendons transmit force generated by muscle to bone and act as a buffer by absorbing external forces to limit muscle damage. Tendons exhibit high mechanical strength, good flexibility, and an optimal level of elasticity to perform their unique role (Kirkendall and Garrett, 1997). The tensile strength of tendons is related to thickness and collagen content, and a tendon with an area of 1 cm^2 is capable of supporting 500–1000 kg (Shadwick, 1990).

Aetiology and pathophysiology

Tendon injuries can be acute or chronic and are caused by intrinsic or extrinsic factors, either alone or in combination. In acute trauma, extrinsic factors predominate. Overuse injuries generally have a multifactorial origin. Tendon vascularity, gastrocnemius–soleus dysfunction, age, gender, body weight and height, pes cavus and lateral ankle instability are common intrinsic factors. Excessive motion of the hindfoot in the frontal plane, especially a lateral heel strike with excessive compensatory pronation, is thought to cause a 'whipping action' on the Achilles tendon and predispose it to tendinopathy (James et al, 1978).

Changes in training pattern, poor technique, previous injuries, footwear and environmental factors such as training on hard, slippery or slanting surfaces are extrinsic factors which may predispose athletes to Achilles tendinopathy (James et al, 1978).

Excessive loading of tendons during vigorous physical training is regarded as the main pathological stimulus for degeneration (Selvanetti et al, 1997). Tendons respond to repetitive overload beyond their physiological threshold by either inflammation of their sheath, degeneration of their body, or a combination of both (Benazzo and Maffulli, 2000). It remains unclear whether different stresses induce different responses. Active repair of fatigue damage must occur, or tendons would weaken and eventually rupture (Ker, 2002).

The repair mechanism is probably mediated by resident tenocytes, which continually monitor the extracellular matrix (Sharma and Maffulli, 2005a). Failure to adapt to recurrent excessive loads results in the release of cytokines leading to further modulation of cell activity (Leadbetter, 1992).

Tendon damage may even occur from stresses within physiological limits, as frequent cumulative microtrauma may not allow enough time for repair (Selvanetti et al, 1997).

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The aetiology of tendinopathy remains unclear, and many factors have been implicated. Free radical damage occurring on reperfusion after ischaemia, hypoxia, hyperthermia, impaired tenocyte apoptosis, cytokines, prostaglandins and fluoroquinolones have all been linked with tendinopathy (Sharma and Maffulli, 2005b).

Pain is the main symptom of Achilles tendinopathy, but the underlying mechanism causing pain is not fully understood. Traditionally, pain has been thought to arise through inflammation, or via collagen fibre separation or degeneration (Khan et al, 2000). However, chronically painful Achilles tendons have no evidence of inflammation, and many tendons with intratendinous pathology detected on magnetic resonance imaging (MRI) or ultrasound are not painful (Khan et al, 2000). As tendinopathies are the result of a failed healing response, and are not inflammatory conditions, pain may originate from a combination of mechanical and biochemical causes (Maffulli et al, 2004).

Histopathology

Tendinopathy is characterized by a disorganized impaired healing response and inflammation is not a typical feature. As a result the authors advocate the use of the term tendinopathy as a generic descriptor of the clinical conditions in and around tendons arising from overuse, and suggest that the terms tendinosis, tendonitis and tendinitis be used only after histopathological examination.

Histologically, tendinopathy is characterized by an absence of inflammatory cells and a poor healing response, with non-inflammatory intratendinous collagen degeneration, fibre disorientation and thinning, hypercellularity, scattered vascular ingrowth, and increased interfibrillar glycosaminoglycans (Leadbetter, 1992).

Clinical presentation

Pain is the cardinal symptom of Achilles tendinopathy. Generally, pain occurs at the beginning and end of a training session, with a period of diminished discomfort in between. As the pathological process progresses, pain may occur during exercise, and, in severe cases, it may interfere with activities of daily living. In the acute phase, the tendon is diffusely swollen and oedematous, and on palpation tenderness is usually greatest 2–6 cm proximal to the tendon insertion. Sometimes, fibrin precipitated from the fibrinogen-rich fluid around the tendon can cause palpable crepitation. In chronic cases, crepitation and effusion diminish, and a tender, nodular swelling, the site of the failed healing response lesion, is present.

The diagnosis of the Achilles tendinopathy is mainly based on a careful history and detailed clinical examination. However, diagnostic imaging may be required to verify a clinical suspicion or, occasionally, to exclude other musculoskeletal disorders.

Imaging

Ultrasonography is commonly used to examine tendon disorders. It is readily available, quick, safe and inexpensive. However, ultrasound is operator-dependent, has somewhat limited soft tissue contrast, and is not as sensitive as MRI. In acute paratendinopathy, ultrasound reveals fluid accumulation around the tendon (*Figure 1*).

MRI provides extensive information on the internal morphology of tendon and the surrounding structures, and is useful to evaluate various stages of chronic degeneration. Excellent correlation between MRI and pathological findings at surgery has been reported (Schepsis et al, 1994).

A longitudinal ultrasound study showed that mild-to-moderate changes were observed frequently in both the involved and uninvolved Achilles tendons, but the occurrence of these changes was not clearly related to the patients' symptoms (Paavola et al, 2000). Owing to the high sensitivity of these imaging modalities, an abnormality should be interpreted with caution and correlated to the patient's symptoms before making any management recommendations.

Management

In the early phase of Achilles tendinopathy, various forms of conservative management are used. Seeking medical attention at an early stage may improve outcome, as treatment becomes less predictable when the condition becomes chronic. Surgical management is recommended for patients who do not adequately respond to a conservative treatment programme over a period of 3–6 months.

The initial conservative programme is directed towards presumed aetiological factors or towards relieving symptoms. Most commonly, it consists of a combination of

Figure 1. Axial ultrasound scan demonstrating swelling of the Achilles tendon with surrounding fluid accumulation.

strategies, including abstention from the activities that caused the symptoms, and correction of training errors, foot malalignments, decreased flexibility and muscle weakness (Alfredson and Lorentzon, 2000).

Decreasing the intensity, frequency and duration of the activity that caused the injury, or modification of that activity, may be the only necessary action to control symptoms in the acute phase. Collagen repair and remodelling is stimulated by tendon loading, and complete rest of an injured tendon can be detrimental. Modified rest, which allows activity in the uninjured body parts and reduces activity in the injured site, has been recommended (Alfredson and Lorentzon, 2000).

Cryotherapy has been regarded as the single most useful intervention in the acute phase of Achilles tendinopathy. Cryotherapy is used for its analgesic effect, to reduce the metabolic rate of the tendon, and to decrease the extravasation of blood and protein from new capillaries found in tendon injuries (Rivenburgh, 1992).

Therapeutic ultrasound may also reduce swelling in the acute inflammatory phase and improve tendon healing.

Deep friction massage has been advocated for tendinopathy and paratendinopathy. In chronic cases, this should be accompanied by stretching to restore tissue elasticity and reduce the strain in the muscle–tendon unit with joint motion. Augmented soft tissue mobilization is a non-invasive soft tissue mobilization technique which has been successfully used in chronic tendinopathy patients, probably through controlled application of microtrauma which would increase fibroblast proliferation (Gehlsen et al, 1999).

Stretching and strengthening of the triceps surae muscle and Achilles tendon are important to preserve function of the triceps surae musculotendinous unit by restoring normal ankle joint mobility and decreasing the strain of the Achilles tendon with normal motion. Eccentric training is superior to concentric training in decreasing pain in chronic Achilles tendinopathy, and promising results have been obtained using an intensive heavy-load eccentric muscle training regimen (Mafi et al, 2001).

If there is a foot alignment problem, orthoses that place the hindfoot in neutral may prove beneficial. A heel lift of 12–15 mm is classically used as an adjunct to the management of Achilles tendon pain. Orthotic correction can alter the biomechanics of the foot and ankle and relieve heel pain. In runners, orthotics may result in pain relief in up to 75% of runners with Achilles tendinopathy (Gross et al, 1991).

Several drugs, such as low-dose heparin, wydase and aprotinin, have been used in the management of peri- and intra-tendinous pathology. Although widely used and promising, evidence of their long-term effectiveness is still unclear. Peritendinous corticosteroid injections are controversial, evidence for their effectiveness is missing, and there are no good scientific reasons to support their use.

In 24–46% of patients with Achilles tendinopathy, conservative management is unsuccessful, and surgery is recommended after exhausting conservative methods of management, often tried for at least 6 months (Maffulli et al, 2004). However, long-standing Achilles tendinopathy is associated with poor postoperative results, with a greater rate of re-operation before reaching an acceptable outcome.

The objective of surgery is to excise fibrotic adhesions, remove degenerate nodules and make multiple longitudinal incisions in the tendon to detect intratendinous lesions and to restore vascularity and possibly stimulate the remaining viable cells to initiate cell matrix response and healing. Investigations show that multiple longitudinal tenotomies trigger neoangiogenesis at the Achilles tendon, with increased blood flow (Friedrich et al, 2001). This would result in improved nutrition and a more favourable environment for healing. Patients are encouraged to weight bear as soon as possible after surgery.

Most authors report excellent or good results in up to 85% of cases, although this is not always observed in routine non-specialized clinical practice. It is difficult to compare the results of studies as most studies do not report their assessment procedure. Also, no prospective randomized studies comparing operative and conservative treatment of Achilles tendinopathy have been published, thus most of our knowledge on treatment efficacy is based on clinical experience and descriptive studies.

Conclusions

Although Achilles tendinopathy has been extensively studied, there is a clear lack of properly conducted scientific research to clarify its aetiology, pathology and optimal management. Most patients respond to conservative measures if the condition is recognized early, while continuing the offending activities leads to chronic changes which are more resistant to non-operative management. Teaching patients to control the symptoms may be more beneficial than leading them to believe that Achilles tendinopathy is fully curable. Surgery usually involves removal of adhesions and degenerate areas, and decompression of the tendon by tenotomy or measures influencing the local circulation. It is still debatable why tendinopathic tendons respond to surgery. For example, it is not known whether surgery induces ordered re-vascularization, denervation or both, resulting in pain reduction. It is also unclear how longitudinal tenotomy improves vascularization. As the biology of tendinopathy is being clarified, more effective management regimens may come to light, improving the success rate of both conservative and operative management. BJHM

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KEY POINTS

- The Achilles tendon exhibits a zone of hypovascularity 2–7 cm proximal to its insertion.
- Tendons exhibit high mechanical strength, good flexibility, and an optimal level of elasticity.
- Excessive loading of tendons during vigorous physical training is regarded as the main pathological stimulus for degeneration.
- The aetiology of tendinopathy remains unclear, and many factors have been implicated.
- Pain in tendinopathy may originate from a combination of mechanical and biochemical causes.
- Tendinopathy is characterized by a disorganized impaired healing response and inflammation is not a typical feature.
- Pain is the cardinal symptom of Achilles tendinopathy.
- Achilles tendinopathy is mainly a clinical diagnosis but magnetic resonance imaging or ultrasound may be used for verification.
- Many non-operative treatment modalities have been described, and these are beneficial in the majority of cases.
- Surgery is reserved for cases resistant to non-operative treatment.