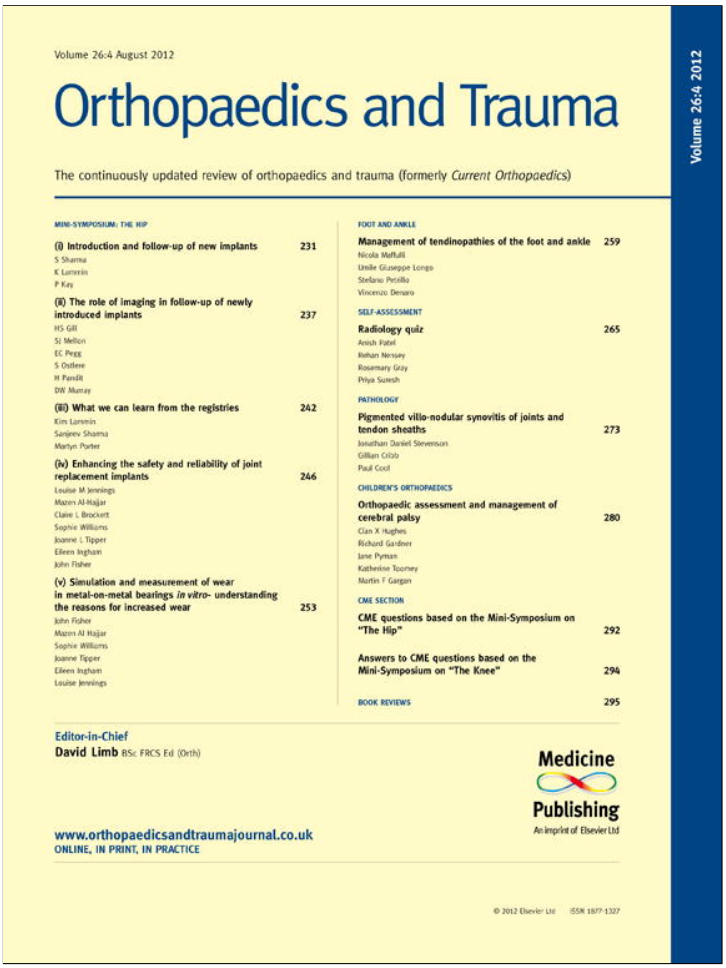

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Management of tendinopathies of the foot and ankle

Nicola Maffulli
Umile Giuseppe Longo
Stefano Petrillo
Vincenzo Denaro

Abstract
Foot and ankle tendinopathies are a significant problem in orthopaedic practice. They represent a failed healing response. Such tendons show increased matrix remodelling, leading to a mechanically less stable tendon which is more susceptible to damage. Diagnosis is based on a careful history and detailed clinical examination and investigations. Initially treatment is by relative rest and modification of physical activity, use of rehabilitative exercises, evaluation and correction of intrinsic and extrinsic causes of injury. Surgical management is indicated if conservative management fails, in stage 3 tendinopathy or if tendon rupture occurs. Randomized controlled trials are awaited to clarify the best therapeutic options.

Keywords Achilles; diagnosis; flexor hallucis longus; foot and ankle; management; peroneal; posterior tibial; tendinopathy

Introduction

Tendinopathy is frequently used to describe overuse tendon injuries in the absence of a pathological diagnosis, and is used to describe what is actually a spectrum of diagnoses including peritendinitis, tendinitis and tendinosis. They are overuse injuries and usually occur when the intensity, or duration of physical activity or athletic training changes in some way. They are not due to persistent inflammation; histological studies of surgical specimens from patients with established tendinopathy consistently show absent or minimal inflammation. The histologically descriptive term 'tendinosis' (a degenerative pathology with a lack of inflammatory change), 'tendonitis' or 'tendinitis' (implying an inflammatory process) should only be used after histo-pathological confirmation.[1]

A physiological response to tendon injury is characterized initially by inflammation (i.e., tendinitis) followed by accumulation and deposition of collagen matrix within the tendon and finally by remodelling (i.e. tendinosis). They generally show hyper-cellularity, a loss of the tightly bundled collagen fibre appearance, an increase in proteoglycan content and, commonly, neovascularization.[2,3] Microscopically it is possible to detect other important pathological changes in the tendon, such as a reduction in the number of neutrophils and macrophages, fibrin deposition and an increase in both collagen breakdown and synthesis. Thus inflammation plays a role only in the initiation but not in the progression and continuation of the disease process. However, an inadequate healing response due to poor blood supply or ongoing mechanical forces on the tendon, or both, may prevent resolution. The resulting tendon tissue consists of a disorganized matrix of hypercellular, hyper-vascular tissue that is painful, weak and unable to respond to functional needs (Figure 1).[4] Thus what may appear clinically as an acute tendinopathy is actually a well-advanced failure of a chronic healing response in which there is neither histological nor biochemical evidence of inflammation.

While several foot and ankle tendons can be affected, tendinopathy of Tibialis posterior, Achilles, Peroneal and Flexor hallucis longus tendons are the most commonly seen. This review aims to summarize the diagnosis and management of those tendinopathies.

Diagnosis

The assessment of a patient with ankle tendinopathy starts with a careful history. Most commonly there is a recent history of trauma, a new sporting activity or an increase in the intensity of physical activity which preceded the onset of symptoms. The pain is commonly of an insidious onset and is localized to the area of the affected tendon. It worsens with sustained activity or on weight bearing. In the early stages of the disease, pain and is not present at rest and decreases after a warm-up period. In the later stages, pain may be present at rest and worsens with any kind of activity. Pain in multiple tendons or joints can represent the manifestation of a rheumatologic pathology, and a joint effusion usually indicates an intra-articular inflammation rather than tendinopathy.

On examination the affected area should be carefully observed to note any asymmetry compared with the unaffected limb and the presence of any swelling, or muscle atrophy. Range of motion is frequently decreased on the affected side. Palpation may reveal tenderness along the affected tendon and always reproduces the patient's pain.

It is important to assess and ultimately treat any intrinsic and extrinsic causes of tendon injury. Important intrinsic factors include flexibility, laxity and strength of the tendon, leg length abnormality, patient age, and vascular supply. Extrinsic factors

Nicola Maffulli *MD MS PhD FRCS (Orth) Centre for Sports and Exercise Medicine, Queen Mary University of London, Barts and The London School of Medicine and Dentistry, Mile End Hospital, 275 Bancroft Road, London E1 4DG, UK. Conflicts of interest: none declared.*

Umile Giuseppe Longo *MD MSc Department of Orthopaedic and Trauma Surgery, Campus Bio-Medico University, Via Alvaro del Portillo, 200, 00128 Rome, Italy. Conflicts of interest: none declared.*

Stefano Petrillo *MD Department of Orthopaedic and Trauma Surgery, Campus Bio-Medico University, Via Alvaro del Portillo, 200, 00128 Rome, Italy. Conflicts of interest: none declared.*

Vincenzo Denaro *MD Department of Orthopaedic and Trauma Surgery, Campus Bio-Medico University, Via Alvaro del Portillo, 200, 00128 Rome, Italy. Conflicts of interest: none declared.*

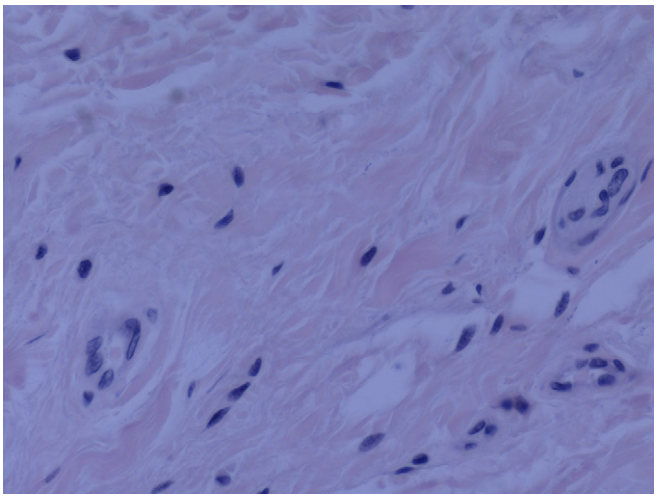

Figure 1 Histopathological features of tendon tissue harvested from a tendinopathic Achilles tendon showing a disorganized matrix of hypercellular, hyper-vascular tissue.

include overuse of the tendon, training errors, medication abuse, smoking and wearing an inappropriate type of shoe (i.e., motion control, cushioning, and stability) for the patient's foot type or other equipment not appropriate for the specific sport or work activity.

Plain radiography should be carried out to exclude bony abnormality and other pathologies. Radiographic findings are usually normal, but may demonstrate calcification of the tendon, osteoarthritis and even undiagnosed fractures. Ultrasonography can be useful to show areas of tendinosis and to demonstrate the dynamic response of the involved tendon. Further imaging is necessary if the diagnosis remains in doubt or if the patient does not respond to conservative treatment. Magnetic resonance imaging is the most useful investigation, especially useful if surgical intervention is being considered as it gives good images of tendon pathology and fatty infiltration (Figure 2).

Treatment – general principles

As tendinopathy is not inflammatory with no overt inflammatory process, there is no rational basis for the use of non-steroidal anti-inflammatory drugs (NSAIDs) in its treatment. They are only useful to reduce inflammation and pain in the acute stage and while acetaminophen and NSAIDs provide short-term pain relief for patients with tendinopathy, they do not affect long-term outcomes. There is no evidence from randomized control trials that NSAIDs are more effective than acetaminophen which should be preferred for pain relief because it has fewer adverse effects. Several studies have demonstrated that NSAIDs do not reduce the time of recovery from tendon injury and may actually interfere with the healing process.[5]

Several treatment options have been proposed for the management of tendinopathy. Regrettably, not only have many therapeutic regimens not been subjected to randomized clinical trials, but it is not known whether a given treatment is applicable to all tendinopathies. That said, treatment is initially based on conservative measures, including relative rest, protection, ice, compression, elevation, medications, and rehabilitative exercise. It is also important to encourage patients to reduce their level of

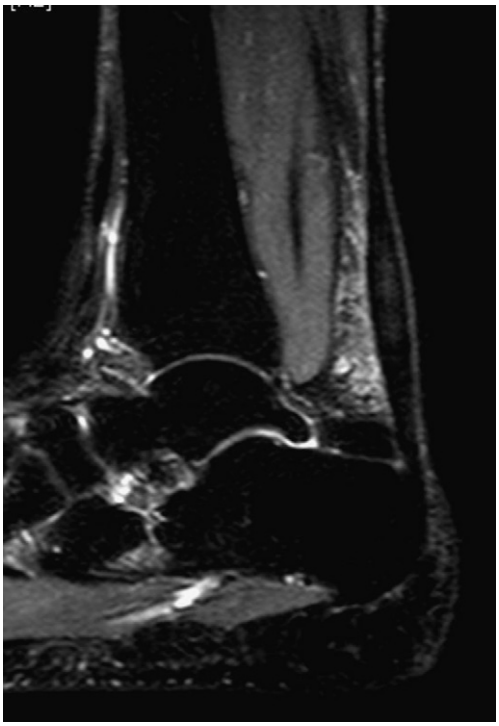

Figure 2 Magnetic resonance imaging showing tendinopathy of the main body of the Achilles tendon.

physical and sporting activity. The duration of relative rest is related to the severity of the injury, the pain and the patient's activity level. Quite early, this should be followed by a stretching and strengthening programme. Eccentric strength training promotes the formation of new collagen and has proved to be effective and is associated with excellent results in the management of Achilles (Figure 3 a–f) and patellar tendinosis, and it may be also useful in other tendinopathies. Overall results suggest a trend for a positive effect of eccentric exercises, with no reported adverse effects.

Orthotics, such as inserts or a heel wedge, are sometimes used to reinforce, protect and help unload the tendon. Other physical therapy options have been used including ultrasound, iontophoresis (electric charge to drive medication into the tissues), and phonophoresis (use of ultrasound to enhance the delivery of topically applied drugs), but there is little evidence of their effectiveness.[6]

In the past decade, many new treatments have been introduced, and developed to manage tendinopathies. These include extra-corporeal shock wave therapy, radio-frequency ablation, percutaneous tenotomy, autologous blood or growth factor injection, prolotherapy, and topical nitrates.[7–22] The use of extra-corporeal shock wave therapy has been approved by the FDA for the treatment of lateral epicondylitis and plantar fasciitis and encouraging results have also been reported in the management of calcifying tendinopathy of the shoulder. Combining eccentric training and shock wave therapy produces higher success rates compared to eccentric loading alone or shock wave therapy alone. Sclerotherapy and the injection of growth factors may improve tendon healing especially in sport practice while the autologous blood injection may reduce pain. These new treatments are

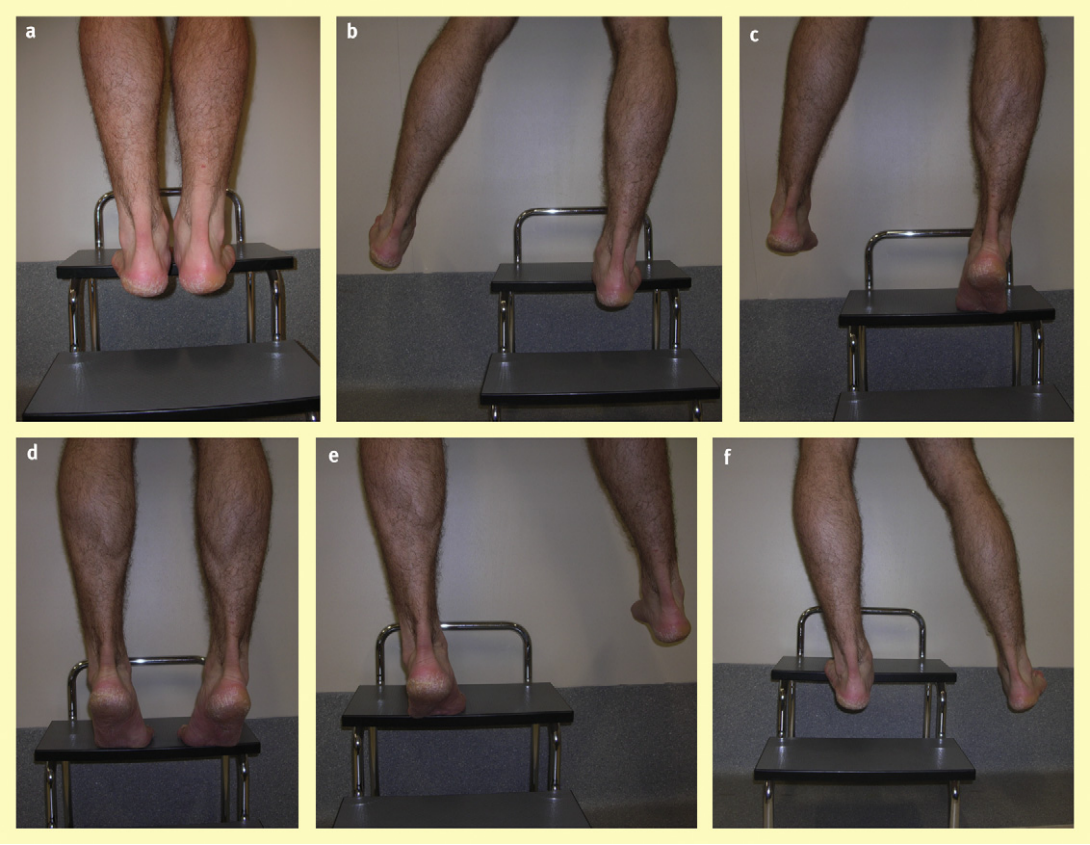

Figure 3 Figures from (**a**–**f**) show the sequence of the eccentric training for the tendinopathy of the main body of the Achilles tendon.

promising but their effectiveness awaits confirmation as do several new treatment options based on tissue engineering.

Very few randomized prospective, controlled trials have been conducted to assist in defining what is the best evidence-based management. There have been trials of non-steroidal anti-inflammatory medications, eccentric exercise glyceryl trinitrate patches, sclerosing injections, ultrasound, shock wave treatment and platelet rich plasma. Recent studies conducted on an off-label use of an FDA-approved medication demonstrate that trans-cutaneous glyceryl trinitrate patches may decrease pain and improve healing in patients with tendinopathy.

Surgery should be considered only if a conservative treatment programme lasting 3–6 months has failed or if a tendon rupture has occurred. Surgery usually aims to excise any abnormal tendon, to release areas of fibrosis or repair a lesion to restore anatomy and/or function of the tendon. Thus fibrous adhesions are excised together with areas of failed healing and multiple longitudinal incisions made in the affected tendon to detect intra-tendinous lesions and to restore vascularity and possibly stimulate the remaining viable cells to initiate cell matrix response and healing.

Specific tendinopathies

Posterior tibial tendon

The tibialis posterior tendon stabilizes the medial longitudinal arch and plantar flexes and inverts the foot. Tibialis posterior tendinopathy occurs more commonly in woman than men, and patients are often older than 40 years. Injuries involving this tendon are frequently associated with the elongation of the hind- and mid-foot ligaments, especially the calcaneo-navicular ligament, leading to a painful flatfoot deformity. Rarely, in severe cases, there is elongation of the deltoid ligament resulting in medial ankle instability. Most posterior tibial tendinopathies are related to an event causing tenosynovitis, such as stepping in a hole, twisting the foot or slipping off a kerb. The condition is commonly misdiagnosed and confused with a medial ankle sprain. However, often there is no history of previous acute trauma.

If flatfoot deformity results, it can progressively increase over months to years and can give rise to pain along the lateral tarsal region. Without adequate treatment, there is a natural progression to worsening deformity, with degeneration of the tendon eventually leading to rupture.

On examination, the commonest clinical examination finding is the “too many toes sign”. When both feet are viewed from behind it appears that there are more toes on the lateral aspect of the involved foot and flattening of the arch. There is excessive pronation of the injured foot and relative weakness of the tibialis posterior. If the patient is asked to stand on tiptoe, the normal heel varus is usually lacking and single leg toe raise may reproduce the patient’s pain. The patient will be unable to do 10 successive toe raises.

The presence of abnormal heel varus or the inability to initiate and maintain plantar flexion with this manoeuvre is usually indicative of rupture of the posterior tibial tendon. Differential

diagnoses includes deltoid ligament sprain, flexor digitorum longus injury, flexor hallucis longus injury, navicular stress fracture and tarsal tunnel syndrome.

Treatment of posterior tibial tendinopathy is based on the severity of the injury and is specific for each stage of the pathology.

- Stage 1. There is no foot deformity, pain and swelling are mild, and provoked by the single leg heel raise test.
- Stage 2. There is midfoot abduction and pes planus deformity, pain and swelling are severe and the patient is unable to perform single-leg heel raise test.
- Stage 3. The subtalar joint is fixed with clinical and radiographic signs of arthritis.

The initial management is usually conservative, commencing with immobilization in a short leg cast or cast boot for two to three weeks, particularly if there is significant pain during walking. A foot orthosis can be helpful to decrease the pathological pronation of the foot. Steroid injections are not indicated because of the associated high tendon rupture rate. If conservative management fails after 3 months or if stage 3 abnormality is initially diagnosed, then surgical treatment is indicated.

Peroneal tendon

Injuries of the peroneal tendon include rupture, tendinopathy, and subluxation, and are frequently associated with chronic lateral ankle pain and lateral ankle instability. Chronic tendinopathy and interstitial tears occur more commonly than complete tear or subluxation. Patients commonly complain of a feeling of ankle instability. Persistent lateral ankle swelling and/or retrofibular pain is often present. A positive peroneal tunnel compression test may be found. This is pain elicited along the posterior ridge of the fibula on active dorsi-flexion and eversion of the foot against resistance. Pain along the lateral foot can occur with a single heel raise or resisted plantar flexion of the great toe. Differential diagnoses include fibular fracture, peroneal subluxation or dislocation and os peroneum syndrome. These are an important group of disorders, particularly the os peroneum syndrome which can cause chronic lateral plantar foot pain.[23] The latter condition can result from a fracture of the os peroneum or entrapment or rupture of the peroneal longus tendon near the os peroneum.[23]

Ankle taping and lateral heel wedges can be useful to unload stress on the peroneal tendon, but there is no evidence that they speed healing. Physical therapy comprises ankle range of motion exercises, peroneal stretching, strengthening and proprioception. Surgical management is indicated if conservative treatment fails, for rupture of the peroneal tendon and recurrent peroneal instability, whether subluxation/or dislocation.

Achilles tendon

Achilles tendinopathy is characterized by pain and swelling in and around the tendon, mainly arising from overuse.[1] In the past three decades, the incidence of Achilles tendinopathy has risen as a result of greater participation in recreational and competitive sports.[24–26] It is a common cause of disability in athletes because of the continuous prolonged and intense functional demands imposed on the Achilles tendon (AT). Achilles tendinopathy is not restricted to athletes: up to one third of people affected are non-athletes; it is common in middle-aged overweight patients with no history of increased physical activity who may lose significant number of working days, with a financial effect on society.[27]

The aetiology is unclear, and many factors have been implicated. Tendon vascularity, gastrocnemius-soleus dysfunction, age, gender, body weight and height, pes cavus, and lateral ankle instability are commonly implicated 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 AT, and predispose it to tendinopathy. Forefoot varus is frequently seen in patients with Achilles tendinopathy. Changes in training pattern, poor technique, previous injuries, footwear and environmental factors such as training on hard, slippery or slanting surfaces are possible pre-disposing extrinsic factors. Overall, excessive loading of the tendon during vigorous physical training is regarded as the main pathological stimulus for tendinopathy,[28] possibly as a result of imbalance between muscle power and tendon elasticity. The AT may respond to repetitive supra-physiological overload by either inflammation of its sheath, or degeneration of its body, or a combination of both.

Degeneration and subsequent rupture of the Achilles tendon is related to peri-tendinitis and tendinosis. The disease process is usually 4–6 cm above the insertion into the calcaneum which is the least vascular area (the vascular watershed). If there is degeneration of the tendon, a nodular area can be palpable. The Thompson test (squeezing the calf belly in the prone patient, which produces plantar flexion of the ankle if the tendon is in tact) is normal in patients with tendinopathy, but in patients with a complete rupture of the Achilles tendon, it is positive. Differential diagnoses includes inflammatory conditions which are always associated with pain, such as retro-calcaneal bursitis and inflammation of the superficial bursa of the Achilles tendon.

Patients should be advised to avoid impact activities, preferring non-impact activities, such as swimming and cycling. Evidence-based support for the management of Achilles tendinopathy is lacking, and patients are at risk of long-term morbidity and clinical outcomes that are unpredictable. Management with eccentric strength exercises does improve symptoms of Achilles tendinosis, reducing the degenerative process, and should be initiated early in treatment. Physical therapy modalities including ultrasound treatments, ionto- and phonophoresis, and gastrocnemius-soleus complex/soleus stretching can be helpful. Steroid injections are not indicated; such injections should be restricted to the treatment of inflammation of the retro-calcaneal bursa as several authors[29,30] have shown that the use of steroids around the Achilles tendon increases the risk of rupture. Results of Platelet Rich Plasma (PRP) injections have been disappointing.

In 24–45.5% of patients with AT, conservative management is unsuccessful. Surgery is recommended after at least 6 months of conservative methods of management. Surgical options range from simple percutaneous tenotomy (which can be ultrasound-guided), to minimally invasive stripping of the tendon, to open procedures (Figure 4). If, during an open procedure, more than 50% of the tendon is debrided, consideration should be given to either tendon augmentation or transfer.

There are many anecdotal reports of excellent or good results in up to 85% of cases. In a systematic review,[31] most of the

Figure 4 Open tenotomy of the Achilles tendon.

articles on surgical success rates have reported successful results in over 70% of cases. Overall, surgery in athletes appears to give better results as does surgery on male patients. However, the articles that reported success rates higher than 70% often had poorer methods scores. As there is a lack of trials of surgical management of AT, the high success rate reported by some authors should be interpreted with caution, particularly as such a relatively high success rate is not always observed in clinical practice.

Post-surgical rehabilitation should focus on early motion and avoid overloading the tendon in the initial healing phase. A period of initial splinting and crutch walking is generally used to allow pain and swelling to subside. After 14 days, patients are encouraged to start daily active and passive ankle range of motion exercises. The use of a removable walker boot can be helpful during this phase, and early weight bearing is encouraged. Generally weight bearing is not limited according to the degree of debridement needed at surgery, but patients who have undergone extensive debridement and tendon transfers may require protected weight bearing for 4–6 weeks postoperatively. After 6–8 weeks of mostly range of motion and light resistive exercises, initial tendon healing will have completed, and more intensive strengthening exercises can be started, gradually progressing to plyometric exercise training and eventually running and jumping.

Flexor hallucis longus tendon

Flexor hallucis longus (FHL) injury is the most common tendinopathy of the lower extremity in classical ballet dancers or in those who participate in activities requiring frequent push-off movements. Chronic tendinopathy can give rise to pain and early arthritis and the development of fibrosis will reduce the range of motion of affected joints.

Patients usually present with an insidious onset of pain in the postero-medial aspect of the ankle or the medial aspect of the subtalar joint. If the patient is asked to flex the great toe against resistance, with the foot in plantar flexion, it will elicit pain along the FHL tendon. Another useful clinical test is to compare the range of passive extension of the first metatarso-phalangeal joint with the foot and ankle in the neutral and plantar flexed positions. If there is little or no extension in neutral but normal passive extension in plantar flexion, FHL injury is likely. Differential diagnoses include osteoarthritis, osteochondritis of the talus, posterior impingement and posterior tibial tendinopathy.

Techniques to prevent FHL injury include reducing turn-out of the hip, avoiding hard floors, strengthening the body's core and using well-fitted shoes with a firm sole. Treatment once injury has occurred is based on the correction of physical training errors, focussing principally on body mechanics. In the case of prolonged tendinopathy, 2–3 weeks of immobilization in a weight-bearing cast or walking boot is recommended.

Conclusion

Tendinopathy represents a failed healing response by a tendon after injury. Despite an abundance of therapeutic options, there is a lack of randomized prospective, placebo controlled trials to assist clinicians in choosing the best evidence-based management. The use of injectable substances in and around tendons such as platelet-rich plasma, autologous blood, polidocanol, corticosteroids has been advocated but there is minimal clinical evidence to support such therapy.

Tendinopathy remains a major problem in orthopaedic practice because its aetiology and pathophysiology are not clearly understood. The foot and ankle tendons, especially the Achilles and posterior tibial tendons can be frequently involved in the pathological process of tendinopathy producing chronic pain and deformity of the foot and ankle, with the association of a relevant limitation in sport practice and daily life activities. Randomized controlled trials are necessary to better clarify the best therapeutic options for the management of tendinopathies of the foot and ankle. ◆

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