ELSEVIER

Operative Techniques in

Sports Medicine

Achilles Injuries in the Athlete: Noninsertional

Nicola Maffulli, MD, PhD, FRCP, FRCS,[*,†] Alessio Giai Via, MD,[‡] and Francesco Oliva, MD, PhD[‡]

Achilles tendinopathy is common in both athletic and nonathletic individuals, and the incidence has risen in the last few decades. Although Achilles tendinopathy has been extensively studied, there is a clear lack of properly conducted scientific research to clarify its cause, pathology, natural history, and optimal management. The treatment of Achilles tendinopathy lacks evidence-based support, and individuals having tendinopathy are at risk of long-term morbidity with unpredictable clinical outcome. Most patients respond to conservative treatments. When conservative management is unsuccessful, surgery is recommended. Similar results have been reported with both open and minimally invasive techniques. However, minimally invasive techniques appear to reduce the risks of infection and provide better cosmetic results. An Achilles tendon rupture is frequent in young athletes and middle-aged people who practice recreational activities, and it is a serious injury. The management should take into account the age, occupation, and level of sporting activity. Open surgery provides good functional results and a lower rerupture rate, but it is frequently associated with a higher risk of superficial skin breakdown and wound problems. Percutaneous repair aims to provide good functional outcome while decreasing the problems associated with open surgery for wound healing and skin breakdown. Percutaneous repair followed by early functional rehabilitation is becoming increasingly common and should be considered in selected patients.

Oper Tech Sports Med ∎:∎∎∎-∎∎∎

KEYWORDS Achilles tendon, tendinopathy, rupture, repair, minimally invasive surgery

Introduction

Achilles tendinopathy and rupture can occur in athletes. Achilles tendinopathy is a clinical condition characterized by both pain and pathologic changes in and around the tendons. In the past 3 decades, the incidence of Achilles tendinopathy has risen because of greater participation in sporting activities. It is estimated to occur in approximately 7%-9% of top-level runners. A 10-fold increase in Achilles tendon (AT) injuries has been reported in runners when compared with that in age-matched controls.[1] Achilles tendinopathy is also common among people participating in racquet sports, track and field, volleyball, and soccer. However, the condition is by no means confined to athletes. In a recent study, 31% of 58 patients with Achilles tendinopathy did not participate in sports activities.[2]

Acute AT ruptures are frequent in young athletes and middle-aged people who practice recreational activities.[3] Most of these injuries occur in soccer, tennis, badminton, and squash players, but 25% of ruptures occur in sedentary patients as well. The incidence rate ranges from 6-18 per 100,000 per year.[3] Although the rupture seems to occur because of a traumatic injury on a healthy tendon, in reality, it is caused by an eccentric contraction on a pathologic asymptomatic tendon.[4]

Etiology and Pathophysiology

The etiology of Achilles tendinopathy remains unclear, and many predisposing factors have been proposed. AT injury can be acute or chronic, and it is caused by either intrinsic or

*Department of Musculoskeletal Disorders, School of Medicine and Surgery, University of Salerno, Salerno, Italy.
†Mary University of London, Barts and the London School of Medicine and Dentistry, Centre for Sports and Exercise Medicine, Mile End Hospital, London, UK.
‡Department of Orthopaedic and Traumatology, University of Rome "Tor Vergata," School of Medicine, Rome, Italy.
Address reprint requests to Nicola Maffulli, MD, PhD, FRCP, FRCS, Mary University of London, Barts and the London School of Medicine and Dentistry, Centre for Sports and Exercise Medicine, Mile End Hospital, 275 Bancroft Rd, London E1 4DG, UK. E-mail: n.maffulli@qmul.ac.uk

http://dx.doi.org/10.1053/j.otsm.2014.09.001
1060-1872/

extrinsic factors. In acute trauma, extrinsic loads exceed the tensile strength of the tendon. Extrinsic factors such as increased frequency and duration of training have been associated with Achilles tendinopathy. Changes in training pattern; poor technique; previous injuries; footwear; and environmental factors, such as training on hard, slippery, or slanting surfaces, may predispose the athlete to this condition.[5] However, a retrospective study showed that tendinopathy was not necessarily associated with the level of physical activity.[6] Fluoroquinolones and corticosteroids have been implicated as risk factors in tendinopathy. Ciprofloxacin causes enhanced interleukin-1β–mediated matrix metalloproteinase 3 (MMP3) release, inhibits tenocytes proliferation, and, as does corticosteroids, reduces the collagen and matrix synthesis.[7]

Intrinsic factors include alteration of lower limb function, biomechanics, sex, age, and genetics. Lower limb alignment and biomechanical faults are claimed to play a causative role in two-thirds of athletes with AT disorders. In particular, hyperpronation of the foot has been linked with an increased incidence of Achilles tendinopathy.[8] The incidence of tendinopathy is lower in women than that in men, and estrogen levels might play an important role in tendon homeostasis.[7] Women showed a lower risk of tendinopathy during premenopausal years than men did, but after menopause, this risk increases.[9] Postmenopausal estrogen deficiency seems to downregulate collagen turnover and decrease the elasticity in the tendons. Cook et al[10] reported that AT health of women using hormone replacement therapy was better than that of the controls. An animal study showed poorer AT healing in estrogen-deficient rats compared with that in controls.[11] Little is known about the effect of estrogen on tenocytes. A recent in vitro study showed that proliferation and tenocyte biosynthesis are negatively affected by age and estrogen deficiency.[12] Tenocytes from excised ovary and old rats showed a significantly lower proliferation rate, decreased collagen type I synthesis, and overexpression of MMP compared with that in young controls, showing that aging and, more significantly, estrogen levels may affect tendon metabolism and healing.

Age and some metabolic diseases, such as diabetes mellitus and obesity, are also predisposing factors. Connective tissue aging is associated with compromised tissue function, increased susceptibility to injury, and reduced healing capacity. This has been partly attributed to collagen cross-linking by advanced glycation end products (AGEs) that accumulate with both age and chronic diseases, particularly diabetes mellitus.[13] Protein glycation is a spontaneous reaction depending on the degree and duration of hyperglycemia, the half-life of the protein, and permeability of the tissue to free glucose. Glycated proteins can undergo further reactions giving rise to poorly characterized structures called AGEs.[13] AGEs are complex, heterogenous molecules that cause protein cross-linking, which alter physical characteristics of collagen fibers. In tendons, AGE formation has been shown to affect the interactions between collagen fibers, extracellular matrix (ECM) protein, and tenocytes.[14] These changes have been associated with both reduced healing capacity and altered mechanical properties of connective tissues. The effects of AGEs on the mechanical properties of tendon collagen fiber have been recently studied in a rat model.[15] The formation of AGEs would change the way a tendon reacts to loading, significantly reducing collagen fiber sliding in particular. Conversely, tendons try to compensate for this loss of function by increasing collagen fiber stretch, which may have potentially important implications for predisposing collagen fibrils to damage during everyday use. The tissue stiffness does not appear to be significantly affected. Therefore, physiological loads in the tendons of patients who are aged and diabetic could involve fiber "overstretching" that leads to accelerated accumulation of damage.

These findings may have important clinical consequences, because middle-aged people who are used to playing sports regularly do not change their habits, but changes in the physiology and function of connective tissues begin to occur, and this leads to a higher rate of tendon injury.

The physiopathology of tendinopathy in patients with obesity is yet to be understood, but some studies show that obesity may affect tendon health and reduce its healing ability.[16] Many authors consider obesity as a risk factor for tendon injury,[17] and poorer outcomes have been reported after arthroscopic rotator cuff repair in patients with obesity than that in controls.[16] Anatomical studies show that AT thickness is significantly higher in individuals with obesity than in control groups,[18] and ultrasound images showed thicker and hypoechogenic tendons in subjects with obesity compared with that in normal people.[19] Histologic changes have been observed in animal studies. Lipid drops accumulate in the ECM.[20] During ultrastructural analysis by transmission electron microscopy, disorganized collagen fibrils can be observed in the ECM of tendons in obese animals.[21] Low levels of glycosaminoglycans (chondroitin and dermatan sulfate), which play an important role in the regulation of the ECM and collagen fibrillogenesis, have been detected, which may be responsible for the inadequate deposition and organization of collagen fibrils.[22] Finally, obesity is frequently associated with other pathologies, such as diabetes mellitus and insulin resistance, which may also play a role in tendon pathology.

Changes in the expression of genes, regulating cell-cell and cell-matrix interactions in Achilles tendinopathy, have been reported, with downregulation of MMP3 and upregulation of MMP2 messenger RNA in tendinopathic AT samples.[23]

Clinical Presentation

Pain is the main symptom of Achilles tendinopathy. Pain occurs at the beginning and a short while after the end of a training session, with a period of diminished discomfort in between. As the pathologic process progresses, pain may occur during the entire exercise session, and in severe cases, it may interfere with activities of daily living. Although significant advances have been made for understanding the pathologic changes in both the ECM and the tenocytes, relatively little is known about pain. Traditionally it has been thought to arise through inflammation or via collagen fiber degeneration. However, chronically painful ATs have no evidence of inflammation, and many tendons with intratendinous pathology detected on magnetic resonance (MR) or ultrasound

images are not painful. Pain seems to be related to changes in neuronal regulation. Substance P is a neuropeptide, which involves an important function in pain perception. The sensory function of substance P is thought to be related to the transmission of pain information into the central nervous system.[24] It coexists with the excitatory neurotransmitter glutamate in primary afferents that respond to painful stimulation.[24] Recent researches showed changes in the peripheral neuronal phenotype in painful human tendinopathy. A significant upregulation of the excitatory glutaminergic system and a significant increase in sensory neuropeptide expression have been found, in addition to changes in the molecular morphology of tenocytes, blood vessels, and nerves.[25] In rotator cuff tendinopathy, substance P has been shown to correlate with pain, and the neural density in the subacromial bursa has been shown to correlate with rest pain.[25] An increased release of substance P was found in pathologic ATs[26,27] in animal studies. So the peripheral neuronal phenotype is currently considered an important factor in the pathogenesis of painful human tendinopathy.

In the acute phase, the tendon is diffusely swollen and edematous, and on palpation, tenderness is usually greatest at 2-6 cm proximal to the tendon insertion.[1] In chronic cases, exercise-induced pain is still the cardinal symptom, but crepitation and edema diminish. In most patients, simple single-leg heel raises are sufficient to cause pain. The differential diagnoses include tendinopathy or dislocation of the peroneal tendon, tendinopathy of plantar flexor tendons, irritation or neuroma of the sural nerve, and systemic inflammatory disease.

In case of an acute AT rupture, patients often give a history of feeling a blow to the posterior aspect of the leg and may describe an audible snap followed by pain and the inability to walk. A gap in the AT is usually palpable. Active plantar flexion of the foot is usually preserved because of the action of the tibialis posterior and the long toe flexors. The calf squeeze test, first described by Simmonds in 1957[28] but often credited to Thompson, is performed with the patient prone and the ankles clear of the table. The examiner squeezes the fleshy part of the calf, causing deformation of the soleus, resulting in plantar flexion of the foot if the AT is intact. The affected leg should be compared with the contralateral leg. The knee flexion test is performed with the patient prone and the ankles clear of the table.[29] The patient is asked to actively flex the knee to 90°. During this movement, the foot on the affected side falls into the neutral or the dorsiflexion position, and a rupture of the AT can be diagnosed (Fig. 1).

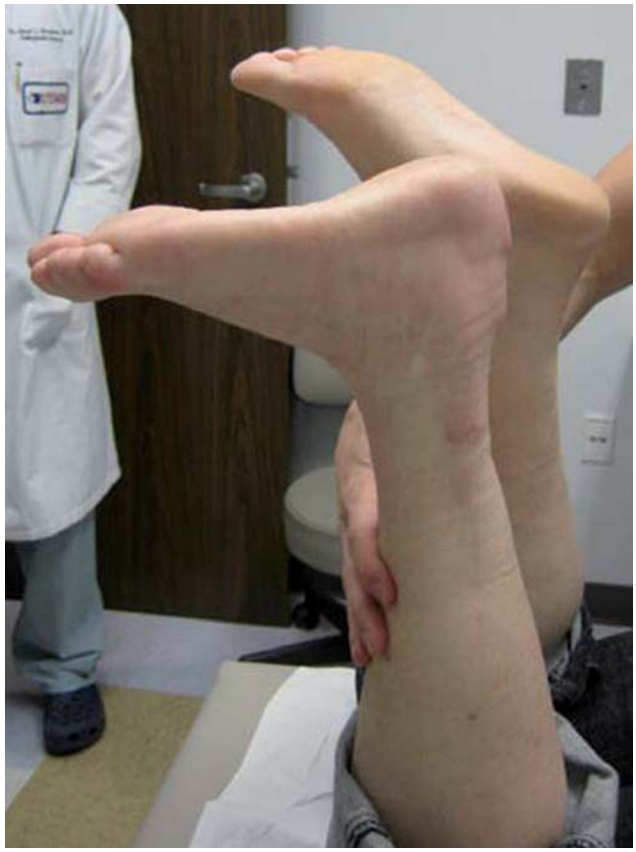

Figure 1 The knee flexion test. The foot on the affected side falls into the neutral or dorsiflexion position. (Color version of figure is available online.)

Imaging Methods

Achilles tendinopathy is a clinical diagnosis mainly based on a careful history recording and detailed clinical examination. However, at times, diagnostic imaging may be required to verify a clinical suspicion or occasionally to exclude other musculoskeletal disorders.[30]

Ultrasonography is widely used. It is easily available, quick, safe, and inexpensive. However, ultrasound is operator dependent, has somewhat limited soft tissue contrast, and is not as sensitive as MR imaging (MRI).[30] Color and power Doppler have recently added a new dimension to standard ultrasound tendon imaging.[31]

MRI is also a helpful diagnostic study. It provides extensive information on the internal morphology of tendons and the surrounding structures. An excellent correlation between MRI and pathologic findings at surgery has been reported.[32]

Management of Achilles Tendinopathy

Conservative Treatment

The initial management of Achilles tendinopathy is nonoperative. Conservative management would include activity modification and correction of training errors. Treatments that have been investigated with randomized controlled trials include nonsteroidal anti-inflammatory medication (nonsteroidal anti-inflammatory drug [NSAID]), eccentric exercise, glyceryl trinitrate patches, electrotherapy (microcurrent and microwave), sclerosing injections, and shock wave treatment (SWT).[33]

Modification of foot posture in some patients can reduce pain and increase the capacity to load the tendon.[34] An orthosis that places the hindfoot in neutral position may prove

beneficial. A heel lift of 12-15 mm is classically used as an adjunct to the management of AT pain.

Cryotherapy has been regarded as a most useful intervention in the acute phase of Achilles tendinopathy, as it has an analgesic effect, reduces the metabolic rate of the tendon, and decreases the extravasation of blood and protein from new capillaries found in tendon injuries.

Eccentric muscle training for treatment of Achilles tendinopathy has been recommended since the mid-1980s,[35] and it has been demonstrated to be superior to controls.[36,37] Several studies reported good short- and long-term results after eccentric training.[38-40] However, not all trials have had favorable results. In 2006, Sayana and Maffulli[41] studied the effects of eccentric exercises in sedentary nonathletic patients. Of the 34 patients with a clinical diagnosis of unilateral tendinopathy of the main body of the AT, 44% did not improve with the eccentric exercise regimen.

SWT is effective in Achilles tendinopathy.[42] Lohrer et al[43] reported significant pain reduction and increased functionality in patients with Achilles tendinopathy who received radial SWT. In a small, randomized, double-blinded, placebo controlled trial consisting of 39 patients, Peers reported a 77% success rate at 12-week follow-up.[44] In a recent randomized controlled trial, Rompe et al[45] compared the effectiveness of 3 management strategies. Group 1 was treated with eccentric loading exercises, group 2 was treated with repetitive low-energy SWT, and group 3 was treated with a "wait-and-see" approach. All 75 enrolled patients had received unsuccessful management with traditional nonoperative methods for a minimum of 3 months. At 4 months from baseline, the VISA-A score increased in all groups and pain rating decreased in all groups. For all outcome measures, groups 1 and 2 did not differ significantly and showed significantly better results than group 3 did.

NSAIDs are widely used in the management of acute athletic injuries. The evidence to support enhanced healing is limited. Some data suggest that NSAIDs impair tendon healing. In a study, they were shown to inhibit tendon cell migration and proliferation.[46] They had little or no effect on the clinical outcome.[47]

High-Volume Injections for the Management of Achilles Tendinopathy

High-volume image-guided injections (HVIGIs) target the neurovascular bundles growing from the paratenon into the AT. HVIGIs produce local mechanical effects, causing the neovessels to break or occlude, with pain relief that could also be explained by the destruction of sensory nerves. Denervation of the AT by releasing the paratenon may be the most important part of this procedure. Several substances have been injected in and around tendons, including normal saline, corticosteroids, and local anesthetics. In preliminary studies in patients with recalcitrant tendinopathy, HVIGIs decreased the extent of pain perceived by them and improved functional activities in the short and long term.[48,49] A mixture of 10 mL of 0.5% bupivacaine hydrochloride and 25 mg of hydrocortisone acetate can be used in chronic Achilles tendinopathy, immediately followed by 4 × 10 mL of injectable normal saline. Hydrocortisone acetate is used to prevent the inevitable acute mechanical inflammatory reaction produced by the large amount of fluid injected in the proximity of the tendon. The injection is performed under ultrasound guidance to avoid intratendinous corticosteroid injections. Patients are allowed to walk on the injected leg immediately, but they are strictly advised to refrain from high-impact activity for 72 hours. After this period, they are instructed to restart a heavy eccentric loading physiotherapy regime twice daily. Good results have been reported with this technique at short-term follow-up.[48] In a recent study, aprotinin has been used for HVIGIs with significant improvement in the VISA-A score, a relatively high rate of return to sport in athletic patients, and no serious adverse events at 1-year follow-up.[50] However, although HVIGI seems to be an effective and safe procedure, it warrants further investigation to understand the basis of its effects and to better study its role in the management of Achilles tendinopathy.

Surgery for Midportion Achilles Tendinopathy

In 24%-45.5% of patients with Achilles tendinopathy, conservative management is unsuccessful and surgery is recommended after at least 6 months of conservative management.[41] The frequency of surgery has been shown to increase with patient age, duration of symptoms, and occurrence of tendinopathic changes. Long-standing Achilles tendinopathy is associated with poor postoperative results with a greater rate of reoperation before reaching an acceptable outcome.[51] Both mini-invasive and open surgical techniques have been described.

Percutaneous Longitudinal Tenotomy

Percutaneous longitudinal tenotomy can be used when there is no paratenon involvement and when the intratendinous lesion is smaller than 2.5-cm long, assessed by MRI or ultrasounds. The procedure is performed under ultrasound guidance. The procedure can be performed as an outpatient case. The patient lies prone on the operating table with the feet protruding beyond the edge and the ankles resting on a sandbag. A bloodless field is not necessary. The tendon is accurately palpated and the area of maximum swelling or tenderness is marked and checked again by US scanning. The skin and the subcutaneous tissues over the AT are infiltrated using 10-15 mL of plain 1% lidocaine. A number 11 surgical scalpel blade is inserted parallel to the long axis of the tendon fibers in the marked area in the center of the region of tendinopathy. The cutting edge of the blade points caudally and penetrates the whole thickness of the tendon (Fig. 2). Keeping the blade still, a full passive ankle dorsiflexion movement is produced (Fig. 3). The scalpel blade is then retracted to the surface of the tendon, inclined 45° on the sagittal axis and inserted medially through the original tenotomy (Fig. 4). Keeping the blade still, a full passive ankle flexion is produced. The whole procedure is

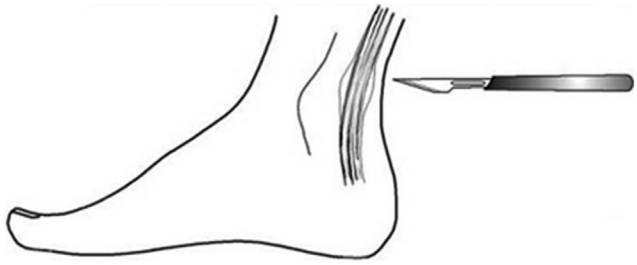

Figure 2 A number 11 scalpel blade inserted into the predetermined area with the blade edge pointing cranially.

repeated by inclining the blade 45° laterally to the original tenotomy. Keeping the blade still, a full passive ankle flexion is produced. The blade is then partially retracted to the posterior surface of the AT and reversed 180°, so that its cutting edge now points cranially, and the whole procedure is repeated taking care to dorsiflex the ankle passively. Preliminary cadaveric studies showed that a tenotomy that is 2.8-cm long on average is obtained through a stab wound in the main body of the tendon. A Steri-Strip can then be applied on the stab wound. The wound is dressed with cotton swabs, and a few layers of cotton wool and a crepe bandage are used.

After surgery, early active dorsiflexion and plantar flexion of the foot is encouraged. On the second postoperative day, patients are allowed to walk using elbow crutches, weight bearing as able, and full weight bearing is allowed after 2 or 3 days when the bandage is reduced to a simple adhesive plaster over the wounds. Stationary bicycling and isometric concentric and eccentric strengthening of the calf muscles are started under physiotherapy guidance after 4 weeks. Swimming and water running are encouraged from the second week. Gentle running is started 4-6 weeks after the procedure, and the mileage is gradually increased.

Excellent and good results have been reported in 63% of athletes with unilateral Achilles tendinopathy treated with ultrasound-guided percutaneous longitudinal tenotomy after failure of conservative management without experiencing significant complications.[52] This technique is simple, can be performed on an outpatient basis, requires minimal follow-up care, and does not hinder further surgery if necessary.

Minimally Invasive Stripping for Chronic Achilles Tendinopathy

In this procedure, 4 skin incisions are made. The first 2 incisions are 0.5-cm longitudinal incisions at the proximal origin of the AT, just medial and lateral to the origin of the tendon. The other 2 incisions are also 0.5-cm long and longitudinal, but 1 cm proximal to the distal end of the tendon insertion on the calcaneus. A mosquito forceps is inserted in the proximal incisions (Fig. 5), and the AT is freed of the peritendinous adhesions. A number 1 unmounted Ethibond suture is inserted proximally, passing through the 2 proximal incisions (Fig. 6). The Ethibond suture is retrieved from the distal incisions (Fig. 7), over the posterior aspect of the AT. Using a gentle seesaw motion, the Ethibond suture thread is made to slide posterior to the tendon (Fig. 8), which is stripped and freed from the fat of the Kager triangle.

The procedure is repeated for the posterior aspect of the AT. If necessary, using a number 11 blade, longitudinal percutaneous tenotomies parallel to the tendon fibers are made.

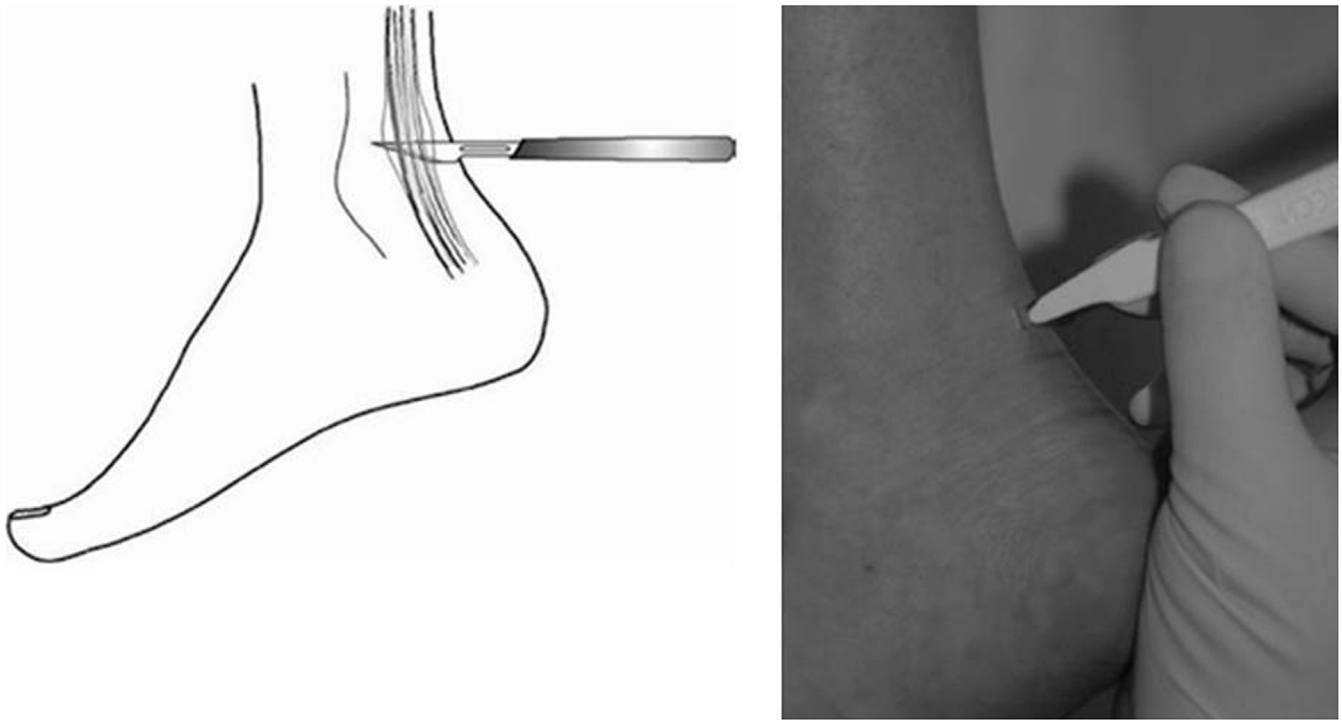

Figure 3 The blade penetrating the whole thickness of the Achilles tendon, and a full passive ankle dorsiflexion movement is produced.

Figure 4 The procedure is repeated with the blade inclining at 45° medial and 45° lateral to the original tenotomy.

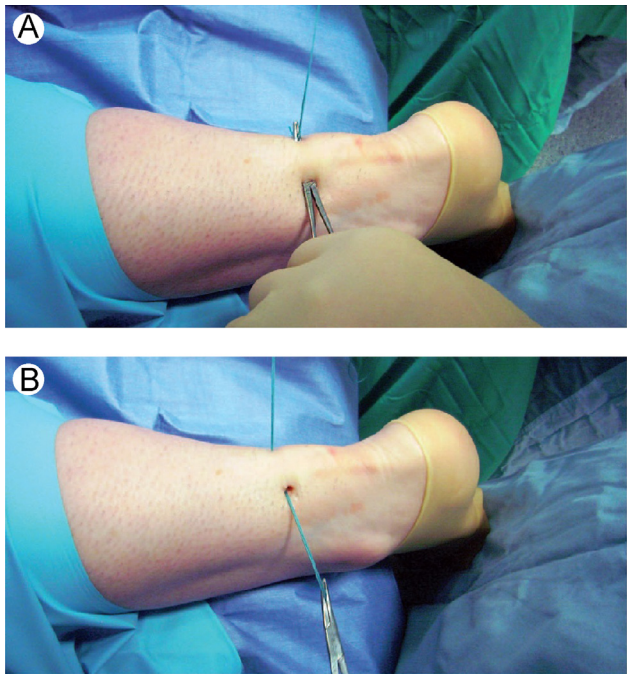

Figure 6 A number 1 Ethibond suture is inserted proximally, passing through the 2 proximal incisions over the anterior aspect of the Achilles tendon. (Color version of figure is available online.)

The subcutaneous and subcuticular tissues are closed in a routine fashion. A cam boot can be used if necessary. Postoperatively, patients are allowed to mobilize with full weight bearing, and after 2 weeks, they can start physiotherapy.

The rationale behind the current management modality is that the sliding of the suture breaks the neovessels and the accompanying nerve supply, thus decreasing pain. This minimally invasive technique reduces the risks of infection and is technically easy to master and inexpensive. It may provide a greater potential for the management of recalcitrant Achilles tendinopathy by breaking neovessels and the accompanying nerve supply to the tendon.[53] It can be associated with other minimally invasive procedures to optimize results.

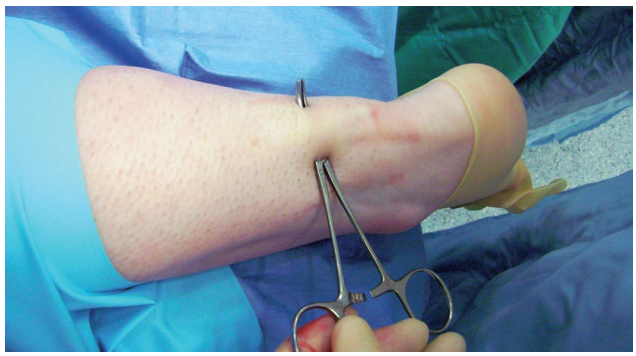

Figure 5 A mosquito forceps is inserted in the proximal incisions. (Color version of figure is available online.)

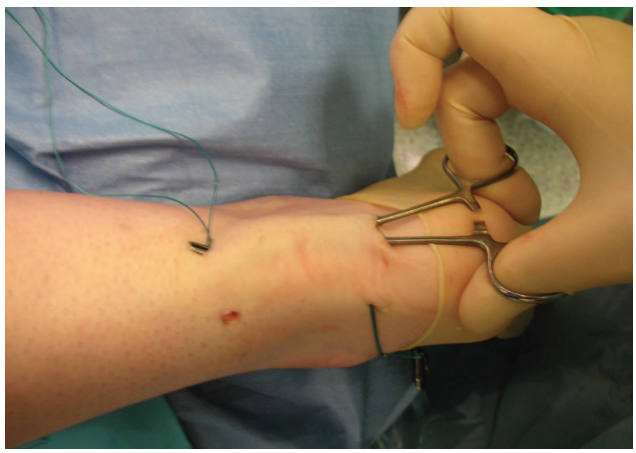

Figure 7 The Ethibond suture is retrieved from the distal incisions. (Color version of figure is available online.)

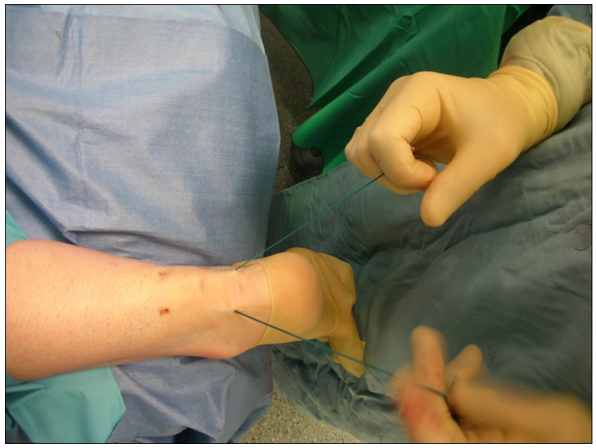

Figure 8 The Ethibond suture is slid over the anterior aspect of the Achilles tendon with a gentle seesaw motion. The whole process is repeated over the posterior aspect of the tendon. (Color version of figure is available online.)

Open Surgery

Under regional or general anesthesia, the patient is placed prone with the ankles clear of the operating table. The limb is exsanguinated, and the tourniquet is inflated to 250 mm Hg. The incision is made on the medial side of the tendon to avoid injury to the sural nerve and the short saphenous vein. The skin edges of the incision should be handled with extreme care, as wound healing problems are serious. The paratenon is identified and incised. In patients with evidence of coexisting paratendinopathy, the scarred and thickened tissue is generally excised. The tendon is incised sharply in line with the tendon fiber bundles. The tendinopathic tissue can be identified as it generally has lost its shiny appearance and it frequently contains disorganized fiber bundles that have more of a "crabmeat" appearance. This tissue is sharply excised (Fig. 9). The remaining gap can be repaired using a side-to-side repair, but the authors leave it unsutured (Fig. 10). If significant loss of tendon tissue occurs during the debridement, a tendon augmentation or transfer could be considered, although the authors find that this is rarely necessary. Then, the subcutaneous tissue is sutured with absorbable material and the skin edges are juxtaposed with Steri-Strips and a routine compressive bandage. The limb is immobilized in a below-knee synthetic cast with the foot plantigrade.

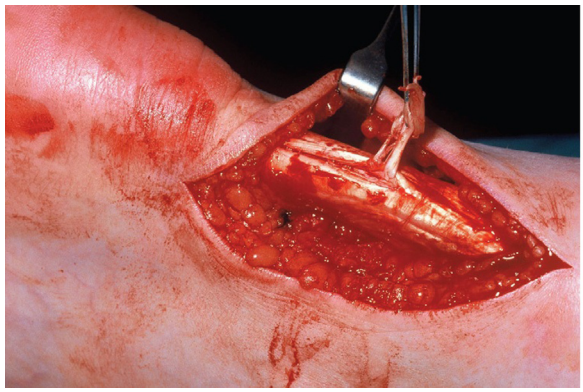

Figure 9 The tendinopathic tissue is excised. (Color version of figure is available online.)

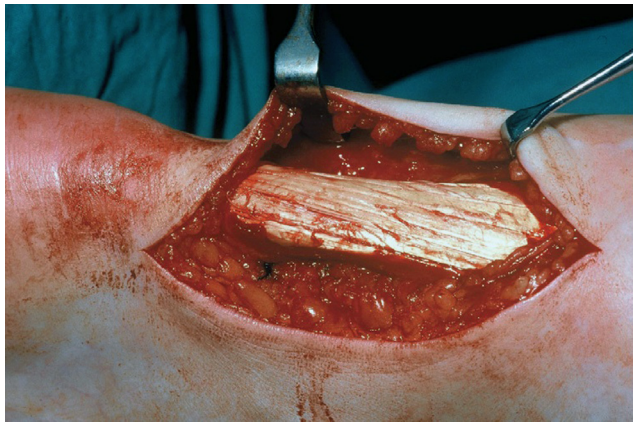

Figure 10 Appearance at the end of the procedure. (Color version of figure is available online.)

A period of initial splinting and crutch-walking is generally used to allow pain and swelling to subside after surgery. After 2 weeks, the wound is inspected and motion exercises are initiated. The patient is 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. Weight bearing is allowed according to the degree of debridement needed at surgery, and early weight bearing is encouraged. However, extensive debridements and tendon transfers may require protected weight bearing for 4-6 weeks postoperatively. After 6-8 weeks, more intensive strengthening exercises are started, gradually progressing to plyometrics and eventually running and jumping.

Successful results have been reported with this surgical procedure. A systematic review of the literature showed successful results in more than 70% of cases,[54] but these relatively high success rates are not always observed in clinical practice. This is probably because of the poor method scores of many articles. Patients should be informed of the potential failure of the procedure, risk of wound complications, and occasional prolonged recovery time. Possible complications of this surgical procedure are wound healing problems, infection, sural nerve injury, rupture of the AT, and deep vein thrombosis.

Management of AT Ruptures in Athletes

Acute AT rupture is a serious injury. Most (75%) acute ruptures occur during recreational activities in men aged between 30 and 40 years, but 25% of ruptures occur in sedentary patients.[55] The incidence ranges from 6-18 per 100,000 per year.[56] Management of acute ruptures of the AT is still controversial. In conservatively managed patients, healing in a lengthened position may result in loss of calf muscle strength. In addition, incomplete healing of the gap between the tendon stumps may contribute to the high rerupture rate. More recent literature reports a rerupture rate up to 13% in

patients treated conservatively,[57] which is not acceptable to the authors in young and active patients. We feel that operative management provides a lower rerupture rate, early functional treatment, less calf atrophy, and stronger push off than nonsurgical treatment does.

Open, minimally invasive, and percutaneous procedures have been successfully used. Open surgery provides good strength to the repair, low rerupture rates, and reliably good endurance and power to the gastrocnemius-AT complex.[58] However, open surgical approaches result in a high risk of infection and morbidity. Review articles and meta-analysis show high costs and a 20-fold higher rate of complications in open procedures than that in conservative treatment.[59] Therefore, minimally invasive and percutaneous procedures have been successfully used to avoid these complications. Minimally invasive repair allows one to see the tendon stumps through a small incision over the defect, although the sutures themselves may be passed through stab incisions. With percutaneous repair, the tendon stumps are not seen directly. The resting tone of the ankle and a calf squeeze test or visualization by ultrasound or endoscopy indicates the restoration of the tendon continuity. These techniques provide many advantages, such as less iatrogenic damage to normal tissues; lower postoperative pain; accurate opposition of the tendon ends, thus minimizing surgical incisions; and improved cosmetics. A recent systematic review reported a rate of superficial infections of 0.5% and 4.3% after minimally invasive and open surgery, respectively.[58] Shorter hospitalization time and average time to return to working activities were also found. The functional outcomes were not significantly different between minimally invasive and open surgeries. A potential complication of percutaneous AT repair is sural nerve injury.

Minimally Invasive AT Repair

A 1-cm transverse incision is made over the defect using a number 11 blade. In this technique, 4 longitudinal stab incisions are made lateral and medial to the tendon, 6 cm proximal to the palpable defect. Furthermore, 2 or 4 longitudinal incisions are made on either side of the tendon, 4-6 cm distal to the palpable defect. Forceps are then used to mobilize the tendon from beneath the subcutaneous tissues. A 9-cm Mayo needle is threaded with 2 double loops of a number 1 Maxon suture, and this is passed transversely between the most proximal stab incision through the bulk of the tendon (Fig. 11). The bulk of the tendon is surprisingly superficial. The loose ends are held with a clip. In turn, each of the ends is then passed distally from proximal to the distal transverse passage through the bulk of the tendon to pass out of the diagonally opposing stab incision. A subsequent diagonal pass is then made to the transverse incision over the ruptured tendon. To prevent entanglement, both the ends of the Maxon suture are held in separate clips. This suture is then tested for security by pulling with both its ends distally. Another double loop of the Maxon suture is then passed between the most distal stab incision through the tendon (Fig. 12), and in turn through the tendon and out of the transverse incision starting distal to the transverse passage (Figs. 13 and 14). The ankle is held in full plantar flexion, and in turn, the opposing ends of the Maxon thread are tied together with a double throw knot, and then 3 further throws before being buried using the forceps. A clip is used to hold the first throw of the lateral side to maintain the tension of the suture. We use a 3-0 VICRYL suture to close the transverse incision and Steri-Strips to close

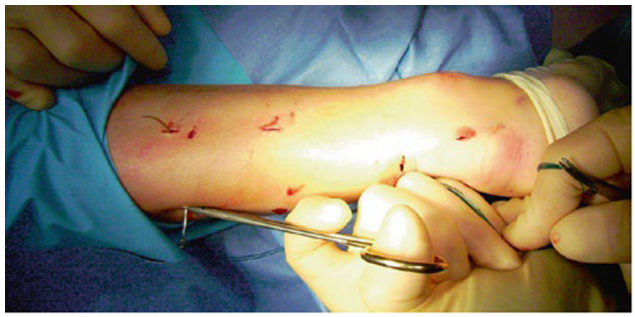

Figure 11 A 9-cm Mayo needle is threaded with 2 double loops of a number 1 Maxon suture, and this is passed transversely between the most proximal stab incision through the bulk of the tendon. (Color version of figure is available online.)

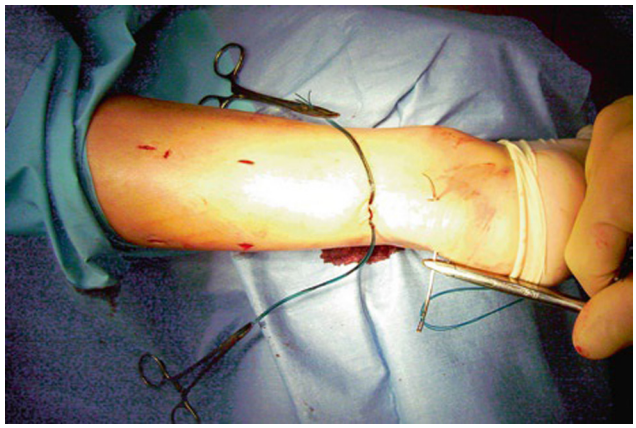

Figure 12 Another double loop of the Maxon suture is then passed between the most distal stab incision through the tendon. (Color version of figure is available online.)

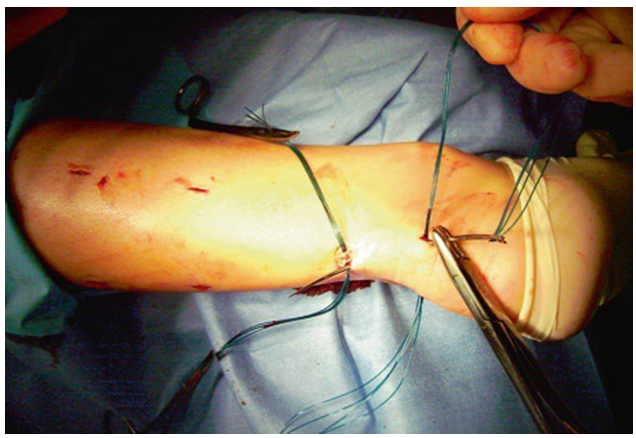

Figure 13 The double loop of the Maxon suture is passed in turn through the tendon and out of the transverse incision, starting distal to the transverse passage and then out the incision at the rupture site. (Color version of figure is available online.)

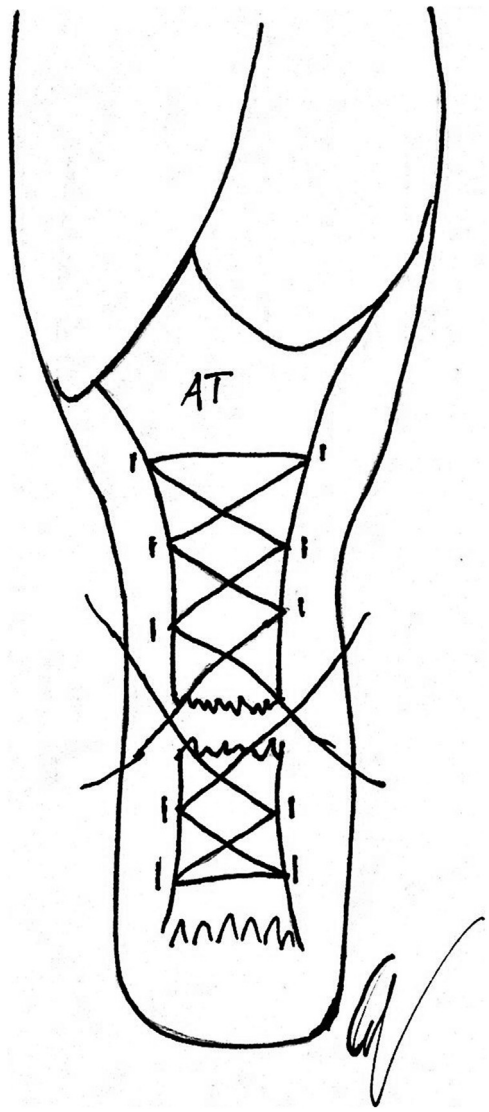

Figure 14 The suture's path. AT, Achilles tendon.

the stab incisions. A nonadherent dressing is applied. A full plaster cast is applied in the operating room with the ankle in physiological equinus. The cast is split on both medial and lateral sides to allow for swelling. The patient is discharged on the same day of the operation.

Excellent results have been reported in 17 elite athletes after percutaneous surgical repair of their AT rupture.[60] All patients came back to compete at a high level, and the average time to return to full sport participation was 4.8 months (range: 3.2-6.5).

Conclusion

Tendinopathy of the AT is common both in athletic and nonathletic individuals. Most patients respond to conservative treatments. When conservative management is unsuccessful, surgery is recommended. Successful results have been reported with an open surgical procedure, but patients should be informed of the long recovery time and possible complications. Minimally invasive techniques show good results, thus reducing the risk of infection. They are technically easy to perform and relatively inexpensive. They can be associated with other minimally invasive procedures to optimize results and should be considered for treatment of Achilles tendinopathy.

Acute AT rupture is a serious injury for athletes. The management should take into account the age, occupation, and level of sporting activity of the patient. Open surgery is frequently associated with a higher risk of superficial skin breakdown and wound problems, which can be minimized by performing a percutaneous repair. Percutaneous repair followed by early functional rehabilitation is becoming increasingly common and should be considered in selected patients.

References

1. Maffulli N, Sharma P, Luscombe KL: Achilles tendinopathy: Aetiology and management. J R Soc Med 97:472-476, 2004
2. Rolf CMT: Etiology, histopathology, and outcome of surgery in achillodynia. Foot Ankle Int 18:565-569, 1997
3. Longo UG, Petrillo S, Maffulli N, et al: Acute Achilles tendon rupture in athletes. Foot Ankle Clin 18:319-338, 2013
4. Hattrup SJ, Johnson KA: A review of ruptures of the Achilles tendon. Foot Ankle 6:34-38, 1985
5. Rompe JD, Furia JP, Maffulli N: Mid-portion Achilles tendinopathy: Current options for treatment. Disabil Rehabil 30:1666-1676, 2008
6. Astrom M: Partial rupture in Achilles tendinopathy. A retrospective analysis of 342 cases. Acta Orthop Scand 69:404-407, 1998
7. Ronga M, Oliva F, Vigneti D, et al: Achilles tendinopathy. Physiopathology. In: Maffulli N, Oliva F (eds): Achilles Tendon. Rome, CIC Edizioni Internazionali, 39-45, 2009
8. Nigg B: The role of impact forces and foot pronation: A new paradigm. Clin J Sport Med 11:2-9, 1994
9. Bryant AL, Clark RA, Bartold S, et al: Effects of oestrogen on the mechanical behavior of the human Achilles tendon in vivo. J Appl Physiol 105:1035-1043, 2008
10. Cook JL, Bass SL, Black JE: Hormone therapy is associated with smaller Achilles tendon diameter in active postmenopausal women. Scand J Med Sci Sports 17:128-132, 2007
11. Circi E, Akpinar S, Akgun RC, et al: Biomechanical and histological comparison of the influence of oestrogen deficient state on tendon healing potential in rats. Int Orthop 33:1461-1466, 2009
12. Torricelli P, Veronesi F, Pagani S, et al: In vitro tenocyte metabolism in aging and oestrogen deficiency. Age (Dordr) 35:2125-2136, 2013
13. Ahmed N: Advanced glycation endproducts—Role in pathology of diabetic complications. Diabetes Res Clin Pract 67:3-21, 2005
14. Gautieri A, Redaelli A, Buehler MJ, et al: Age- and diabetes-related nonenzymatic crosslinks in collagen fibrils: Candidate amino acids involved in advanced glycation end-products. Matrix Biol 34:89-95, 2014
15. Li Y, Fessel G, Georgiadis M, et al: Advanced glycation end-products diminish tendon collagen fiber sliding. Matrix Biol 32:169-177, 2013
16. Warrender WJ, Brown OL, Abboud JA: Outcomes of arthroscopic rotator cuff repairs in obese patients. J Shoulder Elbow Surg 20:961-967, 2011
17. Giai Via A, De Cupis M, Spoliti M, et al: Clinical and biological aspects of rotator cuff tears. Muscles Ligaments Tendons J 3:70-92, 2013
18. Gaida JE, Cook JL, Bass SL: Adiposity and tendinopathy. Disabil Rehabil 30:1555-1562, 2008
19. Abate M, Oliva F, Schiavone C, et al: Achilles tendinopathy in amateur runners role of adiposity (tendinopathies and obesity). Muscles Ligaments Tendons J 2:44-48, 2012
20. Biancalana A, Veloso LA, Gomes L: Obesity affects collagen fibril diameter and mechanical properties of tendons in Zucker rats. Connect Tissue Res 51:171-178, 2010
21. Hills AP, Hennig EM, Byrne NM, et al: The biomechanics of adiposity—Structural and functional limitations of obesity and implications for movement. Obes Rev 7:13-24, 2006

22. Waggett AD, Ralphs JR, Kwan AP, et al: Characterization of collagens and proteoglycans at the insertion of the human Achilles tendon. Matrix Biol 16:457-470, 1998
23. Magra M, Maffulli N: Matrix metalloproteases: A role in overuse tendinopathies. Br J Sports Med 39:789-791, 2005
24. De Felipe C, Herrero JF, O'Brien JA, et al: Altered nociception, analgesia and aggression in mice lacking the receptor for substance P. Nature 392:394-397, 1998
25. Dean BJ, Franklin SL, Carr AJ: The peripheral neuronal phenotype is important in the pathogenesis of painful human tendinopathy: A systematic review. Clin Orthop Relat Res 471:3036-3046, 2013
26. Khan KM, Cook J: Overuse tendon injuries: Where does the pain come from? Sports Med Arthrosc Rev 8:17-31, 2000
27. Schubert TE, Weidler C, Lerch K, et al: Achilles tendinosis is associated with sprouting of substance P positive nerve fibres. Ann Rheum Dis 64:1083-1086, 2005
28. Simmonds FA: The diagnosis of the ruptured Achilles tendon. Practitioner 179:56-58, 1957
29. Maffulli N: The clinical diagnosis of subcutaneous tear of the Achilles tendon. A prospective study in 174 patients. Am J Sports Med 26:266-270, 1998
30. Maffulli N, Kenward MG, Testa V, et al: Clinical diagnosis of Achilles tendinopathy with tendinosis. Clin J Sport Med 13:11-15, 2003
31. Zanetti M, Metzdorf A, Kundert H-P, et al: Achilles tendons: Clinical relevance of neovascularization diagnosed with power Doppler US. Radiology 227:556-560, 2003
32. Maffulli N, Binfield P, Moore D: Surgical decompression of chronic central core lesions of the Achilles tendon. Am J Sports Med 27:747-752, 1999
33. Loppini M, Maffulli N: Conservative management of tendinopathy: An evidence-based approach [Review]. Muscles Ligaments Tendons J 1:133-136, 2011
34. McCrory J, Martin D, Lowery R, et al: Etiologic factors associated with Achilles tendinitis in runners. Med Sci Sports Exerc 31:1374-1381, 1999
35. Stanish WD, Rubinovich RM, Curwin S: Eccentric exercise in chronic tendinitis. Clin Orthop Relat Res 208:65-68, 1986
36. Maffulli N, Walley G, Sayana MK, et al: Eccentric calf muscle training in athletic patients with Achilles tendinopathy. Disabil Rehabil 30:1677-1684, 2008
37. Alfredson H, Cook J: A treatment algorithm for managing Achilles tendinopathy: New treatment options. Br J Sports Med 41:211-216, 2007
38. Alfredson H, Pietila T, Jonsson P, et al: Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. Am J Sports Med 26:360-366, 1998
39. Roos E, Engstrom M, Lagerquist A, et al: Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy—A randomized trial with 1-year follow-up. Scand J Med Sci Sports 14:286-295, 2004
40. de Vos RJ, Weir A, Visser RJ, et al: The additional value of a night splint to eccentric exercises in chronic midportion Achilles tendinopathy: A randomised controlled trial. Br J Sports Med 41:e5, 2007
41. Sayana MK, Maffulli N: Eccentric calf muscle training in non-athletic patients with Achilles tendinopathy. J Sci Med Sport 10:52-58, 2007
42. Frairia R, Berta L: Biological effects of extracorporeal shock waves on fibroblasts. A review [Review article]. Muscles Ligaments Tendons J 1:137-146, 2012
43. Lohrer H, Scholl J, Arentz S: Achilles tendinopathy and patellar tendinopathy. Results of radial shockwave therapy in patients with unsuccessfully treated tendinoses. Sportverletz Sportschaden 16:108-114, 2002
44. Peers K: Extracorporeal Shock Wave Therapy in Chronic Achilles and Patellar Tendinopathy. Leuven, Leuven University Press, 61-75, 2003
45. Rompe JD, Nafe B, Furia JP, et al: Eccentric loading, shock wave treatment, or a wait-and-see policy for tendinopathy of the main body of tendo Achillis: A randomized controlled trial. Am J Sports Med 35:374-383, 2007
46. Tsai WC, Hsu CC, Chou SW, et al: Effects of celecoxib on migration, proliferation and collagen expression of tendon cells. Connect Tissue Res 48:46-51, 2007
47. Astrom M, Westlin N: No effect of piroxicam on Achilles tendinopathy. A randomized study of 70 patients. Acta Orthop Scand 63:631-634, 1992
48. Humphrey J, Chan O, Crisp T, et al: The short-term effects of high volume image guided injections in resistant non-insertional Achilles tendinopathy. J Sci Med Sport 13:295-298, 2009
49. Chan O, O'Dowd D, Padhiar N, et al: High volume image guided injections in chronic Achilles tendinopathy. Disabil Rehabil 30:1697-1708, 2008
50. Maffulli N, Spiezia F, Longo UG, et al: High volume image guided injections for the management of chronic tendinopathy of the main body of the Achilles tendon. Phys Ther Sport 14:163-167, 2013
51. Rees JD, Maffulli N, Cook J: Management of tendinopathy. Am J Sports Med 37:1855-1856, 2009
52. Testa V, Capasso G, Benazzo F, et al: Management of Achilles tendinopathy by ultrasound-guided percutaneous tenotomy. Med Sci Sports Exerc 34:573-580, 2002
53. Longo UG, Ramamurthy C, Denaro V, et al: Minimally invasive stripping for chronic Achilles tendinopathy. Disabil Rehabil 30:1709-1713, 2008
54. Tallon C, Coleman BD, Khan KM, et al: Outcome of surgery for chronic Achilles tendinopathy: A critical review. Am J Sports Med 29:315-320, 2001
55. Wong J, Barrass V, Maffulli N: Quantitative review of operative and nonoperative management of achilles tendon ruptures. Am J Sports Med 30:565-575, 2002
56. Maffulli N, Waterston SW, Squair J, et al: Changing incidence of Achilles tendon rupture in Scotland: A 15-year study. Clin J Sport Med 9:157-160, 1999
57. Guillo S, Del Buono A, Dias M, et al: Percutaneous repair of acute ruptures of the tendon Achillis. Surgeon 11:14-19, 2013
58. Del Buono A, Volpin A, Maffulli N: Minimally invasive versus open surgery for acute Achilles tendon rupture: A systematic review. Br Med Bull 109:45-54, 2014
59. Maffulli N: Rupture of the Achilles tendon. J Bone Joint Surg Am 81:1019e36, 1999
60. Maffulli N, Longo UG, Maffulli GD, et al: Achilles tendon ruptures in elite athletes. Foot Ankle Int 32:9-15, 2011