Chapter 12
Foot and Ankle: Conservative Management, Operative Management, and Return to Sport

Monique C. Chambers, Dukens LaBaze, Jesse Raszeswki, and MaCalus V. Hogan

Achilles Tendon Injury

Introduction

Achilles tendinopathy is one of the most frequent foot and ankle injuries that results from overuse [1]. Achilles tendon ruptures occur more commonly in healthy, active individuals with a mean age of 37 years [2]. Achilles injury is more likely present in running and jumping activities, with rates as high as 9% in recreational runners [1]. It has an incidence of 5.6% in nonathletes [1]. Males are predisposed with as high as a 30:1 ratio to women [2]. Achilles tendinopathy can occur at two anatomical locations: *insertional* tendinopathy at the calcaneus-Achilles tendon junction or *non-insertional* tendinopathy which is approximately 2–6 cm proximal to the insertion site of the Achilles tendon [1]. Insertional tendinopathy tends to occur in active individuals, whereas non-insertional is more frequently observed in older, less active, and overweight individuals [1].

Tendinous fibers from the gastrocnemius and the soleus muscles coalesce toward the calcaneal tuberosity to form the Achilles tendon, which is the largest tendon in the body [3]. The tendon spans three separate joints and is integral for knee flexion,

M. C. Chambers, MD, MSL · D. LaBaze, BS
Department of Orthopaedic Surgery, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

J. Raszeswki, MBS
Alabama College of Osteopathic Medicine, Dothan, AL, USA
e-mail: raszewskija@acomedu.org

M. V. Hogan, MD (✉)
Foot and Ankle Division, Department of Orthopedic Surgery, University of Pittsburgh Medical Center, Pittsburgh, PA, USA
e-mail: hoganmv@upmc.edu

V. J. Wright, K. K. Middleton (eds.), *Masterful Care of the Aging Athlete*,
https://doi.org/10.1007/978-3-319-16223-2_12

foot plantar flexion, and hind foot inversion [2]. The normal blood supply is variable but is derived from three sources: the muscle-tendon junction, the bone-tendon junction, and the length of the tendon [1]. The most important blood supply derives from the paratenon in the middle zone, with the most abundant blood supply located at the insertion [1]. This distribution of the blood supply leaves the Achilles tendon prone to injury in the watershed area approximately 2–6 cm from its insertion on the posterior calcaneus [4]. The tibial nerve supplies the superficial and deep nerves to local tissues that innervate the Achilles tendon [1].

Insertional tendinopathy is due to the degeneration of the tendon at the insertion on the calcaneus, stemming from old age, steroid use, obesity, diabetes, and inflammatory arthropathies [2]. Conservative treatment, activity modification, is the mainstay of therapy. When surgical intervention is necessary, a posterior central tendon approach for debridement of the tendon with removal of the prominent calcaneal projection is often utilized [2].

Non-insertional Achilles tendinopathy is due to an inflammatory reaction that leads to circulatory impairment and edema, which can progress to fibrinous adhesions in the chronic state [2]. Conservative management is also the mainstay of therapy, with activity modifications, eccentric exercises, NSAIDs, injections, and shock wave therapy [2]. If pain is not alleviated with conservative measures, surgical intervention can be used. Surgical treatment includes debridement and excision of the fibrous adhesions, with the goal of denervating and devascularizing the paratenon while promoting a scarring response within the tendon [2].

Achilles tendinopathy is often multifactorial in origin, and the risk factors can be divided into intrinsic and extrinsic factors. Intrinsic factors include biomechanical abnormalities of the lower extremity, and extrinsic factors include excessive mechanical overload and training errors [1]. For intrinsic examples, individuals with hyper-pronation or cavus feet are prone to higher incidence of Achilles tendon problems [3]. Advanced age also correlates with Achilles tendinopathy. As for extrinsic factors, individuals that use footwear with insufficient heel height or inadequate shock absorption have been shown to magnify the stress placed on the Achilles tendon during athletic activity [3]. Achilles tendinopathy is severely debilitating, and injuries can have a substantial socioeconomic impact regardless of the treatment selected [4].

In this chapter, the clinical presentation, physical examination, diagnostic work-up, and treatments will be reviewed and illustrated (Table 12.1).

Table 12.1 Features of Achilles tendinopathy

Clinical manifestations	• Pain in the posterior aspect of the foot/ankle • Pain with plantar flexion motion • Inability to bear weight
Diagnosis	• Inability to perform single-leg raise • Confirm with MRI/ultrasound
Treatment	• Tendinopathy → physical therapy • Anatomical deformity (tear/rupture) → surgery

Clinical Evaluation

Achilles tendinopathy is primarily diagnosed based on history and physical exam. Patients usually describe sudden onset of pain in the posterior aspect of the foot and ankle, with activities that incorporate forceful plantar flexion [4]. Patients who have ruptured the tendon often have a "pop" sensation, lose the ability to bear weight, and report sudden weakness in plantar flexion of the ankle [4]. Sufficient evaluation of the Achilles tendon requires bilateral examination of the foot and ankle while the patient is standing, ambulating, and prone with their feet suspended over the edge of an examination table.

A complete musculoskeletal examination entails thorough inspection and palpation of both feet and ankles with side-by-side comparisons for reference. Begin by inspecting the posterior ankle for signs of fracture, sprain, or tendon rupture, which are typically associated with bruising, swelling, and/or foot misalignment. Also, assess for peripheral artery disease, which presents as dry, shiny, hairless, hyperpigmented, or edematous skin. Evaluate for signs of poor gait mechanics, evidenced by asymmetric diminution of the patient's footwear, foot deformity, and leg-length discrepancy [5].

Next, palpate the Achilles tendon in plantar flexion, dorsiflexion, and neutral position. Engage the proximal aspect between the thumb and index finger, applying mild pressure in a stepwise fashion distally toward the insertion site on the calcaneus. Assess for tenderness, defects, thickening, and crepitus, remaining aware that the presence of edema or hematoma may result in false-negative examination.

Examination should be conducted with the patient prone and the feet hanging off the ledge of the examination table [3]. The entire gastrocnemius-soleus-myotendinous complex should be thoroughly palpated, while the ankle is gently maneuvered through active and passive ranges of motion [3]. Positive physical examination findings include increased passive ankle dorsiflexion, weak plantar flexion strength, and palpable defect overlying a tear [4].

Location can help differentiate tendinopathy from other causes of posterior ankle tenderness. Tendinopathy classically exhibits a pattern of localized tenderness 2–6 cm proximally from the calcaneal insertion site as well as crepitus with motion. Tenderness directly on the Achilles tendon at the insertion site is more consistent with enthesopathy. Tenderness and warmth at the insertion site, superficial to the Achilles tendon, is more consistent with subcutaneous calcaneal bursitis. Tenderness and warmth at the insertion site, deep to the Achilles tendon, is more consistent with subtendinous bursitis (retrocalcaneal bursitis), which can be evaluated by grasping and laterally displacing the Achilles tendon with one hand while palpating the underlying soft tissue with the contralateral hand [6]. The American Academy of Orthopaedic Surgeons (AAOS) clinical practice guidelines note that a diagnosis can be made when two or more of the following exam findings are noted: a positive Thompson test (when compression of the calf in supine position does not elicit passive plantar flexion), decreased plantar flexion strength, palpable defect distal to insertion site, or positive Matles test (increased passive ankle dorsiflexion at rest) [2].

Potential for misdiagnosis of tendon pathology on physical examination exists in the presence of certain signs and symptoms: intact active plantar flexion of the foot, intact ambulation, absence of pain, and undetectable Achilles tendon defects on examination. In fact, 20–30% of ruptured Achilles tendons are missed during evaluation due to the patients' ability to ambulate and actively plantar flex the ankle [7].

Imaging studies, such as MRI and ultrasound, can be utilized to confirm physical exam findings. MRI is one of the most useful imaging tools because it allows for evaluation of the Achilles tendon in the sagittal plane to determine the length of the injured or diseased tendon and subsequent surgical planning. MRI has a sensitivity of 95% and a specificity of 50% when evaluating Achilles tendon pathology [8]. Ultrasound can verify the existence and location of intra-tendinous lesions. Ultrasound provides many advantages, quick, safe, and inexpensive, but also has many disadvantages being operator-dependent and not as readily accessible. Plain radiographs can also be used to evaluate retrocalcaneal bursitis and a possible Haglund's deformity, which is a prominence of the posterosuperior angle of the os calcis that causes mechanical irritation of the retrocalcaneal bursa that can exacerbate Achilles tendinopathy [8].

Nonoperative Treatment

The purpose of conservative treatment is to facilitate the return to activity and manage symptoms. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be a good short-term option for pain management. Steroid injections have also been used to provide some short-term relief; however, Achilles tendon rupture has been observed following these injections and are usually avoided [9].

A number of studies have demonstrated significantly improved outcomes with the use of eccentric exercise training. Eccentric heel-drop training (ECC) for 6 weeks exhibited a patient satisfaction score of >7 in more than 80% of patients, return to premorbid activity on average of 10 weeks. Additionally pain scores as measured by visual analog score (VAS) decreased from 7.2 at initiation to 2.9 after 6 weeks and 1.1 after 6 months [10]. Eccentric exercise for 12 weeks as the sole treatment for Achilles tendinopathy demonstrated clear changes to objective assessments of inflammation on MRI, tendon volume decreases from 6.6 ± 3.1 cm^3 to 5.8 ± 2.3 cm on T1-weighted images, and proton density on average decreased 23% [11]. Another study assessed changes to the Achilles tendon after 12 weeks of eccentric exercise using ultrasonography (US). At the widest portion of the Achilles tendon, patients in the ECC group showed significant changes compared to the control group, with change from 8.8 to 7.6 mm. Additionally, after exercise treatment 73% of the ECC subjects had normal Achilles tendon anatomy on US after treatment compared to the hypoechoic areas present at onset of the study [12].

Heavy slow resistance (HSR) training is another exercise treatment option. In a randomized control trial, comparing HSR to ECC, both groups showed improvement on US, the Victorian Institute of Sport Assessment-Achilles (VISA-A) questionnaire, and VAS scores. Patients in the HSR group were more satisfied at 12 weeks and were significantly more compliant. Differences in satisfaction and

compliance may be attributed to the time commitments for each. HSR required three 36-min training sessions per week, whereas ECC required two 22-min training sessions per day, 7 days a week [13].

Shoe modifications are commonly recommended to reduce Achilles tendon strain, promote movement variability of the hind foot, and correct eversion of the calcaneus when there is excessive pronation. Munteanu et al. conducted a randomized control trial comparing customized foot orthoses to sham orthoses and found no difference in the mean VISA-A score between groups, 82.1 ± 16.3 and 79.2 ± 20.0, respectively [14]. Night splints are also an effective treatment option, but are not as effective in return to play at 12 weeks and pain reduction when compared to ECC. There is no additive effect observed when night splints are used in conjunction with ECC [15].

Low-level laser therapy (LLLT) has demonstrated anti-inflammatory, regenerative, and anti-apoptotic properties. The use of LLLT in conjunction with ECC accelerates recovery faster than ECC alone. Extracorporeal shock wave therapy (ESWT) is another promising treatment option that improves VISA-A, VAS, and Likert scores; ESWT also has an additive effect when combined with ECC. Platelet-rich plasma (PRP) is a highly researched topic garnering much attention. PRP is beneficial to other tendinopathies. However, currently there are no RCTs that show significant improvement with the use of PRP specifically for Achilles tendon injuries [16].

Operative Treatment

Once patients fail conservative management, appropriate indications for surgical repair are evaluated. Operative treatment is pursued based on the nature and acuity of the injury. For patients that have sustained an acute rupture, undergo surgical repair once the initial swelling and ecchymosis subsides. In a meta-analysis performed by Bhandari et al., surgical repair showed a significant reduction in the risk of re-rupture compared to conservative treatment [17]. However, surgical repair increases the rate of infection. There were no major differences in return to normal function following rehabilitation.

For patients with chronic Achilles tendinopathy, there is much controversy regarding the approach (tendoscopy vs. open), the type of suture method, and need for augmentation. Percutaneous repair has shown to have lower infection rates than open repair, and the re-rupture rate of 2% is lower [18]. However, there is also an increased risk of damage or injury to the sural nerve. Re-rupture risk is partially related to the gap distance between the ends of the tendon and early weight bearing in the postoperative period [19].

Although simple end-to-end repair is common, evidence to support the use of augmentation has been increasing in the literature. Nonetheless, superiority of augmented tendon repair has yet to be established. A recent meta-analysis showed no major difference in outcomes of patients with augmented versus repairs without augmentation. Patients had no statistically significant difference for re-rupture rates, patient satisfaction, return to activity, or infection rate [20].

Conclusion

Injuries to the Achilles tendon can be debilitating for the masters athletes. Thorough assessment of the extent of damage to the structure is required. Appropriate patient selection for surgical intervention is necessary to achieve the best possible outcomes for athletic performance and to minimize unnecessary risk of infection associated with more of an aggressive approach. Most athletes can expect to return to sport around 6 months postoperatively and regain full function between 9 and 12 months after surgery.

Plantar Fasciitis

Introduction

Plantar fasciitis is a common condition leading to more than one million clinical visits per year. Approximately 60% of those visits were patients 45 years of age or older with patients between the ages of 45 and 64 having two times the average incidence of 8.2 per 1000 persons [21]. The reported prevalence of plantar fasciitis in runners is as high as 10% [22].

Clinical Evaluation

Patients with plantar fasciitis often complain of an insidious onset of pain on the plantar surface of the heel. The pain is worse with the first step down after sleeping and improves as they walk and my worsen at the end of the day. On exam, they may be observed walking on their toes, as this relieves the aching and tearing type of pressure from the fascia. Tenderness to palpation occurs at the plantar fascia insertion site on the medial tuberosity of the calcaneus, particularly with dorsiflexion of the forefoot. A tight Achilles tendon may also be noted on exam.

Nonoperative Treatment

First-line treatment is plantar fascia stretching and Achilles tendon stretching. Patients on a plantar fascia stretching protocol have VAS scores that reflect significantly reduced pain when compared to Achilles tendon stretching alone, although this effect is not sustained at 2-year follow-up [23]. The plantar stretch is performed by having the patient position their fingers at the base of the toes, with the toes dorsiflexed while using a free hand to apply tension on the toes until a stretch is felt in

the arch. Patients are encouraged to stretch before taking their first steps in the morning and after extended period of weight bearing that leads to pain [23, 24]. A tennis ball can also be used to assist with stretching and pressure massage of the scar tissue. NSAIDs can be added to the treatment regimen, but there is currently no literature to support the use of NSAIDs as the sole treatment for plantar fasciitis [25]. Cryotherapy application for 20 min at bedtime reduces pain and has been shown to have a significant impact when used in conjunction with NSAIDs [26].

The use of orthoses in the treatment of plantar fasciitis is common and in combination with stretching has better outcomes than stretching alone [27]. A study looking at patients 65 years of age or older demonstrated that prefabricated foot orthosis are the best at reducing pressure on heel and is 5 times better at pressure reduction than heel inserts or heel pad [28].

Injections are another line of treatment available for patients. Corticosteroid injections can provide relief of symptoms for several months. However, corticosteroids increase the risk of plantar fascia rupture and chances compound with more injections [29, 30]. Botulinum toxin A (BTX-A) is a novel injection option that has exhibited the ability to reduce pain and increase function up to 1 year. At 6 months, BTX-A on average decreased VAS scores from 7.2 to 3.6 and increased Foot and Ankle Ability Measure (FAAM) from 36.3 to 73.8 [31]. Though more long-term studies are needed to assess BTX-A injections, it currently has less side-effects than steroids.

Extracorporeal shock wave therapy (ESWT) is an FDA-approved treatment option for plantar fasciitis. A randomized control trial demonstrated significant difference in VAS and Roles and Maudsley scores in middle-aged patients treated with ESWT compared to placebo group, with a success rate of 50–65%. Temporary swelling and pain during treatment were the only device-related unfavorable events observed [32].

Operative Treatment

When all other conservative options are exhausted, then surgery becomes an option. Plantar fasciotomy is a regularly performed procedure for this condition and requires persistent pain after 9 months of failed conservative measures. This procedure has an outcome success rate up to 90% for distal tarsal tunnel decompression and dual plantar fascial release. This procedure may result in prolonged heal healing and rehabilitation time. Plantar fascia release is thought to decrease foot arch and ankle stability [24].

Conclusion

Irritation of the plantar fascia occurs in many athletes. Inflammatory flares results in nagging pain that can prevent continuous activity, especially in runners. Aggressive stretching and therapy are required to minimize the limits on exercise. Injections are

usually avoided to minimize the risk of rupture. For patients that undergo fasciotomy with or without tarsal tunnel decompression, success rates are high, 70–80% with a full return to activity expected. However, complications and slower recovery often occur and should be thoroughly considered prior to surgical intervention.

Stress Fractures

Introduction

Stress fractures are a common sports injury that account for 10% of all sports injuries, with as many as 30% of injuries in like runners and ballet dancers. They occur in roughly 20% of elite athletes and 1% among recreational athletes. An overwhelming 90% of stress fractures are in the lower extremities [33, 34]. Stress fractures are either low-risk or high-risk; high-risk fractures have increased rates of nonunion/delayed union and tend to be in areas not well vascularized [33–35]. Low-risk fractures tend to be in areas that are well vascularized and carry a decreased risk of nonunion/delayed union.

There are several factors that predispose athletes to stress fractures. Training pattern is a factor that is particularly important for masters athletes. An abrupt increase in training intensity increases probability of stress fractures. This principle has been observed with military cadets who have significantly higher rates of stress fractures during the first week of boot camp training. This phenomenon is also observed in professional soccer leagues, where a shorter preseason resulted in higher rates of stress fractures during high activity in the season [33].

Bone health is a predisposing factor that should be emphasized, particularly with female athletes. Athletes with irregular menstrual cycles can have a relative risk up to four times higher than their eumenorrheic counterparts [36]. Female naval cadets given daily calcium and vitamin D showed a 20% decrease in stress fractures compared to placebo. A similar study compared daily calcium supplementation to placebo in men and revealed no difference in stress fracture rates. Other predisposing factors include biomechanics, training surface, and footwear [33].

Clinical Evaluation

Navicular stress fractures are a common high-risk stress fracture most frequently seen in basketball players, ballet dancers, and runners [37, 38]. Patients usually complain of pinpoint pain that is worse with activity. The pain will usually have an insidious onset that is relieved with limited motion/activity. Approximately 40% of navicular stress fracture changes are absent on radiographs; CT should be used when patients continue to report pain despite negative radiographs [39]. The outcome is correlated to the severity of the fracture, which is assessed by using the Saxena classification (Table 12.2) [40].

Table 12.2 CT findings and treatment based on Saxena classification

Grade	CT findings	Treatment
Type 1	Involves only the dorsal cortex	• NWBC
Type 2	Dorsal cortex and the body are involved	• NWBC • ORIF once NWBC fails
Type 3	Dorsal and volar involvement	• ORIF or fusion

Nonoperative Treatment

There is no consensus on the treatment of navicular stress fractures due to the lack of randomized controlled trials comparing surgical to conservative treatments [22, 35, 37, 38, 40, 41]. The primary nonoperative treatment is the use of a non-weight-bearing cast (NWBC). NWBC for at least 6 weeks has a success rate of 80% in healing the fracture and return to normal activity. Treatment plans that allow patients to bear weight only have a success rate of 29% with higher rates of nonunion, delayed union, and/or refracture [37]. Bone stimulators are used in some elite athletes to accelerate the bone healing process. Bone stimulators cause no harm and have not proven to be beneficial for navicular fractures. However, this method has demonstrated the ability to accelerate healing in tibia and distal radius fractures [41]. Like bone stimulators, shock wave therapy has demonstrated a benefit in other types of osseous injury but lacks evidence for use in stress fractures due to the paucity of literature [41]. The average time of return to play is 21.7 weeks for nonoperative treatment [37].

Operative Treatment

Surgical intervention is indicated for patients with displaced or complete fractures (Saxena Grade III). Patients who undergo surgical fixation have a return to play time of 16.4 weeks and lower rates of nonunion, delayed union, and refracture when compared to nonoperative management [37]. Complications with open reduction and internal fixation (ORIF) include superficial infection, return to OR for removal of hardware, and nonunion [37]. In elite athletes and patients with high functional demands, surgical intervention is favored [41]. Patients with partial fractures observed on CT will improve with inactivity, but may become symptomatic upon return to sport. Surgical intervention reduces the uncertainty associated with nonoperative treatment, while also providing a faster return to play [41].

Conclusions

Stress fractures result from overuse and become more common with repeated force and load, especially as the arch changes over time. For patients that have relief with

inactivity, conservative measures such as bone stimulation and vitamin supplementation with vitamin D and calcium are the mainstay of treatment to promote bone healing. However, the navicular bone of the foot represents an area with low healing potential due to the lack of vascularity in the region. Therefore, some patients will require surgical fixation to return to sport. Surgical management of navicular stress fractures may offer a faster return to sport for high-level athletes. Further randomized control trails are needed to establish if this is true for the management of all navicular stress fractures.

References

1. Li HY, Hua YH. Achilles tendinopathy: current concepts about the basic science and clinical treatments. Biomed Res Int. 2016;2016:6492597.
2. Egger AC, Berkowitz MJ. Achilles tendon injuries. Curr Rev Musculoskelet Med. 2017;10(1):72–80.
3. Saltzman CL, Tearse DS. Achilles tendon injuries. J Am Acad Orthop Surg. 1998;6(5):316–25.
4. Pedowitz D, Kirwan G. Achilles tendon ruptures. Curr Rev Musculoskelet Med. 2013;6(4):285–93.
5. Johnston CA, Taunton JE, Lloyd-Smith DR, McKenzie DC. Preventing running injuries. Practical approach for family doctors. Can Fam Physician. 2003;49:1101–9.
6. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. Am J Sports Med. 2002;30(2):287–305.
7. Maffulli N. The clinical diagnosis of subcutaneous tear of the Achilles tendon. A prospective study in 174 patients. Am J Sports Med. 1998;26(2):266–70.
8. Reddy SS, Pedowitz DI, Parekh SG, Omar IM, Wapner KL. Surgical treatment for chronic disease and disorders of the Achilles tendon. J Am Acad Orthop Surg. 2009;17(1):3–14.
9. Kleinman M, Gross AE. Achilles tendon rupture following steroid injection. Report of three cases. J Bone Joint Surg Am. 1983;65(9):1345–7.
10. Verrall G, Schofield S, Brustad T. Chronic Achilles tendinopathy treated with eccentric stretching program. Foot Ankle Int. 2011;32(9):843–9.
11. Shalabi A, Kristoffersen-Wilberg M, Svensson L, Aspelin P, Movin T. Eccentric training of the gastrocnemius-soleus complex in chronic Achilles tendinopathy results in decreased tendon volume and intratendinous signal as evaluated by MRI. Am J Sports Med. 2004;32(5):1286–96.
12. Ohberg L, Lorentzon R, Alfredson H. Eccentric training in patients with chronic Achilles tendinosis: normalised tendon structure and decreased thickness at follow up. Br J Sports Med. 2004;38(1):8–11; discussion 11.
13. Beyer R, Kongsgaard M, Hougs Kjaer B, Ohlenschlaeger T, Kjaer M, Magnusson SP. Heavy slow resistance versus eccentric training as treatment for Achilles tendinopathy: a randomized controlled trial. Am J Sports Med. 2015;43(7):1704–11.
14. Munteanu SE, Scott LA, Bonanno DR, et al. Effectiveness of customised foot orthoses for Achilles tendinopathy: a randomised controlled trial. Br J Sports Med. 2015;49(15):989–94.
15. Roos EM, Engstrom M, Lagerquist A, Soderberg B. 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. 2004;14(5):286–95.
16. Filardo G, Di Matteo B, Kon E, Merli G, Marcacci M. Platelet-rich plasma in tendon-related disorders: results and indications. Knee Surg Sports Traumatol Arthrosc. 2016:1–16.
17. Bhandari M, Guyatt GH, Siddiqui F, et al. Treatment of acute Achilles tendon ruptures: a systematic overview and metaanalysis. Clin Orthop Relat Res. 2002;400:190–200.
18. Khan RJ, Carey Smith RL. Surgical interventions for treating acute Achilles tendon ruptures. Cochrane Database Syst Rev. 2010;(9):CD003674.

19. Gulati V, Jaggard M, Al-Nammari SS, et al. Management of Achilles tendon injury: a current concepts systematic review. World J Orthop. 2015;6(4):380–6.
20. Zhang YJ, Zhang C, Wang Q, Lin XJ. Augmented versus nonaugmented repair of acute Achilles tendon rupture: a systematic review and meta-analysis. Am J Sports Med. 2017:363546517702872.
21. Riddle DL, Schappert SM. Volume of ambulatory care visits and patterns of care for patients diagnosed with plantar fasciitis: a national study of medical doctors. Foot Ankle Int. 2004;25(5):303–10.
22. Kindred J, Trubey C, Simons SM. Foot injuries in runners. Curr Sports Med Rep. 2011;10(5):249–54.
23. Digiovanni BF, Nawoczenski DA, Malay DP, et al. Plantar fascia-specific stretching exercise improves outcomes in patients with chronic plantar fasciitis. A prospective clinical trial with two-year follow-up. J Bone Joint Surg Am. 2006;88(8):1775–81.
24. Neufeld SK, Cerrato R. Plantar fasciitis: evaluation and treatment. J Am Acad Orthop Surg. 2008;16(6):338–46.
25. Gill LH, Kiebzak GM. Outcome of nonsurgical treatment for plantar fasciitis. Foot Ankle Int. 1996;17(9):527–32.
26. Knobloch K, Grasemann R, Spies M, Vogt PM. Midportion Achilles tendon microcirculation after intermittent combined cryotherapy and compression compared with cryotherapy alone: a randomized trial. Am J Sports Med. 2008;36(11):2128–38.
27. Pfeffer G, Bacchetti P, Deland J, et al. Comparison of custom and prefabricated orthoses in the initial treatment of proximal plantar fasciitis. Foot Ankle Int. 1999;20(4):214–21.
28. Bonanno DR, Landorf KB, Menz HB. Pressure-relieving properties of various shoe inserts in older people with plantar heel pain. Gait Posture. 2011;33(3):385–9.
29. Acevedo JI, Beskin JL. Complications of plantar fascia rupture associated with corticosteroid injection. Foot Ankle Int. 1998;19(2):91–7.
30. Kim C, Cashdollar MR, Mendicino RW, Catanzariti AR, Fuge L. Incidence of plantar fascia ruptures following corticosteroid injection. Foot Ankle Spec. 2010;3(6):335–7.
31. Ahmad J, Ahmad SH, Jones K. Treatment of plantar fasciitis with botulinum toxin. Foot Ankle Int. 2017;38(1):1–7.
32. Gollwitzer H, Saxena A, DiDomenico LA, et al. Clinically relevant effectiveness of focused extracorporeal shock wave therapy in the treatment of chronic plantar fasciitis: a randomized, controlled multicenter study. J Bone Joint Surg Am. 2015;97(9):701–8.
33. Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. Sports Health. 2013;5(2):165–74.
34. Robertson GA, Wood AM. Lower limb stress fractures in sport: optimising their management and outcome. World J Orthop. 2017;8(3):242–55.
35. Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. J Am Acad Orthop Surg. 2000;8(6):344–53.
36. Beck BR, Matheson GO, Bergman G, et al. Do capacitively coupled electric fields accelerate tibial stress fracture healing? A randomized controlled trial. Am J Sports Med. 2008;36(3):545–53.
37. Mallee WH, Weel H, van Dijk CN, van Tulder MW, Kerkhoffs GM, Lin CW. Surgical versus conservative treatment for high-risk stress fractures of the lower leg (anterior tibial cortex, navicular and fifth metatarsal base): a systematic review. Br J Sports Med. 2015;49(6):370–6.
38. Mann JA, Pedowitz DI. Evaluation and treatment of navicular stress fractures, including nonunions, revision surgery, and persistent pain after treatment. Foot Ankle Clin. 2009;14(2):187–204.
39. Greaney RB, Gerber FH, Laughlin RL, et al. Distribution and natural history of stress fractures in U.S. Marine recruits. Radiology. 1983;146(2):339–46.
40. Saxena A, Fullem B, Hannaford D. Results of treatment of 22 navicular stress fractures and a new proposed radiographic classification system. J Foot Ankle Surg. 2000;39(2):96–103.
41. Shakked RJ, Walters EE, O'Malley MJ. Tarsal navicular stress fractures. Curr Rev Musculoskelet Med. 2017;10(1):122–30.